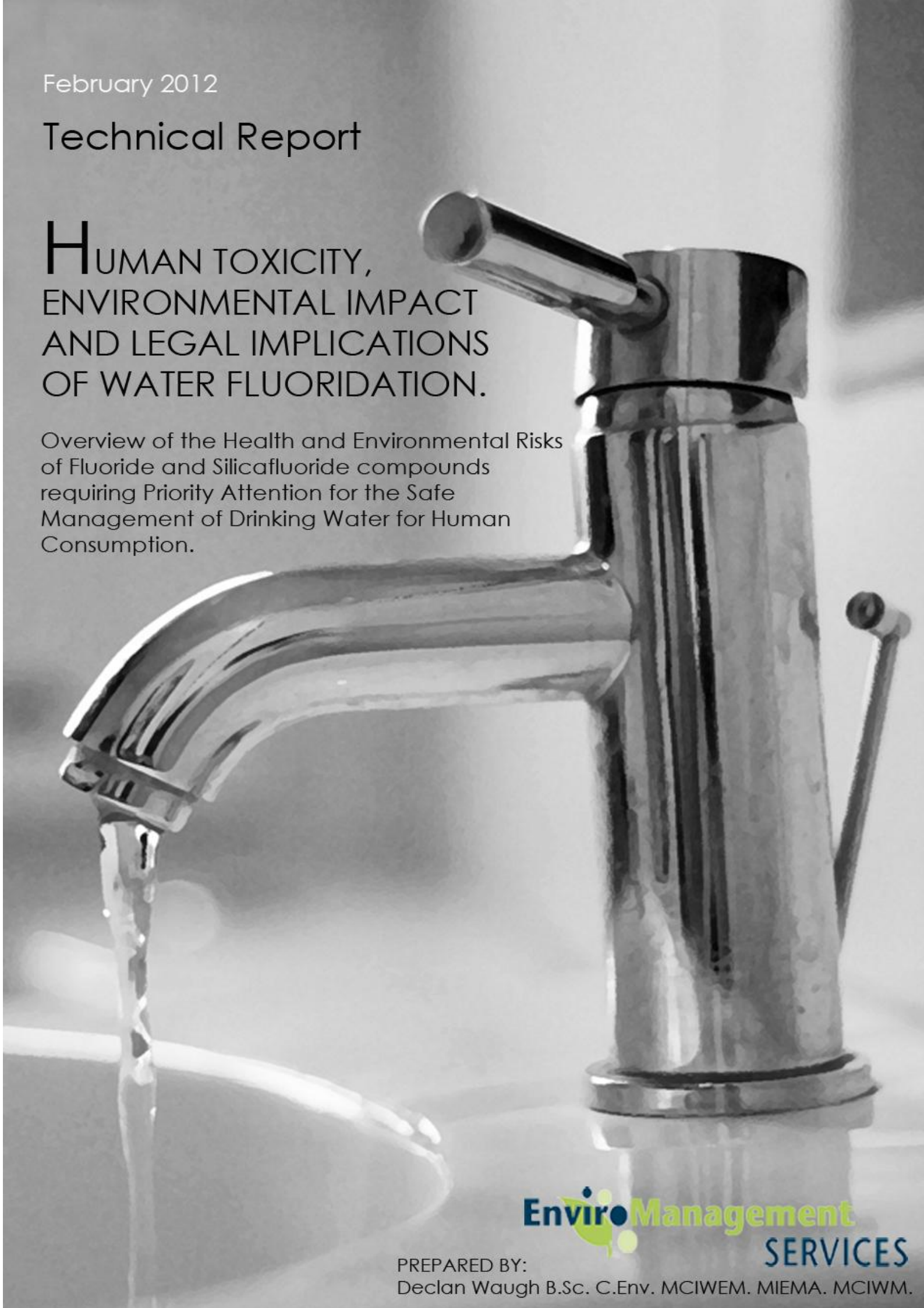
February 2012

Technical Report

HUMAN TOXICITY, ENVIRONMENTAL IMPACT AND LEGAL IMPLICATIONS OF WATER FLUORIDATION.

Overview of the Health and Environmental Risks of Fluoride and Silicafluoride compounds requiring Priority Attention for the Safe Management of Drinking Water for Human Consumption.



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Overview of the Health and Environmental Risks of Fluoride and Silicafluoride Compounds requiring Priority Attention for the Safe Management of Drinking Water for Human Consumption.

SUBMITTED TO:
The Government of Ireland and the European Commission

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National Executive Council, SIPTU	Mr. Martin Schulz, President of the European Parliament.

Article 174 of the TREATY OF EUROPE
mandates
that Community policy on the environment
must contribute to the preservation,
protection and improvement of the
quality of the environment,
the protection of human health and
the prudent and rational utilisation of natural
resources based on the

PRECAUTIONARY
PRINCIPLE.

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Acknowledgements

The Author wishes to acknowledge the comprehensive work of the committee members of the European Commission's Scientific Committee on Health and Environmental Risks (SCHER), the European Commission Directorate General for Health and Consumers who sought the review of fluoride and fluoridating agents of drinking water, the USA National Research Council (NRC) Scientific Committee on Fluoride in Drinking Water, whose members are drawn from the councils of the National Academy of Sciences, the National Academy of Engineering and the Institute of Medicine in the United States of America; the Forum for Fluoridation; Fluoride Action Network, NHS Centre for Reviews & Dissemination, University of York and Russian Academy of Sciences. There are many other data sources that the Author would like to acknowledge but the list is too extensive, it is hoped that they are appropiately referenced amongst the more than 1,200 scientific references contained within this report. A detailed list of peer-reviewed scientific publications cited in this report is provided in Appendix 1. The Author wishes to acknowledge the patience, support and assistance of Serena Corcoran BA. MA. H.Dip Education who volunteered her time to assist in editing this report without which this report would never have been finished.

The opinions of this Author present the view of an independent Environmental Scientist, a Chartered Environmental Consultant and member of the Chartered Institution of Water and Environmental Management, the Institute of Environmental Management and Assessment and the Chartered Institution of Waste Management. While the Author is a member of the Environmental Pillar for Social Partnership, this paper and the views expressed herein do not reflect the views of the Environmental Pillar of Social Partnership unless otherwise stated. Any opinions, findings, conclusions or recommendations expressed in this report are those of the Author and do not reflect the view of the organisations or agencies that provided information for this report.

In consideration of the concerns raised in this report, as documented in the extensive peer-reviewed risk assessment data examined in undertaking this study, the Author urges healthcare professionals, public health authorities, environmental health officers, environmental scientists, occupational health and safety officers, environmentalists, elected representatives and healthcare providers to review all current information available and referenced in this study and to use this information to seek an urgent re-evaluation of current policy and practices on fluoridation.

Potential conflicts of interest

The Author has no financial interest in fluoridation or alternate treatments for public water supplies nor in any form of defluoridation. This report was undertaken voluntarily by the Author for public dissemination to support a review of the existing regulatory policy of mandatory legislation for water fluoridation based on the scientific data presented in this study. The Author received no gratuities, commission, compensation nor professional fees for preparing this report. It has been undertaken in the public interest with no financial gain. The review was undertaken for the people of Ireland, particularly to benefit consumer rights, consumer protection, the health and well-being of individuals and the environment. This review is intended for informational purposes only and is not intended to be used as a substitute for medical advice.

Limitations

EnviroManagement Services herein referred to as 'the Author' produced this report for the sole purpose of public interest and dissemination of up-to-date scientific facts and background information relating to water fluoridation, public health and the environment. At the beginning of his research the author had no intention of writing such an extensive technical report. This report evolved both as a consequence of the alarming evidence and information reviewed as well as the lack of specific risk assessment data from which to comprehensively examine and determine the overall risks and environmental impacts associated with water fluoridation. The Author thus acknowledges that this is by no means a comprehensive synthesis of the environmental or public healthcare risks associated with water fluoridation. They are too many to report in full within the context of this report. This is a complex issue, and the author of this report does not purport to be an expert in all the areas addressed in this report. Nevertheless, as an Environmental Scientist with over twenty years professional experience, the Author is conversant in many of the areas addressed in this report. The completion of this review was particularly challenging owing to a limited timeframe and the difficulties encountered by the Author in finding relevant scientific information and reliable evidence with which to inform existing policy. A comprehensive review of international scientific research is, however, provided in this report. It should thus help provide more detailed specific information to assist with a significant and urgent re-appraisal of current policy.

This report is a synthesis report – it synthesises the important issues around this topic: the environmental risks, the doubts surrounding the proclaimed benefits of water fluoridation, the known and potential public health risks, some of the important legislative issues that must be addressed, the scientific, medical and epidemiological research that is requried and has yet to be undertaken on fluoride and fluoridation products as well as the opinions and conclusions of the author as informed from the research undertaken. The restricted timeframe for researching this given topic denotes that there are some areas within this report that could not be specifically covered. Therefore, it was not possible to undertake a comprehensive investigation of the many and varied environmental and public health risks associated with water fluoridation. A number of areas in need of further research are identified throughout the report.

Disclaimer

A note on terminology:

Fluorine is an element in the halogen group as are chlorine and iodine. Fluorine is the most chemically active non-metallic element of all the elements and also has the most reactive electro-negative ion. Because of this extreme reactivity, fluorine is never found in nature as an un-combined element. Fluorine compounds or fluorides are listed on the Priority list of Hazardous Substances by the U.S. Agency for Toxic Substances and Disease Registry (ATSDR) as substances that pose the most significant threat to human health.

Fluoride: Any combination of fluorine with another element or chemical group of elements. Thus, the addition of fluorides to drinking water can indicate the addition of a large number of chemical agents. The most commonly used fluorides for this purpose are sodium fluoride, NaF, hydrofluorosilicic acid, H_2SiF_6, (which is the product used in Ireland), a compound that contains both fluorine and silicon. Such agents are collectively called "Fluorosilicictes." They include fluorosilicic acid, fluorosilicicte, hydrofluosilicic acid and hexafluorosilicic acid.

Fluoride is a List ii substance under the Council Directive 80/68/EEC relating to the prevention of discharges of certain toxic, persistent and bioaccumulable substances into groundwater.

Fluoride is listed as an undesirable substance in Annex 1 of Directive 80/778/EEC relating to the quality of water intended for human consumption.

In a comprehensive hazard identification study undertaken by the U.S. EPA examining 254 pollutants that may cause adverse effects to public health and the environment involving quantatitive risk assessment, including dose response evaluation, exposure assessment and risk characterisation, the pollutant fluoride was identified as one of the top twelve critical pollutants.

1.0 INTRODUCTION

The author of this report had no fixed opinion on water fluoridation before commencing this research and originally had no intention of ever writing a report on this subject. The author undertook this research as a concerned parent and care provider following the recommendations of scientific committees that parents should be knowledgeable about the adverse health effects of fluoride exposure on their children as well as their own individual dietary fluoride intake as a measure towards preventative healthcare. A major factor in deciding to write this report was the recommendation by the British Medical Council that more robust information on the potential harm of fluoridation is needed. This report attempts to address this concern.

Following a review of the technical information provided by international scientific committees and peer-reviewed scientific journals it has become abundantly clear that the perceived benefits of fluoridation are minor compared to the acknowledged risks to public health and the environment from systemic water fluoridation. There has never been any comprehensive environmental risk assessment examining human toxicity, the environmental impact and the legal implications of water fluoridation. Many previous studies focused on one particular area, generally health and highlighted the lack of available data to prove fluoride was safe often overlooking certain scientific observations from academic scientific journals not readily accessible to the public and entirely ignoring due diligence on environmental risk assessment or legislative due diligence. There has also been some controversy regarding transparency and inadequate disclosure of scientific reporting. In an attempt to update scientific knowledge on new research findings associated with fluoride while providing a more theoretical knowledge-based approach to environmental risk management through the examination of the broader implications of water fluoridation, this report provides a more detailed review of the human health risks, environmental impacts and legal implications of water fluoridation than have been previously examined.

The author believes that public policy should evolve with developments in science and that scientific research has demonstrated that continuation of the policy of systemic water fluoridation is no longer justifiable. It is absolutely certain that the policy of mandatory water fluoridation violates both European and International Law. Regardless of the findings of this report, in current economic times when public finances for critical healthcare, education and social policies are being cut, it is absolutely incomprehensible that funding would be provided by the Government of Ireland to continue with such an ineffective policy against the recent recommendation of the EU Scientific Committee on Health and Environmental Risks, who found that not only was the policy ineffective, but any data supporting its benefits was lacking scientific credibility. Ireland is the only country in the European Union with a mandatory legislative policy on water fluoridation. The worst that could happen if the policy ended tomorrow is that Irish citizens would have the same rights as every other European citizen to clean safe non-fluoridated drinking water.

It is particularly significant that traditionally perceived medical claims in support of water fluoridation have been disputed by international scientific committees, who were unable to find any conclusive studies to support the existing medical claims in support of fluoridation, if, in fact, such claims have ever been made in a sound scientific manner. Simply put the benefit of fluoride for dental decay has been proven to be by the application of fluoride toothpaste onto the enamel of the tooth. not by the ingestion of fluoride into the body and the interaction of fluoride in blood plasma with the developing teeth of children.

The ever-growing body of scientific literature examining the potential impact on humans and the environment of the toxic effects of fluoridation compounds are reviewed in this report. Over two hundred and twenty separate peer-reviewed scientific publications covering every aspect of medicine and environmental assessment from dental health to biochemistry, toxicology, metabolism, the blood, bone research, the brain, metabolism, epidemiology, pharmacology, neurotoxicology, molecular neurobiology, dental health and environmental toxicology have been examined and reviewed. In total over twelve hundred scientific references are provided in this report allowing the health, legal and environmental impacts to be examined in some detail alongside associated risks that have not yet been previously examined elsewhere. All of the evidence is convergent and demonstrates that fluoride compounds should not be added to public water supplies, when examined collectively the evidence clearly demonstrates that fluoridation of drinking water supplies is both unsafe and having significant negative health implications for human health, society and the natural environment.While the search for knowledge is never complete, this report usefully identifies research that should inform public policy going forward as part of our continual efforts to refine the evidence-base for policy-making. This report provides overwhelming evidence-based information to allow the State to urgently review the policy of water fluoridation. Following a comprehensive review of the most up-to-date scientifically valid information available, the inescapable conclusion reached in this study, is that the practice of water fluoridation results in the ingestion by the public, and release into the environment, of dangerous chemicals that are harmful to public health and the environment.

One central and astonishing fact that has been documented repeatedly by every assessment to date is that the products used for water fluoridation have never been tested for safety on humans nor the environment. This is both illogical and unlawful. Water Fluoridation in the Republic of Ireland is in fact a State-sponsored policy and not EU Law, nor does it support EU policy in any shape or form. As an outdated national policy it is in breach of over thirty EU Directives and policy documents in addition to international legal treaties on human rights and the environment. The potential financial, legal, healthcare, social and political impacts of such a policy are truly enormous not to mention the future damage to international public relations, tourism and food exports for Ireland if it were to continue to support such a damaging policy. In light of recent scientific findings addressed in this report continuation of the water fluoridation policy in this country could have serious implications for the health and welfare of its citizens as well as for its economy.

2.0 THE MAIN FINDINGS ON HUMAN HEALTH TOXICITY AND THE ENVIRONMENTAL IMPACT OF WATER FLUORIDATION

This investigation has been undertaken in order to update the Government of Ireland, the European Commission, their agencies and the public on recent scientific findings on the human health risks and environmental impacts of water fluoridation. The report further addressess, for the first time, the legal implications of fluoridation relating to drinking water, food safety, consumer rights and protection of natural resources.

Water is necessary not only for drinking, but also for food production and preparation, personal hygiene, care of the sick, cleaning, washing, waste disposal and care of domestic animals. Access to safe drinking water is a basic human right. It is now known that the policy of fluoridation of drinking water supplies is unsafe and places an unacceptable health burden on people and an unsustainable impact on the environment.

While the practice of fluoridation of drinking water was intended to have a beneficial effect on caries prevention and to reduce social inequalities in dental health, there is now unequivocal evidence to show that the practice is now contributing to adverse public health risks and environmental impacts. The public have always been assured that there was absolutely no possibility of any harm or risk from fluoridation of water. There is now unequivocal evidence that demonstrates that this is not the case. This report presents the scientific and medical evidence from over twelve hundred peer-reviewed scientific articles that demonstrates beyond any reasonable doubt that fluoridation of drinking water is a significant contributory factor to the negative health burden of Ireland. This report presents a summary of the published peer-reviewed health and environment related literature on fluoride and its implications for human health and biodiversity.

Reseach findings have demonstrated the ability of fluoride to act as an enzymatic poison in the human body inhibiting critical metabolic pathways required for healthy living. This report clearly demonstrates how inhibition of certain metabolic pathways is linked with increased neurological and cardiovascular diseases as well as dental, skeletal and mental fluorosis. Apart from bones and teeth many of the essential human organs in the body are directly affected by fluoride including the heart, kidneys, liver and pineal gland. Fluoride is now known to cause calcification in human arteries resulting in plaque formation and increased risk of stroke and heart disease. Significant calcification also occurs in the pineal gland and kidneys.

It is now known that individuals with renal disease and infants bottle-fed with formula milk reconstituted with fluoridated water are the most at risk from the impacts of fluoride. Fluoride is now known to act as a destabiliser of calcium and magnesium bonding in the human body with serious consequences for human health. Fluoride is also known to have major co-toxicity health implications when it complexes with other pollutants present in water such as

aluminium. This is known to have major health implications including neurological and chronic bone pain.

Largely as a consequence of fluoridation of drinking water, fluoride has become one of the most widely available elements that is present in artificially elevated concentrations in potable water, processed foods and drinks prepared with treated water as well as cooked foods prepared with fluoridated water. As a consequence, the daily intake and exposure of the population to fluoride is now considerably higher than that of any previous generation.

The Health (Fluoridation of Water Supplies) Act, 1960 is a public health measure in the field of preventative dentistry that legislates for fluoride or fluoride products to be added to waters to prevent disease in human beings. As with any product, the State must ensure that it has undertaken rigorous scientific examination of the health and safety risks of fluoridation products. In order to quantify the potential public health risk and economic costs of fluoridation of drinking water, in excess of fifty comprehensive epidemiology, toxicology, clinical medicine and environmental exposure assessments were identified requiring urgent assessment by international scientific committees representing the National Research Council of the United States of America and the European Commission's Scientific Committee on Health and Environmental Risks. In reality, there have been limited and incomplete studies on the health and environmental effects of exposure to fluoride. None of the peer-reviewed recommended assessments have been undertaken by the public health authority with responsibility for water fluoridation in Ireland. This in itself is unusual since populations, especially in countries where water fluoridation is national policy, are already being exposed to this element without adequate health and safety assessments being undertaken on how they might be affected.

Within Europe Ireland remains the only remaining country with a legislative policy for fluoridation of drinking water. A policy which is mandated through national legislation that predates its membership of the European Union. The provisions of the 1960 Health (Fluoridation of Water Supplies) Act were brought into force in 1965 by a series of Statutory Instruments containing Ministerial Regulations. This 1960 Act is the primary legislation governing the fluoridation of public drinking water supplies in Ireland. There is no EU legislative provision allowing for the fluoridation of drinking water supplies. In contrast to this Act, almost every other piece of active water or environmental legislation with legal status was introduced following Ireland's entry into the European Union in 1973.

In the context of existing EU and national regulatory legislation concerning the environment, health and food, it has been found that the policy of water fluoridation contravenes thirteen EU Directives, three EU Food Regulations, four Statutory Regulatory Instruments, one EU Medical Directive, One EU Product Directive, seven international Treaties, three European Conventions and six European Action Policies totalling thirty-eight separate acts of legislation. This may indeed not be the final number but reflects the significance of the violations as examined by the Author of this report. It may

perhaps also provide a clear answer as to why no other European country accepts the position of Ireland with respect to water fluoridation.

In 2000, the NHS YORK Review, following a critical and detailed examination of water fluoridation, concluded remarkably that it was unable to identify one high-quality study to show that the practice is effective or safe.

In 2002, the British Medical Council observed that there are many detrimental impacts of water fluoridation and that more robust information on the potential harms of fluoridation is needed.

In 2003, the European Commissions Scientific Committee on Cosmetic Products and Non-Food Products (SCCNFP) intended for consumers undertook a study of the safety of fluorine compounds in oral hygiene products for children under six years of age. SCCNFP observed that systemic exposure to fluoride, resulting from fluoridation of drinking water supplies not only contaminates infant formula food but may impair normal development of enamel in the pre-eruptive tooth and cause fluorosis.

In 2006, the U.S.A. National Research Council (NRC) Scientific Committee in their comprehensive report on fluoridation, highlighted an alarming number of potentially adverse public health risks associated with water fluoridation. Furthermore, the NRC documented the growing weight of toxicological and epidemiological evidence identifying clear public health risk associated with the addition of fluoride to public drinking water supplies.

All independent scientific reviews have agreed on one important legal principle, that is that there exist both known and probable risks that can cause harm to the public and to the environment. Furthermore, all international scientific reviews have themselves independently raised concerns regarding the lack of appropiate scientific risk assessment that would demonstrate beyond reasonable doubt that no harm will result from water fluoridation.

More recently, in 2010, the European Commission's Scientific Committee on Health and Environmental Risks (SCHER) was unable to demonstrate the benefit of fluoridation of drinking water for dental health. SCHER concluded that while the scientific evidence for the protective effect of topical fluoride (toothpaste) application is strong, the respective data for systemic application via drinking water is less convincing. These finding are remarkable as they indicate quite clearly that SCHER believes that the evidence for supporting water fluoridation, despite half a century of implementation in some countries, like Ireland, is inconclusive and lacking scientific merit.

Ultimately, the Irish Government must examine the policy of water fluoridation in the context of one of the key principles of the National Health Strategy for the Republic of Ireland, that is, ensuring equity. Ensuring equity demands that health inequalities are targeted and that people are treated fairly according to their needs. The information in this report clearly demonstrates how the objective of ensuring equity cannot be maintained by continuation of the policy that specifically targets risks at the most vulnerable in our society.

Parents and care providers must be allowed to make informed choices on child-care and it is inconceivable that any parent or government would willingly allow infants under one year of age to consume multiples of the daily recommended intake of a known toxic substance by consuming infant formula food contaminated with fluoride from artificially fluoridated water. The State has a duty of care to protect the most vulnerable and to act on scientific evidence.

Equally, every consumer has a right to be fully informed of the quality of drinking water they consume and the health risks or implications of any interventions made on their behalf by the State that may impact on their health and wellbeing. European citizens are advised to be aware of their total dietary fluoride intake yet Ireland is the only country with a policy to fluoridate its public drinking water supplies. Consumers in Ireland are completely unaware of the health and environmental impacts of water fluoridation because they are not informed by the authorities of the risks.

Fluoridation of water supplies may be regarded as an insidious poison that accumulates in the human body and environment over time. As with any poison the severity of the health problems depends on how much fluoride an individual is exposed to and at what stage in their development. In many respects, the toxicity of fluoride is similar to both lead and arsenic.

As with both of these harmful substances, young children are most susceptible to fluoride poisoning. Similarly as with lead or arsenic poisoning there may be no initial signs or symptoms and it may take some time before the signs and symptoms of poisoning become manifest. Consumers are advised not to consume toothpaste as it may be fatal if swallowed. In the same manner consumers should not need to ingest fluoride with drinking water to derive its purported benefit to the surface of teeth in contact with water. All water consumed as part of the daily fluid or food intake for infants and adults includes fluoride and fluoride compounds. All water used in industry and the household for whatever purposes, including washing and sanitation, is needlessly fluoridated. This has resulted in ever-increasing quantities of fluoride accumulating not only in humans (at alarming levels - thousands of times higher than originally present in water), but also in the wider environment in rivers, soils and ecosystems where it persists indefinately.

It is time for Irish citizens to have the same standard of care and protection as other European citizens. To this end, the Government must adopt a policy based on current scientific knowledge and harmonise its public health and water management policies with those of all other EU Member States by ending its policy of water fluoridation. It is clear, based on the information contained herein that the Health Service Executive(HSE), the Food Safety Authority (FSA), the Environmental Protection Agency(EPA), Inland Fisheries and other State Agencies and Departments must adopt a precautionary approach to risk prevention and in doing so align Ireland with every other European Member State by ending the policy of water fluoridation forthwith.

It is also apparent that by ending the policy of water fluoridation, it would save the Exchequer much needed tax revenue that could be spent on critical healthcare, education, infrastructure or community and social programmes. Public finances could also be more appropriately directed to public dental health programmes for the disadvantaged. It is additionally apparent, given the alarming number of health risks and diseases resulting from exposure to fluoride, that the single biggest contribution and potential cost-saving exercise the current Government could make to support the Health Service in Ireland, while benefitting the health and welfare of its citizens, could be achieved by following the precautionary approach and ending the policy of water fluoridation.

2.1 HEALTH RISKS

The most recent investigations of fluoride and water fluoridation have documented the growing weight of toxicological and epidemiological evidence that there is a clear public health risk associated with the addition of fluoride (or fluoride substances) to public drinking water supplies. Fluoride is now known to be a risk factor in developing many of the most serious health problems prevalent in the population of Ireland. This includes neurological and cardiovascular disease, type ii diabetes, osteoporosis, hypercalcemia, sarcoidosis, skeletal fluorosis, skeletal muscular disorders and pediodontal disease.

The incidence of these diseases in Ireland is far above the global average. It is reasonable to conclude that fluoridation of drinking water supplies is having both a significant negative health and economic impact on consumers and wider society. Apart from the many health risks associated with over-exposure to fluoride, it is classified as a persistent inorganic pollutant that can bioaccumulate in the environment and food chain. Fluoride in the body is retained in calcified tissue, bone and teeth as well as the pineal gland, kidney and other tissues. In blood, fluoride acts an an enzymatic poison inhibiting normal metabolic processes in the body.

The toxicity of fluoride is associated with its high chemical and biological activity. Fluoride is known for its aggressive interactivity properties and actively seeks out essential elements like calcium and magnesium interfering with their capacity to fulfil important metabolic processes in the body. It is known that the level of fluoride absorption in the body depends significantly on the presence of calcium, magnesium and aluminium. Calcium deficiency is associated with higher risk of fracture in children, certain neurodegenerative diseases, pre-term birth and low weight at birth and some types of cancer as well as cardiovascular mortality from cardiovascular, ischaemic heart and hypertensive heart disease. In addition, low calcium is associated with increased risk of sudden death. Low magnesium seems to be associated with a higher risk of motor neuronal disease, pregnancy disorders (so-called pre-eclampsia), certain cancers, hypertension, coronary vascular disease (CDV) and Type 2 diabetes.

Aluminium is a potent neurotoxic agent in humans, alarmingly it has been found that in drinking water treated with aluminium compounds the addition of fluoride increased the amount of soluble aluminium by a factor of ten. It has been suggested that aluminium by itself may not exert toxic effects on the nervous system, but is a dangerous toxin after binding to fluoride to become an aluminium fluoride.

Fluoride induced apoptosis (cell injury death) was demonstrated in the cells from different organs and tissues including lungs, kidneys, liver, brain, pancreas thymus, endometrium, bone marrow, hair follicles, erythrocytes and leukemic cells. It has been found that fluoride is a toxic anion that stimulates cellular oxygen consumption producing highly destructive free radicals such as superoxide radicals that can damage cell membranes and lead to oxidative stress. Oxidative stress is also a common mechanism by which chemical toxicity can occur in the liver. Fluoride depletes the energy reserves and the ability of white blood cells to properly destroy foreign agents by the process of phagocytosis. Fluoride inhibits AdoHydrae and homocysteine metabolism which is linked to cardiovascular disease, atherosclerotic disease, congenital heart defects, Down Syndrome, neurodegenerative disorders including depression, schizophrenia, bi-polar disorder, epilepsy, behavioural disorders, Alzheimer's disease and carcinogenesis.

Research has recently found an inverse association between fluoride in drinking water and decreased intelligence in children. Fluoride has been found to depress melatonin synthesis in the pineal gland and induce accelerated sexual maturity in both humans and animals. Fluoride has profound effects on the skeleton. It has been found to cause decreased cortical bone mineral density, poor bone quality, increased skeletal fragility, osteomalacia, rickets, periodontal disease, osteoporosis, osteoporotic hip fractures and is positively associated with rhematopid arthritis, bone pain and proximal myopathy (neuromuscular disease resulting in muscle weakness).

A body of scientific evidence indicates that fluoride intake from fluoridation of drinking water supplies exposes a significant sector of the population to unnecessary health risks; in particular bottle-fed babies, young boys (pre-adolescent boys who drink fluoridated water are at a seven-fold increased risk of osteosarcoma) patients with renal impairments, individuals with diabetes, individuals at risk of bone fractures or bone development problems, individuals with sensitive gastrointestinal systems and individuals who are immunocompromised and who could be at greater risk of the immunologic effects of fluoride, individuals with Alzheimer's disease or dementia and children with special healthcare needs who have a developmental, mental, sensory, behavioural, cognitive or emotional impairment related to neurological development disorders.

Given the disturbing findings regarding the neurotoxicity of fluoride, it is not beyond consideration that Ireland, regarded by many as the most fluoridated country in the world (over 78% of the population consume fluoride in drinking water), may therefore show an association with increased neurological disorders. It is accepted that Ireland has one of the highest incidences of neurological disorders in the world, including epilipesy, as well

as cardiovascular disease and dental fluorosis. In addition, alarmingly, it is estimated that 300,000 people in Ireland over the age of 50 have osteoporosis and sadly that over 500 patients die each year in Ireland from complications resulting from osteoporotic hip fractures.

In the past, serious environmental health problems have arisen whenever the efficacy and safety of chemical agents have not been comprehensively studied before their introduction. It is precisely because of this that both researchers and regulators have adopted the "precautionary principle". Effective management of the health system and environment requires society to continuously review scientific understanding of risks and cost benefits of Government policy. In the absence of proper studies into the efficacy and safety of water fluoridation, the following question must be asked; is it acceptable for the Government of Ireland to continue to conduct what amounts to an uncontrolled experiment on its own people?

The professional approach to risk assessment in toxicology and the environment is to identify the high risk groups in the community and to set safety standards for daily doses with sufficient margin to protect them with a high degree of certainty. For such purposes the adequate intake of fluoride for infants aged from 0 to 6 months as defined by the Food and Nutrition Board (FNB) in the Institute of Medicine of the U.S. National Academies is 0.01mg/l. It is an absolute certainty that all bottle-fed infants (>55,500 infants) under 6 months of age using formula reconstituted from fluoridated water would exceed this recommended level by multiples of 6-10.

This represents a serious breach of the most basic fundamental right of citizens of Europe, as mandated in the Charter for Fundamental Rights which demand that Member states ensure a high level of consumer protection and in particular the rights of children to protection and adequate care.

It is perhaps unsurprising that upwards of 400,000 children under 18 years of age (40%) are known to have dental fluorosis, demonstrating a chronic over-exposure to fluoride in the wider population. At the most basic bio-monitoring level, this demonstrates that fluoride added to water, so that it may make contact with teeth in an attempt to reduce the incidence of dental disease, increases the concentration of fluoride in blood plasma to a level so high that it causes visible physical structural damage to teeth. It is absolutely nonsensical to suggest that the only negative impact of water fluoridation to humans is dental fluorosis.

While the incidence of dental fluorosis is extraordinarily high in children and young adults in Ireland, even more alarming is the fact that over the period of time that water fluoridation has been practised in the Republic of Ireland approximately 75% of individuals born in the country whom are alive today and under the age of 50 (which represents the largest population group in Ireland), and who were bottle-fed as infants, would have been exposed to even higher levels of fluoride in drinking water. While the health implications of this for society have never been properly examined many are addressed in this report.

It is consequently a very serious and alarming development to observe that up to 8.1% of the water samples taken from public group water schemes are reported to have exceeded the legal standard for fluoride, a level that is coincidentally twice the recommended concentration for drinking water in either Ireland or the U.S.A. The most recent EPA report noted that one public water supply of 51 exceeded the fluoride standard of 1.5mg/L in 2010 while the SCHER review reported that levels in Ireland in excess of 5mg/l have been documented. No details are provided for the location of the water supply, the period for which these exceedances occurred nor the population numbers at risk from the chronic fluoride exposure as a consequence of the reported exceedances.

Astonishingly, it has also been reported by the EPA that the population are regularly exposed to extremely high concentrations of this toxin due to repeated accidental releases as a consequence of persistant operator errors at water treatment facilities. The health or environmental implications of these incidences have never been examined or reported.

As an environmental health policy, it is remarkable that approximately only half of one % of the fluoride added to drinking water ends up being used for the purpose it was intended. In total approximately 99.45%[1] of the fluoridated water is not used for the purpose for which the policy is undertaken, in effect this singularly represents one of the most ineffective health policies of any country globally.

This report also highlights how the policy of water fluoridation in Ireland has acted as a continuous source of fluoride pollution of the environment over the past four decades, releasing approximately 78,400,000kgs of fluoride into the environment through wastewater effluent directly discharged into rivers and treated wastewater sludge disposed of onto agricultural land.

2.2 ENVIRONMENTAL RISKS

In addition to the health risks of fluoride, it is acknowledged on the material safety data sheet for the product used for fluoridation that the substance is also harmful to aquatic ecosystems in low concentrations. Fluorides released into the environment as a consequence of water fluoridation act as persistent pollutants that accumulate in the aquatic and terrestrial environment. Fluoride is classified as a persistent inorganic pollutant that will bioaccumulate within the environment and food chain; the policy of water fluoridation in Ireland has resulted in the indirect release of significant quantities of fluoride into soils, groundwater and surface waters in significant concentrations with little or no controls.

Fluoride added to drinking water enters the freshwater ecosystem directly from multiple point source emissions and from surface run-off. Surface run-off from fire-fighting, washing cars and watering gardens may enter streams directly or through storm sewers at optimal concentration. Most fluoride enters from point sources, such as wastewater treatment plants, and from leakage

in drinking water infrastructure. To my knowledge, no comprehensive environmental impact assessment has ever been undertaken examining the potential impact on surface water ecosystems of fluoride in wastewater discharges. Nor to my knowledge have any field studies ever been undertaken to examine the effect of fluoride on salmon or other freshwater species in Ireland.

The Irish EPA have documented that potential waters at risk from fluoride pollutant include receiving waters located downstream of drinking- and waste-water treatment plants and areas where there is significant leakage from the drinking water distribution system. The agency further reported a number of exceedances of the standard for fluoride associated with the infiltration of drinking water into surface waters as well as leaking drinking water distribution mains into groundwater aquifers, which the agency regarded as a significant potential source of fluoride.

It is known that as a water pollutant, elevated concentrations of fluoride may affect a number of organisms; including fish, amphibians, insects, snails, shellfish, protozoa and some aquatic plants. It has also been documented that fluoride is an endocrine disruptor in the freshwater environment. If behaviour-altering pollutants such as fluoride are present in critical concentrations, it is likely that the migrating adult salmonids would respond to them in a short timeframe. Serious hazards to fish could arise through unperceived or unavoidable low level pollutants, in particular altering predator or food detection, reproduction or migration.

It is considered that the affects of fluoride emissions would be most pronounced in soft waters representative of the best salmonid rivers of Ireland. One must consider that with over 240 water fluoridation plants located in every water catchment area within the country and over 478 wastewater treatment plants discharging fluoride into freshwater, estuarine and coastal waters that the potential ecological impact over time may be enormous. This is especially so when it is known that over 78,400,000kgs of fluoride have been discharged into the environment from these facilities since fluoridation of water commenced in Ireland.

Fluorides are known to be toxic to trout and other fish, in particular smolting juvenile salmon. It is very worrying therefore to note that anthropogenic fluoride emissions are discharged from wastewater treatment plants into some one hundred and forty eight salmon rivers across the Republic of Ireland. While other factors also have contributed to the decline in freshwater salmon populations in Ireland, it is remarkable how the start of the decline in stocks mirrors exactly the commencement of water fluoridation in Ireland.

Apart from fisheries it also known that fluoride can either inhibit or enhance the population growth of algae, depending upon fluoride concentration, exposure time and algal species. It is also known that fluoride has a strong binding potential to sediments in rivers. Additional research has demonstrated that fluoride released into marine environments has been shown to accumulate in aquatic organisms.

Fluoride is also a pollutant that is present in sewage sludge and processed waterwater sludge where fluoridated waste has been used in the manufacturing process. Sewage and processed sludge has been and continues to be applied to land in Ireland. While the State has an obligation to protect public health and the environment from the anticipated adverse effects of this pollutant on the environment, inadequate research has been undertaken to examine the potential impact of this pollutant on groundwater, surface waters and human health in Ireland.

The U.S. EPA undertook a hazard identification study of pollutants that may cause adverse effects to public health and the environment. Based on the results of this study a list of 254 pollutants were identified and of these 31 were then evualated in a comprehensive hazard identification study. In their study, a quantitative risk assessment, including dose response evaluation, exposure assessment and risk characterisation, was performed. The goal was to identify pollutants that may potentially cause human health or ecological risks for a highly-exposed individual. Only pollutants that were deemed to be of risk to human health based on available human and ecological toxicity data were examined. Based on the results of the risk assessment and hazard identification, twelve pollutants were found to have critical exposure pathways for humans. Fluoride was identified as one of the principal pollutants.

This finding is particularly concerning for a small island nation such as Ireland where agricultural land is limited and food production is a significant part of the economy. The EPA in Ireland have documented that over 120,000tonnes tonnes of dried sludge is produced nationally from wastewater treatment plants annually, of which approximately eighty % is used for agriculture.

The concentration of fluoride is not measured in sewage sludge disposed of or re-used in agriculture in Ireland and despite the alarming findings of the U.S. EPA no risk assessment on the impact of fluorides on food production and human health has ever been undertaken in Ireland.

2.3 INADEQUATE RISK ASSESSMENT

While it is accepted that the toxicology of hexafluorosilicic acid and hexafluorosilicictes compounds used to fluoridate drinking waters are incompletely investigated and that additional epidemiology, toxicology, clinical medicine and environmental exposure assessments need to be undertaken in order to fill data gaps in the hazard profile, the health effects and the exposure assessment of fluoride; the Government of Ireland continues to support the policy of fluoridation in the absence of any scientific data to demonstrate that the practice is safe or effective.

The State is required, for the protection of public health, to undertake detailed risk assessment and clinical trials including comprehensive toxicological and pharmacological tests to demonstrate the effectiveness and risks associated with fluoridation of water. Despite a legal requirement,

no such toxicological or pharmacological tests on the products used for water fluoridation have been undertaken by the State or its agencies.

Common sense should suffice in preventing unnecessary risk to consumers, yet the ongoing systematic fluoridation of public water supplies without proof of safety poses extraordinarily inherent dangers for the public. In effect the State is conducting unmonitored clinical trials of a known toxin on an entire population over an extended period of time without their knowledge or consent. Naturally, if any unwelcome side-effects occur they are likely to be felt by tens of thousands of people, both young and old. While the policy of water fluoridation continues in Ireland it is politically, morally and ethically unacceptable that the public and ultimate consumers of drinking water still await the commencement of comprehensive epidemiology, toxicology, clinical medicine and environmental exposure assessments. This is entirely unacceptable.

There is an obligation by public health services to assess whether adequate information on the safety of fluoridation products is available. Where adequate information is not available (as is the situation), the State is required legally to enact the precautionary principle as defined in European law and international treaties.

The authorities should be aware that the WHO has recommended that where the risk for skeletal and dental fluorosis is high (as is the case for Ireland with approximately forty % of the population known to suffer from dental fluorosis), fluoride levels in drinking water should be reduced to safe levels, or a lower fluoride source used, especially for young children.

Parents and households in Ireland do not have access to non-fluoridated public water supplies to prepare infant formula or foodstuffs. The Government of Ireland intends to introduce a household charge and a water service charge for all households. Notwithstanding the numerous violations in EU law resulting from fluoridation of drinking water the Government of Ireland has a moral and legal obligation to provide non-fluoridated water to households in the interests of public health and safety, otherwise it is inconceivable that the State would charge households for the safe supply of drinking water.

As part of the provision of 'safe' drinking water, it is particularly disturbing that despite the numerous health concerns regarding systemic exposure of the population to silicofluorides in drinking water, no risk assessment has ever been undertaken on the commercial grades of silicofluorides used in water treatment in Ireland. Alarmingly this lack of accurate scientific information on risk assessments has been noted in all scientific reviews of fluoridation. The question must then be asked as to how the policy of water fluoridation can continue without accountability and proper review?

The Government of Ireland has ratified international treaties upholding the precautionary principle for environmental risk management. The precautionary principle forms the basis of both European law and international treaties. It must be upheld particularly when the general health and wellbeing, both mentally and physically, of an entire nation's population

may be unwittingly at risk from exposure to a known toxin that is added to public water supplies. The information contained in this report is taken from respected, verifiable and reliable peer-reviewed scientitic journals of international standing and must be acknowledged and immediately acted upon to protect the public interest, above all else the health and safety of the public.

Given the scientific evidence presented in this report the government of Ireland or its agencies cannot ethically or morally continue to support a policy that is lacking so clearly in intellegence, judgement and professional due diligence. To continue to support such a policy, in light of the information contained in this report, would represent gross culpable negligence.
Even the most rational person would agree that, given the potential risks and potent health hazards associated with water fluoridation, the small likelihood of any perceived benefit cannot be used as a basis for placing the entire population at risk to compounds that have not been thoroughly tested for their toxicity in humans.

Currently, despite the numerous recommendations from scientific bodies that efforts be made to determine the toxicity of fluoride and silicafluoride products, there is no information available on the mutagenic, teratogenic, developmental toxicity, cytotoxicity, carcinogenic effects, cogenotoxicity, short-term and sub-chronic exposures or synergistic/antagonistic effects of fluoride or Hexafluorosilicic acid on human beings.

Clearly therefore the only sensible, pragmatic, scientific, moral and legal approach would be to end the policy of water fluoridation immediately.

3.0 FLUOROSILICICTES AND DRINKING WATER

It is now known that the policy of fluoridation of drinking water supplies is unsafe and places an unacceptable health burden and unsustainable environmental impact on the environment. Ireland is the only EU country with a legislative policy for the addition of fluoride compounds to public water supplies. In all other European countries alternative vehicles for fluoride exposure, such as toothpaste, are the accepted practice. According to the WHO[1], where a population has a high level of dental awareness and uses toothpaste there is no need to supply fluoridated water.

Water fluoridation is the addition of a fluoride compound to drinking water supplies in order to increase its fluoride concentration. In Ireland public water supplies are fluoridated with fluorosilicic acid (also known as hydrofluorosilicic acid or hexafluorosilicic acid). The toxicology of hexafluorosilicic acid and hexafluorosilicictes compounds are incompletely investigated[2]. It is incorrect to assume that fluoride compounds used in water fluoridation dissociate entirely into fluoride ions, and harmless hydration compounds of silicon. In recent years, many studies have indicated that ingested fluoride induces free radical toxicity in humans and animals.[3],[4],[5],[6] From a toxicological perspective fluoride is regarded as more toxic than lead, and only slightly less toxic than arsenic.[7] Pure fluoride solutions do behave relatively predictably, both over the permissible pH range of municipal water supplies and in the extremely acidic environment of the human stomach. In contrast, fluorosilicictes dissociate in highly complex fashion in water, with a range of complex derivatives forming at different pH values.[8,9,10]

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[1] Nutrients in Drinking Water, Water, Sanitation and Health Protection and the Human Environment World Health Organization, Geneva, 2005.

[2] Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, Scientific Committee on Health and Environmental Risks, Director General for Health & Consumers 2010

[3] Kaushik T, Shyam R, Vats P, Suri S, Kumria MML, Sharma PC, et al. Glutathione metabolism in rats exposed to high-fluoride water and effect of spirulina treatment. Fluoride 2001;34:132-8.

[4] Guan ZZ, Xiao KQ, Zeng XY, Long YG, Cheng YH, Jiang SF, et al. Changed cellular membrane lipid composition and lipid peroxidation of kidney in rats with chronic fluorosis. Arch Toxicol 2000;74:602-8.

[5] Kalyanalakshmi P, Vijayabhaskar M, Naidu MD. Lipid peroxidation and antioxidant enzyme status of adult males with skeletal fluorosis in Andhra Pradesh, India. Fluoride 2007;40:42-5.

[6] Shivarajashankara YM, Shivashankara AR, Bhat PG, Rao SH. Effect of fluoride intoxication on lipid peroxidation and antioxidant systems in rats. Fluoride 2001;34:108-13.

[7] Williams and Williams (1984), Clinical Toxicology of Commercial Products, pplI-4, II-112, II-129

[8] Crosby NT; "Equilibria of Fluosilicate Solutions with Special Reference to The Fluoridation of Public Water Supplies"; *J Appl Chem*; v19; pp 100-102, 1969

[9] Busey RH et al; "Fluorosilicicte Equilibria in Sodium Chloride Solutions from 0 to 60 ◦ C"; Inorg. Chem V 19; pp 758-761, 1980.

Silicafluoride

When fluorosilicates are added to water they dissociate to form fluorosilicic ions [SiF6] 2- with two negative electrical charges, accompanied by either two individual ions of hydrogen H+ (from fluorosilicic acid) or of sodium (Na+) (from sodium fluorosilicates). The individual elements, silicon (Si) and fluorine (F) in the fluorosilicic ion cannot move independently - at neutral pH they act as the complex substance fluorosilicic. At around the normal pH of 7, approximately 97% of the fluorine in fluorosilicic added to the water is present in the form of ionised fluoride, F+.

At the very slightly acidic pH of 6, only 27% of the fluorine in fluorosilicates is present as fluoride - the rest is associated with other ions, and forms a number of complex and unstable compounds and ions that change over variable periods of time and at different pH values. At the acidity of the human stomach - pH2 to 3 - the proportion of fluorine atoms that are present as fluoride ions changes dramatically and effectively no fluorine atoms are present in the ionic state. Research[11] undertaken by Dr. Johannes Westendorf, (Toxicology Department, Eppendorf-Hamburg University Hospital) found that under physiological conditions, dissociation of silicafluorides was no more than 66% in the concentration range considered optimum for fluoridated water.

Westendorf concluded, based on actual laboratory experimental evidence, that dilution of fluorosilicic acid to a nominal 1 part per million of free fluoride in water at pH 7.4 induces each [SiF6]2- to release 4 fluorides to be replaced by hydroxyls. The partially dissociated residue would be the ion [SiF2(OH)4]2- which would then be present in the water at the same concentration as the originally introduced SiF. The findings of Westendorf were supported in a scientific paper by Milton[12] which observed that silicafluoricic acids contain, in addition to such primary silica, a secondary silica component with its own characteristic chemical behaviour. Because the composition of silica-saturated fluorosilicic acids approaches closely that of H2SiF6-SiF4 (fluorosilicic acid) for which S=1.2, the secondary silica will be considered to be present in this form.

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[10] Urbansky, E.T., and Schock, M.R.. Can fluoridation affect water lead levels and lead neurotoxicity? In: American Water Works Association Annual Conference Proceedings, Denver, CO, June 11-15, 2000

[11] Westendorf J. Die Kinetik der Acetylcholinesterasehemmung und die Beeinflussung der Permeabilitat von Erythrozxytenmembranen durch Fluroid und Fluorokomplex-Jonen. Doctoral Dissertation, Hamburg Universitat Hamburg Fachbereich Chemie, 1975.

[12] Thomsen, Milton S, High-silica fluosilicic acids : specific reactions and the equilibrium with silica, Am, Chem, Soc. 74 : 1690-1692

The presence of dissociated silica fluorosilicic acid compounds was further acknowledged in a paper[13] presented at the *International Fertiliser Association* technical committee meeting in 1999 which stated the following:

> *" The chemical formula of fluorosilicic acid is H2SiF6. However, things are not as simple as that due to the fact that rarely is fluorosilicic acid present as pure H2SiF6. . . There are well reported references to the existence of H2SiF6 SiF4. . . Hereon in this presentation, FSA [fluorosilicic acid] means a mixture of HF, H2SiF6 and H2SiF6 SiF4".* Meaning there is more than one ionic compound.

The dissociation of hexafluorosilicate has been investigated by Finney et al.[14] to determine if fluorosilicate intermediates may be present in appreciable concentrations in drinking water. No intermediates were observable at 10(-5)M concentrations under excess fluoride forcing conditions over the pH range of 3.5-5.

A single intermediate species, assigned as SiF5(-) or its hydrate, was detected below pH3.5. This research was limited as the full range of pH associated with gastric pH was not examined, however, it did importantly prove the existence of fluorosilicicte intermediates outside of *H2SiF6.*

The dissociation status of silicofluorides (SiF) depends on pH and its concentration in water so that incompletely dissociated SiF residues may re-associate both at intra-gastric pH around 2.0 [15] and during food preparation, producing SiF species including silicon tetrafluoride, (SiF4), a known toxin.[16,17,18,19,20,21]

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[13] Smith Paul A. Société Chimique Prayon-Rupel SA, Belgium, HISTORY OF FLUORINE RECOVERY PROCESSES, Paper presented at the IFA Technical Sub-Committee and Committee Meeting, 15-17 September 1999, Novgorod, Russia

[14] Finney WF, Wilson E, Callender A, Morris MD, Beck LW.
Re-examination of hexafluorosilicicte hydrolysis by 19F NMR and pH measurement. Environ Sci Technol. 2006 Apr 15;40(8):2572-7.

[15] Ciavatta L, et al; "Fluorosilicicte Equilibria in Acid Solution"; Polyhedron Vol 7 (18);1773-79;1988

[16] Gabovich RD; "Fluorine in Stomatology and Hygiene"; translated from the original Russian and published in Kazan (USSR); printed by the US Govt Printing Office on behalf of the Dept of Health Education and Welfare. US Public Health Service, National Institute of Dental Health; DHEW pub no (NIH) 78-785, 1977

[17] Roholm K; "Fluorine Intoxication; A Clinical-Hygiene Study"; H. K. Lewis & Co. Ltd, London; 1937

[18] Lewis RJ, jr.; "Hazardous Chemicals Desk Reference": Van Nostrand Reinhold; Fourth Edition.

[19] Matheson Gas Products; 30 Seaview Drive, Secaucus, NJ; "Effects of Exposure to Toxic Gases" and MSDS for CAS # 7783-61-1; created 1/24/89.

[20] Voltaix, Inc.; Material Safety Data Sheet for Silicon Tetrafluoride (SiF4).

[21] Rumyantseva GI et al; "Experimental Investigation of The Toxic Properties of Silicon Tetrafluoride"; *Gig Sanit* ;(5):31-33, 1991.

Of interest also is the finding by Taves et al.[22] who concluded that fluoride interacts with the silica, forming an aqueous fluorosilicicte in the vessel: "*Plasticware (Falcon Plastics) was used for all analytical procedures to avoid contamination by fluoride from glass.*" It is further acknowledged that commercial SiFs are likely to be contaminated with fluosiloxanes, [23] arsenic and heavy metals, [24] and radionuclides,[25] since they are waste products from fertilizer manufacture and uranium extraction from phosphate rock.[26],[27],[28],[29]

Alarmingly, it is acknowledged that while the toxicological properties of silicafluorides or their derivatives have not been investigated[30],[31],[32], they continue to be used as medical intervention additives in drinking water supplies. No short-term or sub-chronic exposure, chronic exposure, cytotoxicity, reproductive toxicity, teratology, carcinogenicity, or initiation/promotion studies are available. This is particularly alarming since fluorosilicicte compounds were used as pesticides in the USA till 1999.

In August 1995, the act was amended, eliminating fluorosilicate compounds from the registration list and their sale for pesticide use (40CFR153, Subpart H) (U.S. EPA, 1995). In the United States, all pesticide uses have been cancelled (U.S. EPA, 1999). Fluorosilicicte acid is listed in Section 8(b) of the USA Toxic Substances Control Act (TSCA; chemical inventory section).

There is concern regarding the health safety of SiFs on a number of counts:

- possible formation of toxic complexes with aluminium, iron and other cations commonly present in treated drinking water; fluorosilicictes are particularly likely to react with aluminium compounds Al(OH)3 used in water treatment to produce several derivative compounds.

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[22] Guy WS, Taves DR, Brey WS, Organic Fluorocompounds in Human Plasma: Prevalence and Characterization, ACS symposium series, Washington, Symposium Series, No. 28 117 - 134.
[23] Ricks GM et al; "The Possible Formation of Hydrogen Fluoride from the Reaction of Silicon Tetrafluoride with Humid Air": *Am. Ind. Hyg. Assoc. J.* (54); 272-276, 1993.
[24] Craig JM; "Fluoride Removal from Wet-Process Phosphoric Acid Reactor Gases"; Ph. D. Dissertation; Univ. Fla. at Gainseville, 1970.
[25] Murray RL; "Understanding Radioactive Waste"; Third Ed.(ed Powell JD); 1982
[26] Becker Pierre; "Phosphates and Phosphoric Acid: Raw materials, technology, and economics of the wet process"; Marcel Dekker: New York (First ed.) 1983, Second ed., 1988.
[27] Slack AV; "Phosphoric Acid"; Part I; Marcel Dekker: New York, 1968.
[28] Greek BF, Allen OW, Tynan DE; "Uranium Recovery from Wet Process Phosphoric Acid"; *Industrial & Engineering Chemistry*; vol 49 (4); 628-636, 669-671, 1957.
[29] Rahn FJ et al; "A Guide to Nuclear Power Technology"; John Wiley & Sons' New York; 1984
[30] Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water; Scientific Committee on Health and Environmental Risks, Director General for Health & Consumers (2010).
[31] U.S National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C.
[32] The Use of Data on Cholinesterase Inhibition for Risk Assessments of Organophosphorous and Carbamate Pesticides, Office of Pesticide Programs US Environmental Protection Agency, August 18, 2000.

- potential toxic effects from SiF dissociation residues in municipal drinking water that may be present.
- the environmental impact of SiF on freshwater and marine ecosystems has not been properly investigated.
- the biological and human health consequences of ingesting such a species have never been properly investigated.

The only extensive examination of the actual biochemical effects of SiF that has been encountered is the aforementioned German study[33] which shows substantial changes in membrane permeability and enzymatic changes capable of substantially modifying neuronal excitability. Westendorf's results showed that fluoride in the form of the silicafluoride complex (SiF), as well as several other complexes, was a substantially more powerful inhibitor of cholinesterase than the simple fluoride ion released by sodium fluoride (NaF).

The inhibition of cholinesterase by silicafluorides is not unusual as it has been widely scientifically documented that fluoride is capable of inhibiting a number of enzymes, including pre-glycolytic enzymes, phosphatases, and cholinesterase.[34]

These finding are of staggering significance as:

- cholinesterase is one of many important enzymes needed for the proper functioning of the nervous systems of humans, other vertebrates, and insects.
- glycolytic enzymes have been found to play a central role in dementia[35] and
- phosphatases metabolism plays a critical role in cellular function, physiology and disease, including allergy, asthma, obesity, myocardial hypertrophy, and Alzheimer's disease. [36],[37]

Cholinergic pathways innervate virtually every organ in the body, including the brain and peripheral nervous system. Acetylcholine plays an important role in the functioning of the nervous system. Acetylcholine (AChE) is a neurotransmitter which enables chemical communication to occur between a nerve cell and a target cell. This target cell may be another nerve cell, muscle fiber or gland.[38] Inhibition of AChE leads to an accumulation of acetylcholine and a prolongation of the action of acetylcholine at the nerve-

[33] Westendorf J. Die Kinetik der Acetylcholinesterasehemmung und die Beeinflussung der Permeabilitat von Erythrozxytenmembranen durch Fluroid und Fluorokomplex-Jonen. Doctoral Dissertation, Hamburg Universitat Hamburg Fachbereich Chemie, 1975.

[34] United States National Library of Medicine, Hazardous substances databank

[35] Iwangoff, P, Armbruster,R, Enz,A, Ruge W. M, Glycolytic enzymes from human autoptic brain cortex: Normal aged and demented cases. Mechanisms of Ageing and Development, Volume 14, Issues 1–2, September–October 1980, Pages 203–209

[36] Hong Zheng, Shawn Alter, and Cheng-Kui Qu, SHP-2 tyrosine phosphatase in human diseases Int J Clin Exp Med. 2009; 2(1): 17–25.

[37] Bottini N, Bottini E, Gloria-Bottini F, Mustelin T. Low-molecular-weight protein tyrosine phosphatase and human disease: in search of biochemical mechanisms. Arch Immunol Ther Exp (Warsz). 2002;50(2):95-104.

[38] Dementi, B. "Cholinesterase Literature Review and Comment"; Pesticides, People and Nature; 1 (2); 59-126; 1999

nerve, nerve-muscle or nerve-gland interface. Peripherally, the accumulation of acetylcholine can result in cholinergic responses such as smooth muscle contractions (e.g., abdominal cramps), glandular secretions (e.g., sweating), skeletal muscle twitching, and, at higher concentrations, flaccid paralysis. In addition, there may be centrally mediated effects on learning, memory and other behavioral parameters. Thus, the inhibition of AChE potentially results in a broad range of adverse effects, having an impact on most bodily functions, and depending on the magnitude and half-life of an exposure dose, these effects can be serious, even fatal.

Little information exists describing effects following long(er)-term, low-level exposures to humans to anticholinesterase substances. Fluorinated organic compounds have widespread applications as pesticides, herbicides, pharmaceuticals, flame-retardants, refrigerants and foam-blowing agents, and are consequently accumulating in the environment.

Populations that are at increased risk from the effects of fluoride are infants who are bottle-fed formula milk reconstituted with fluoridated water and individuals that suffer from diabetes insipidus or some forms of renal impairment.

The potential human health hazards and social costs of injecting fluoride or silicafluoride compounds into drinking water are staggering. The fact that fluoride is known to be an enzymatic poison of critical metabolic pathways clearly demonstrates that these compounds should never be added to drinking water. These findings have very significant implications for the Health Service Executive, Local Authorities and EPA with responsibility for the provision of safe drinking water. Astonishingly, no human hazard identifications studies have ever been undertaken on silicofluoride compounds. The EPA, as the overal responsible regulatory authority for drinking water quality in Ireland, must address the potential effects of silicafluoride compounds and the completeness (or lack thereof) of databases with respect to their toxicity and exposure. In the interim, there is no justification in any circumstances to continue with a policy of water fluoridation that could further endager the health and wellbeing of consumers.

Apart from the possibility of direct toxicity the dissociated fluoride ions are known to bind to calcium and magnesium. If diets are low in calcium or magnesium the products of silicofluoride dissociation can exacerbate the competition between calcium and lead for bone and soft tissue sites. In addition, fluoride has a high affinity for proteins[39] and is known to modify

[39] Emsley, J., Jones, D.J., Miller, J.M, Overill, R.E., and Waddilove, RA. An unexpectedly strong hydrogen bond: Ab initio calculations and spectroscopic studies of amide-fluoride systems. I. Am. Chem. Soc. 1981: 203;24-28

enzyme action[40],[41] with potential for disrupting a wide range of endocrine, immune and neural processes. Fluoride has been implicated in disturbing the functionality of calcium, both directly by the U.S. ATSDR[42] and indirectly in interaction with Vitamin D.[43]

Of the very limited research that has been undertaken on silicofluorides on animals one study[44] undertaken by the Ohio Agricultural Department in 1935 demonstrated that silicofluorides were significantly more bio-available than other forms of fluoride.

A third relevant study by Monacha et al.[45] conducted around the same time as Westendorf's research, involved feeding water treated with the same fluosilicic acid used to fluoridate the local water supply to squirrel monkeys for up to 14 months. Morphological and cytochemical effects were reported for the liver, kidney and nervous system due to ingestion of 1-5 ppm of fluoride in water. The report later observes that work by others in the 1940's and 1950's "*showed that fluoride has an inhibitive effect on the activity of succinate dehydrogenase. These studies indicate that under the effect of fluoride intake, a serious metabolic distress may develop in the kidneys.*" In concluding, Manocha et al. indicated that inorganic fluorides have a strongly adverse effect on the activity of some enzymes and of these, mitochondrial enzymes, acid and alkaline phosphatases and ATP-utilizing enzymes and aldolase may be the most affected.[46],[47]

The research on Silicofluorides (SiFs), Neurotoxicity, and Behavior[48] by Professor Roger D. Masters and Coplan M.J published in the journal *Neurotoxicology* provides, however, a comprehensive examination of the chemical and biological factors showing association of silicofluoride water treatment

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[40] Westendorf J. Die Kinetik der Acetylcholinesterasehemmung und die Beeinflussung der Permeabilitat von Erythrozxytenmembranen durch Fluroid und Fluorokomplex-Jonen. Doctoral Dissertation, Hamburg Universitat Hamburg Fachbereich Chemie, 1975.

[41] Padmanabhan J and Shelanski ML,. process Formation In Astrocytes: Modulation Of Cytoskeletal Proteins. Neurochem. Res. 1998 Mar; 23(3):377-84

[42] ATSDR, Toxicologial Profile for fluorides, Hydrogen Fluoride, and Fluorine (F) Wastington: US. Department of Health and Human Services (TP-91/17), 1993

[43] Bayley TA, Harrison JE, Murra VM, Josse RG, Sturtridge w, Pritzker KP, Strauss a, Vieth R, Goodwin s. Fluoride-induced fractures: Relation to osteogenic effect: J Bone Miner Res. 1990 Mar; 5 Suppl 1:S217-22.

[44] Kick CH, et al. "Fluorine in Animal Nutrition"; Bulletin 558, Ohio Agricultural Experiment Station; Wooster, Ohio; November 1935; pp 1-77.

[45] Manocha SL, et al. "Cytochemical response of kidney, liver and nervous system to fluoride ions in drinking water"; Histochemical Journal, 7 (1975); 343-355.

[46] Batenburg, J.J., Van Den Bergh S.G.,The mechanism of inhibition by fluoride of fatty acid oxidation in uncoupled mitochondria, Biochimica et Biophysica Acta (BBA) - Lipids and Lipid Metabolism. Volume 316, Issue 2, 23 August 1973, Pages 136–142

[47] Katz S, Tenenhouse A. The relation of adenyl cyclase to the activity of other ATP utilizing enzymes and phosphodiesterase in preparations of rat brain; mechanism of stimulation of cyclic AMP accumulation by NaF. Br J Pharmacol. 1973 Jul;48(3):505-15.

[48] Coplan MJ, Patch SC, Bachman MS- Confirmation of and explanations for elevated blood lead and other disorders in children exposed to water disinfection and fluoridation chemicals. Neuortoxicology,2007Sep;28(5):1032-42

(SiFW)with increased absorption of lead and other harmful effects on health and behaviour in children.

Interestingly, a limiting factor that assists in controlling the absorption of toxic substances such as lead from the digestive system into the blood is the concentration of calcium and magnesium.[49],[50],[51],[52],[53],[54] Although this protective effect is limited, it should not be dismissed. Populations supplied with low-mineral water may be at a higher risk in terms of adverse effects from exposure to toxic substances compared to populations supplied with water of average mineralization and hardness.

This is of concern to some communities in Ireland where lead piping is still being used for drinking water. According to the EPA's latest report[55] on water supplies, the agency has warned that there is still a real health risk from the remaining lead water mains around Ennis, Co Clare; Mallow, Co Cork; Lough Guitane, Co Kerry and central Co Longford. In total roughly 5,500 metres of lead piping is still being used for drinking water in these four areas.

In addition to the exposure risk posed by lead piping for public water supplies there remains a significant proportion of houses built before 1970 that could still have internal lead service pipes in them. As noted by Copland et al. the presence of SiFW is associated with serious corrosion of lead-bearing brass plumbing, producing elevated water lead (PbW) at the faucet with obvious implications for human health.[56]

Contrary to previous understanding it has been reported and demonstrated in laboratory experiments that small amounts of incompletely dissociated [SiF6]2- or low molecular weight (LMW) silicic acid (SA) oligomers may remain in SiFW following injection of SiFs into drinking water.

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[49] Thompson, D.J. (1970) *Trace element in animal nutrition*. 3rd ed. Int. Minerals and Chem. Corp., Illinois.

[50] Levander, O.A. (1977). Nutritional factors in relation to heavy metal toxicants. *Fed. Proc.* 36, 1683-1687.

[51] Hopps, H.C. and Feder, G.L. (1986) Chemical qualities of water that contribute to human health in a positive way. *Sci. Total Environ.* 54, 207-216.

[52] Nadeenko, V.G., Lenchenko, V.G. and Krasovskii, G.N. (1987) Combined effect of metals during their intake with drinking water. (In Russian.) *Gig. Sanit.* No.12 /1987 (volume not given), 9-12.

[53] Durlach, J., Bara, M. and Guiet-Bara, A. (1989) Magnesium level in drinking water: its importance in cardiovascular risk. In *Magnesium in Health and Disease* (ed. Y.Itokawa and J.Durlach), pp. 173-182, J.Libbey & Co Ltd, London.

[54] Plitman, S.I., Novikov, Yu.V., Tulakina, N.V., Metelskaya, G.N., Kochetkova, T.A. and Khvastunov, R.M. (1989) On the issue of correction of hygenic standards with account of drinking water hardness. (In Russian.) *Gig. Sanit.* No. 7/1989 7-10.

[55] Office of Environmental Enforcement, The Provision and Quality of Drinking Water in Ireland, A Report for the Year 2010, Environmental Protection Agency

[56] Coplan MJ, Patch SC, Masters RD, Bachman MS, Confirmation of and explanations for elevated blood lead and other disorders in children exposed to water disinfection and fluoridation chemicals. Neurotoxicology. 2007 Sep;28(5):1032-42. Epub 2007 Mar 1.

Their existence and the health risk of exposure to SiFc has been acknowledged by the U.S. EPA who tendered for risk management research to be undertaken on fluorosilicates in drinking water in 2002 and again by the U.S. National Research Council who requested animal testing on the health effects of SiFc in 2006.

In the knowledge that no human or animal health risk assessments have ever been completed on silicofluorides, the continued injection of these products into public drinking water supplies poses an unacceptable risk to public health. This risk would be increased for individuals living in dwellings who may be unaware of the existence of lead pipes within their household water system.

It is alarming and unexplainable therefore how the biological or toxicological impacts have never been fully examined and that the State continues to use such products without undertaking the necessary precautions for public health and the environment. Common sense suggests that wide-spread, albeit clinically vague, adverse health effects should be expected when a strong enzyme inhibitor is added to the daily diets of every consumer of public water supplies in Ireland. Given the paucity of direct knowledge about bio-mechanisms depending on exposure to commercial silicofluorides, and the magnitude of the potential risks to all sectors of society but especially infants and the elderly – large-scale epidemiological studies, chemical analyses and animal experimentation on silicofluorides and their effects deserve the highest priority. In the absence of such data the use of silicofluorides must end immediately to protect public health and consumers.

The significance of any association between silicofluoride compounds and increased bioavailability and toxicity of fluoride, its cytochemical effects on the body and interactivity with other compounds, in particular aluminium, combined with its documented effect as an inhibitor of cholinesterases and other metabolic pathways cannot be underestimated. These findings are of major significance and demand in the interests of public health and safety an immediate response from the State requiring the cessation of the water fluoridation policy without delay.

4.0 ENVIRONMENTAL FATE OF FLUORIDE

The environmental abundance of inorganic fluoride is increasing, because it is released into the environment in wastewater effluents through water fluoridation, wastes from sewage and sludges, use of dental and pharmaceutical products as well as industrial processes such as the production of aluminium fluoride and use of phosphatic fertilizers.[57]

An examination of the lifecycle of fluoride is beyond the remit of this report and would require extensive study; nevertheless some aspects have already been dealt with in previous sections of this report. In general, very little if any data is available for Ireland examining the environmental impact of fluoride on the environment. This is despite over 40 years of fluoridation and the direct and indirect discharge of significant quantities of fluoride as an anthropogenic contaminant and pollutant.

It is generally accepted that the toxicology of hexafluorosilicic acid and hexafluorosilicictes compounds are incompletely investigated.[58] In Ireland public water supplies are fluoridated with hydrofluorosilicic acid, which is harmful to aquatic life at low concentrations.[59]

As discussed in previous sections, neither risk assessment nor environmental impact assessments were ever undertaken on the impact of these compounds on either human health or the environment. Despite this the Forum for Fluoridation, a body representing an expert panel of consultants in Ireland, came to the conclusion that "*the effects of fluoride on the general environment and on the aquatic environment in particular are imperceptible*".[60]

It is rather astonishing that in the absence of conducting or evaluating field surveys or environmental/ecological assessments for damage by fluoride to animals, plants, and vegetation and in the absence of bio-monitoring for fluoride contamination or determining the amounts of fluoride in air, soil, vegetation and water, that the Forum for Fluoridation could come to any such conclusion.

The policy of water fluoridation in Ireland requires some 1,600 million litres of water to be dosed daily with hydrofluorosilicic acid.[61] The Department of Environment have estimated that the level of leakage in the water systems

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[57] Jha, S. K, Mishra K.V, Sharma K,D, Damodaran T, Fluoride in the Environment and its metabolism in Humans. Reviews of Environmental Contamination and Toxicology, Volume 211, March 2007. Springer-Verlag New York Inc.

[58] Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, Scientific Committee on Health and Environmental Risks, Director General for Health & Consumers (2010

[59] Material Safety Data Sheet Hydrofluosilicic Acid

[60] Forum on Fluoridation 2002, Appendix 10, Fluoride and the Aquatic Environment.

[61] Forum on Fluoridation Report 2002.

varies from 16.8 to 58.6pc[62] with hundreds of millions of gallons of fluoridated water being lost both in and on the ground. This results in the indirect release of the pollutant fluoride into soils, groundwater and surface waters.

Therefore approximately 4500kg of fluoride is discharged every day through leakage alone, representing 164,000kg of fluoride discharged to the environment including groundwater annually. With regard to the remainder of water treated comprising some 600 millions litres, 1.971000kg (1,971 tonnes) of fluoride is added to it annually.[63] Approximately 10,643kg of the total amount is used in drinking water by households and 1,960,357kg ends up as household or industrial wastewater from toilets, washing clothes etc. This enormous environmental load of fluoride is collected in wastewater treatment plants and ultimately ends up being discharged as a pollutant directly to surface water and the environment.

The EPA has also documented that fluoride binds strongly to sediment and bioaccumulates in the environment.[64] The Irish EPA have acknowledged and reported that major anthropogenic sources of fluoride include fluoridation of public water supplies, leakage of mains water from the drinking water distribution system and municipal wastewater treatment plants.[65] In the latter report the Irish EPA have also acknowledged that potential waters at risk from fluoride pollutant include receiving waters located downstream of both drinking- and waste-water treatment plants and areas where there is significant leakage from the drinking water distribution system.

The Agency further reported a number of exceedances of the standard for fluoride associated with the infiltration of drinking water into surface waters as well as leaking drinking water distribution mains into groundwater aquifers which the agency regarded as a significant potential source of fluoride (as drinking water contains fluoride at levels of between 800 and 1000 µg/l particularly in urban areas).

Remarkably approximately half of one % of the fluoride added to drinking water ends up being used for the purpose it was intended. The remainder is discharged as household and industrial wastewater into the public sewer. It is astonishing that approximately 99.45% of the fluoridated water is not used for the purpose for which the policy is undertaken.[66]

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[62] The Irish Times - Saturday, January 8, 2011

[63] Forum on Fluoridation Report 2002

[64] McCarthy, T., Duggan, S., McCarthy J., Lambe, A. Regulatory Impact Analysis of the proposed Surface Water Classification Systems including Environmental Quality Standards Final Report, Environmental Protection Agency December 2007

[65] Clenaghan, C,. O'Neill N, Page, D., Dangerous Substances Regulations National Implementation Report, 2005 Under the Water Quality (Dangerous Substances) Regulations, 2001 (S.I. No. 12 of 2001), Environmental Protection Agency, 2006.

[66] Commission on Health and Social Issues (GSK) GSK - "Bericht der Gesundheits-und Sozialkomission des Grossen Rates zum Anzug Rene Brigger betreffend Fluoridierung des Basler Trinkwassers" [9229/P975485]. 2003 Report which led to the cessation of water fluoridation in Basel.

This must represent one of the most ineffective and unsustainable water management policies of any country globally. Over the period of water fluoridation in Ireland the total amount of human-contributed artificial sources of fluoride discharged into the environment including rivers, streams and soils from water fluoridation would approximately be an astonishing 78,400,000 kgs or 78,400 tonnes of fluoride.

In the age of sustainability, with ever-growing pressures on the environment, such a policy is completely unacceptable and should in the 21st century be against the ethics and environmental principles of any and all modern educated democratic states.

From a lifecycle analysis the end result of such a dangerous policy for the environment is that an environmental load of almost two million kg of fluoride is dumped on the environment annually. This is undertaken without any risk assessment of the potential environmental or ecological impacts in the full knowledge that fluoride bioaccumulates in sediments, soil and the environment. The environmental implications of this are reviewed in the legal framework chapter of this report.

4.1 Fluoride in Surface Water and Sewer Systems

In a recent study[67] undertaken by Basel's Health & Social Commission (GSK) on fluoridation, they observed that "over 99 % of the fluoride" added to water is never consumed by humans, leading in turn to "an unnecessary load on the environment." According to GSK's president, Dr Jurg Merz, *"more than 99% of the water is not drinking water but is used for washing cars, cleaning stairs, showering, to pour on flowers and so on. Fluoride is poison that loads our rivers."*

In fluoridated areas, drinking water, obtained from surface water with an average fluoride concentration of 0.1-0.2 mg/L[68], is raised to the "optimal" level of 0.8-1.5 mgF/L by the addition of hydrofluosilicic acid. Fluoride added to drinking water "to improve dental health" enters the freshwater ecosystem in various ways. Surface run-off from fire-fighting, washing cars and watering gardens may enter streams directly or through storm sewers at optimal concentration, 0.7-1.2 mgF/L. Most enters during wastewater treatment. Consequently the fluoride added to protect dental decay ends up in the aquatic ecosystem. No environmental impact assessment has ever been undertaken examining the potential impacts of fluoride in wastewater discharges on surface water ecosystems. Nor have any field studies ever been undertaken to examine the effect of fluoride on salmon or other species in Ireland.

While fluoride is a normal constituent of surface water samples, it is generally

[67] GSK - "Bericht der Gesundheits-und Sozialkomission des Grossen Rates zum Anzug Rene Brigger betreffend Fluoridierung des Basler Trinkwassers" [9229/P975485].
[68] Carpenter R. Factors controlling the marine geochemistry of fluorine. *Geochemical et Cosmochimica Acta 33* 1153-1167 1969.

observed at concentrations less than 0.2mg/l in natural waters. In countries that fluoridate water, wastewaters coming from homes and industries contain elevated levels of fluoride. Normal primary and secondary wastewater treatment processes are not effective at removing fluoride to background levels.[69,70,71] It has been reported[72] that as a water pollutant, elevated concentrations of fluoride may affect a number of organisms, including fish, amphibians, insects, snails, shellfish, protozoa and some aquatic plants.[73] As with any pollutant, the State has a requirement to prevent its adverse impacts on surface water quality in accordance with the Water Framework Directive. As noted in S.I. 272 of 2009, the State must take measures to eliminate pollution of surface waters by priority substances and to progressively reduce pollution by other substances which would otherwise prevent achievement of the environmental objectives established by these regulations.[74]

The Environmental Quality Standard for fluoride in inland surface waters is set at 0.5mg/L. The regulations do not list fluoride as a priority substance, however given the evidence provided in the NRC review and Harvard studies as well as other data reviewed in this report, including information presented by the U.S. EPA officials on the mutagenicity of fluoride supporting the conclusion that fluoride is a probable human carcinogen, it is possible this may change in the near future.

The concentrations of fluoride in wastewaters for a large number of U.S. cities have previously been examined by Masuda[75] and it was demonstrated that the concentrations in wastewaters were in excess of the concentration in the city's fluoridated water supplies. Further studies by Singer and Armstrong[76] found 0.38 mgF/L in non-fluoridated sewage and 1.16-1.25 mgF/L fluoridated sewage. It has been observed that, in the case of artificially fluoridated communities the concentration of fluoride in both surface run-off and sewer effluent exceeds 0.2 mgF/L. International studies have shown that elevated concentrations in freshwater systems receiving fluoridated effluent may persist for some distance downstream within the river system.

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[69] Benefield LD, Judkins JF, Weand BL: Process Chemistry for water and waste water treatment. Englewood Cliffs, NJ: Prentice-Hall, 405-421. (1982)

[70] Link W E, Rabosky JG: Fluoride ion removal from waste water employing calcium precipitation and iron salt coagulation. Lafayette, IN: Purdue University, 31st annual Purdue Industrial waste Conference, May 4-6, (1976).

[71] Masuda TT: Persistence of fluoride from organic in waste waters. Devel Industry Microbial; 5: 53-70. (1964)

[72] Kushwah et al. Int. J. Res. Chem. Environ. Vol. 1 Issue 2 Oct. 2011(169-172)

[73] Water Quality Planning Branch, Division of Environmental Management: North Carolina Water Quality Standards Documentation: Toxicity of Fluoride to Freshwater Biota. Raleigh: North Carolina Department of Natural Resources and Community Development, Report No. 86-01, (1986).

[74] European Communities Environmental Objectives Regulations 2009- Surface waters

[75] Masuda TT. Persistence of fluoride from organic origins in waste waters. *Developments in Industrial Microbiology 5* 53-70 1964.

[76] Singer L. Armstrong WD. Fluoride in treated sewage and in rain and snow. *Archives of Environmental Health 32* 21-23 1977.

One such study[77] by Bahls showed that the effluent from Bozeman, Montana of 0.6-2.0 mgF/L, discharged into the East Galletin River did not return to the background level of 0.33 mgF/L for 5.3 km. Though dilution reduces concentration over distance, the amount of fluoride in effluent is either deposited in sediment locally or is carried to the estuary where it may persist for 1-2 million years[78] or may re-contaminate if dredging were to take place. Decreases in water volume and/or flow velocity have the potential to increase fluoride concentration. Increased water temperature will enhance fluoride toxicity. Very little research has been undertaken to examine the impact of fluoride on aquatic systems, in particular how artificially fluoridated wastewater may impact on fisheries or ecosystems. However, from what information is available, it would appear that fluoride may have an impact on fisheries and that meeting the requirements of the Water Framework Directive may not be possible as long as fluoridation of drinking waters continues.

4.2 Fluoride and Fisheries

Hydrofluorosilicic acid is harmful to aquatic life at low concentrations.[79] It has also been found that fluoride is an endocrine disruptor in the aquatic environment.[80]

If behaviour-altering pollutants such as fluoride are present in critical concentrations, it is likely that the migrating adult salmonids would respond to them in a short time-frame. [81] A large body of evidence shows that salmonids have an acute sense of smell with a low threshold value for many chemicals.[82],[83],[84]

Pollutants may cause avoidance or preference, overwhelm biologically relevant odours or damage chemoreceptive mechanisms. Complete avoidance of pollutants may prevent deleterious exposures.

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[77] Bahls LL. Diatom community response to primary waste water effluent *Journal Water Pollution Control Federation 45* 134-144 1973.

[78] Carpenter R. Factors controlling the marine geochemistry of fluorine. Geochemical et Cosmochimica Acta 33 1153-1167 1969.

[79] Material Safety Data Sheet Hydrofluosilicic Acid

[80] USA National Research Council, Fluoride in Drinking Water: A Scientific Review of EPA's Standards, Committee on Fluoride in Drinking Water, (2006)

[81] Damkaer DM, and Dey DB 1989. Evidence for fluoride effects on salmon passage at John Day Dam, Columbia River, 1982-1986. *N. Am. J. Fish. Manage*.9:154162 Coastal Zone and Estuarine Studies.

[82] Brown, S.B., R.E. Evans, B.E. Thompson, and T.J. Hara.1982. Chemoreception and aquatic pollutants, p. 363-393. In T.J. Hara (ed.) Chemoreception in fishes. Amsterdam. Elsevier.

[83] Cooper, J.C., and P.J. Hirsch.1982. The role of chemoreception in salmonid homing, p. 343-362

[84] Kleerekoper, H. 1982. The role of olfaction in the orientation of fishes, p. 201-225. In T.J. Hara (ed.) Chemoreception in fishes. Amsterdam. Elsevier.

It is reported that serious hazards to fish could arise through unperceived or unavoidable low level pollutants, in particular altering predator or food detection, reproduction or migration. Fluorides are known to be toxic to trout and other fish.[85]

The median toxic limits (concentration required to kill 50% of the test fish) in trout have been reported between 2.3 and 7.5ppm fluoride.[86,87] While there is very little information regarding the effect of fluorides on fish behaviour, however, there is evidence to suggest that fluorides are known to be enzyme inhibitors and could, therefore, have the potential to reduce activity at sub-lethal concentrations. Limited studies[88] have been undertaken that indicate damage to salmon and fisheries including plant ecosystems from fluoride. A number of studies[89],[90],[91] point out that many factors influence susceptibility of fish to fluoride: temperature; water hardness; pH; chloride concentration; and, the strain, age and physiological and reproductive condition of the fish. Warrington undertook research in British Columbia, where the softness of major salmonid watercourses is the rule, combined the findings of Angelovic, and others to calculate that the chronic threshold for rainbow trout at 12 degrees and water hardness of 10 mg/L (calcium carbonate) is 0.2 mgF/L.

In the field study by Damkaer and Dey[92] it was demonstrated that high salmon loss was caused by the inhibition of migration by fluoride contamination. Damkaer and Dey confirmed the cause-and-effect relationship by means of a two-choice flume for fluoride gradient salmon behaviour tests. The study determined that the "critical level" was 0.2 mgF/L. Damner further reported that there were also significant alterations in levels of blood-T 4 in smolting juveniles kept in fluoride concentrations of 0, 0.3, 0.5, and 1.0 ppm. With the juvenile rainbow/steelhead trout hybrids, there was an elevation of plasma T4 at 0.3 and 0.5 ppm fluoride but not at 1.0 ppm. T4 has been implicated in migratory behaviour of juvenile salmonids.[93]

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[85] Damkaer DM, and Dey DB 1989. Evidence for fluoride effects on salmon passage at John Day Dam, Columbia River, 1982-1986. *N. Am. J. Fish. Manage*.9:154162 Coastal Zone and Estuarine Studies.

86 Neuhold, J.M., and W.F. Sigler. 1960. Effects of sodium fluoride on carp and rainbow trout. Trans. Amer. Fish. Soc. 89(4):358-370.

[87] Angelovic, J.W., W.F. Sigler, and J.M. Neuhold. 1961. Temperature and fluorosis in rainbow trout. J. Water Pollution Control Federation 33:371.

[88] Damkaer DM, and Dey DB 1989. Evidence for fluoride effects on salmon passage at John Day Dam, Columbia River, 1982-1986. *N. Am. J. Fish. Manage*.9:154162 Coastal Zone and Estuarine Studies.

89 Groth III E. An evaluation of the potential for ecological damage by chronic low-level environmental pollution by fluoride. Fluoride 6 (4) 224-240 1975.

90 Warrington PD. Ambient Water Quality Criteria for Fluoride. Technical Appendix. British Columbia Ministry Of Environment. 1990.

[91] Angelovic JW, Sigler WF, Neuhold JM. Temperature and fluorosis in Rainbow trout. *Journal. Water Pollution Control Federation 33* 371-381 1961.

[92] Damkaer DM, and Dey DB 1989. Evidence for fluoride effects on salmon passage at John Day Dam, Columbia River, 1982-1986. *N. Am. J. Fish. Manage*.9:154162.

[93] Godin, J.G., P.A. Dill, and D.E. Drury. 1974. Effects of thyroid hormones on behavior of yearling Atlantic salmon (Salmo salar). J. Fish. Res. Board Can. 31:1787-1790.

It is very alarming that with over 240 water fluoridation plants located in every water catchment area within the country and over 478 wastewater treatment plants[94] discharging fluoride into freshwater, estuarine and coastal waters that the potential ecological impact of fluoride emissions has never been examined. It is not improbable to suggest that the impact may indeed be enormous. This is the case when it is known that over 78,400,000kgs of fluoride has been discharged into the environment from these facilities since fluoridation of water commenced in Ireland. Of this a significant percentage has been discharged directly into the 148 salmon rivers across the Republic of Ireland. It is perhaps not coincidental that the start of the decline in freshwater salmon fish stocks in Ireland mirrors exactly the commencement of water fluoridation. A similar decline has been observed in the USA where the practice of fluoridation also exists (though not as prevalent as Ireland). This decline constrasts with the generally healthy status of salmon in Canada where fluoridation is not practised.

There are other studies that indicate that fluoride at levels below 1.5 mg/L have lethal and other adverse effects on fish. Delayed hatching of rainbow trout[95] occurred at 1.5 mgF/L; brown mussels[96] died at 1.4 mgF/L; an alga (Porphyria tenera)[97] was killed by a four-hour fumigation with fluoride with a critical concentration of 0.9 mgF/L; and levels below 0.1 mgF/L were shown to be lethal to the water flea, Daphnia magna.[98] These latter two studies suggest that salmon species may be affected by fluoride-induced reduction of food supply.

The SCHER review's examinsation of the impact of fluoride on the marine environment was very simplistic and limited. The analysis of the aquatic effects was based on a bibliographic search and based much of its analysis on the review of fluoride toxicity to aquatic organisms by Camargo (2003).[99] Camargo observed that in aquatic animals, fluoride tends to be accumulated in the exoskeleton of invertebrates and in the bone tissue of fishes. The toxic action of fluoride resides in the fact that fluoride ions act as enzymatic poisons, inhibiting enzyme activity and, ultimately, interrupting metabolic processes such as glycolysis and synthesis of proteins.

Fluoride toxicity to aquatic invertebrates and fishes increases with increasing fluoride concentration, exposure time and water temperature, and decreases with increasing intraspecific body size and water content of calcium and chloride. Freshwater invertebrates and fishes, especially net-

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[94] Urban Waste Water Discharges in Ireland for Population Equivalents Greater than 500 Persons Environmental Protection Agency

[95] Ellis MM, Westfall BA, Ellis MD. *Determination of Water Quality Research Report 9.* Fish and Wildlife Service, Department of Interior, Washington DC 1938 pp 81-82.

[96] Hemens J: Warvick RJ, Oleff WD. Effect of extended exposure to low fluoride concentration on estuarine fish and crustacea. *Progress in water Technology 7* 579-

[97] Ishio S, Makagawa H (1971). Cited in: Rose D. Marier J. *Environmental Fluoride* 1977. National Research Council of Canada, Ottawa 1977 p 30.

[98] Dave G. Effects of fluoride on growth reproduction and survival in Daphnia magna. *Comparative Biochemistry and Physiology 78c* (2) 425-431 1984.

[99] Camargo JA (2003) Fluoride toxicity to aquatic organisms: a review. Chemosphere 50: 251-64

spinning caddis fly larvae and upstream-migrating adult salmons, appear to be more sensitive to fluoride toxicity than estuarine and marine animals. Because, in soft waters with low ionic content, a fluoride concentration as low as 0.5 mg F-/l can adversely affect invertebrates and fishes, safe levels below this fluoride/l concentration are recommended in order to protect freshwater animals from fluoride pollution.

Camargo further observed that aquatic organisms living in soft waters may be more adversely affected by fluoride pollution than those living in hard or seawaters because the bio-availability of fluoride ions is reduced with increasing water hardness. It was further found that fluoride can either inhibit or enhance the population growth of algae, depending upon fluoride concentration, exposure time and algal species.

It is clear therefore that much greater research is needed on the lifecycle impacts of fluoride on the environment; in particular any potential impacts on ecological receptors such as fisheries, given that 243 fluoridation plants are located in Ireland with many in sensitive fisheries catchment areas.

Additional research has demonstrated that fluorides released into marine environments have been shown to accumulate in some aquatic organisms.[100] It has been further demonstrated that foods characteristically high in fluoride content are certain types of seafood (1.9-28.5 mg/kg).[101] In a study by Hemens and Warwick[102] toxic effects due to fluorosis were observed in species of mussel, mullet, crab and shrimp in an estuary where waste from an aluminium plant was released.

Hemens and Warick also noted the apparent lack of information on bio-concentration in aquatic organisms and bio-magnification in food chains which the researchers observed would be helpful in assessing the importance of bioaccumulation of fluorides as a route of human exposure to this contaminant.

4.3 Fluoride as a Pollutant in Soil.

Fluoride is a pollutant that is present in sewage sludge and processed waterwater sludge where fluoridated waste has been used in the manafacturing process.[103]

Sewage and processed sludge has been and continues to be applied to land in Ireland. While the State has an obligation to protect public health and the environment from the anticipated adverse effects of this pollutant on the

[100] Toxicological Profile For Fluorides, Hydrogen Fluoride, And Fluorine, Agency for Toxic Substances and Disease Registry U.S. Public Health Service April 1993.
[101] Kumpulainen J, Koivistoinen P. 1977. Fluorine in foods. Residue Rev 68:37-55.
[102] Hemens J, Warwick RJ. 1972. Effects of fluoride on estuarine organisms. Water Res 6:1301-1308.
[103] U.S EPA Technical Support Document for the Round Two Sewage Sludge Pollutants, Office of Water, EPA-822-R-96-003, August 1996.

environment inadequate research has been undertaken to examine the potential impact of this pollutant on groundwater, surface waters and human health in Ireland. Implementation of the Water Framework Directive requires the highest levels of wastewater treatment, leading to greater pollutant removal from the wastewater before discharge and the generation of larger amounts of sewage sludge. The disposal of sewage sludge is undertaken but not limited to, land application, surface disposal and incineration.

The U.S. EPA undertook a hazard identification study[104] of pollutants that may cause adverse effects to public health and the environment. Based on the results of this study a list of 254 pollutants were identified and of these thirty one were then evaluated in a comprehensive hazard identification study. In their study, a quantitative risk assessment, including dose response evaluation, exposure assessment and risk characterisation was performed. The goal was to identify pollutants that may potentially cause human health or ecological risks for a highly exposed individual. Only pollutants that were deemed to be of risk to human health based on available human and ecological toxicity data were examined. Based on the results of the risk assessments of the comprehensive hazard identification, twelve pollutants were found to have critical exposure pathways. Fluoride was one of the principal pollutants.
This is extremely worrying for Ireland as the EPA have documented that over 120,000 tonnes of dried sludge is produced nationally from wastewater treatment plants annually, of which approximately eighty per cent is used for agriculture.[105] Despite the findings of the U.S. EPA, no risk assessment on the impact of fluorides on food production and human health has ever been undertaken in Ireland.

The aforementioned U.S. EPA study highlights the very serious limitations of the environmental assessment undertaken as part of SCHER review, given that SCHER concluded that fluorides would not be present in sewage sludge. The U.S. EPA peer-reviewed scientific study concluded that fluoride is a pollutant that is present in sewage sludge. The study observed that the 95 percentile sewage sludge concentrations for fluoride was 411mg/kg dry weight while background concentration of pollutants in soil were calculated at 220mg/kg, demonstrating that the concentration of fluoride in sewage sludge was twice that found in uncomtaninated soil.

The U.S. EPA examined the human exposure to pollutants through consumption of animals that ingested forage/pasture from sewage sludge amended soils. Evaluation of human exposure to fluoride must examine pollutant exposure pathways for humans ingesting groundwater with higher than the ambient concentrations of fluoride (due to contamination from leakage in fluoridated water supplies), individuals drinking artificially fluoridated drinking water, infants ingesting infant milk made up from fluoridated drinking water, eating food cooked or prepared using fluoridated water, drinking hot beverages using boiled fluoridated water and ingestion of plants or animals that have taken up pollutants (fluoride) from sewage sludge

[104] U.S EPA Technical Support Document for the Round Two Sewage Sludge Pollutants, Office of Water, EPA-822-R-96-003, August 1996.
[105] Urban Waste Water Discharges in Ireland for Population Equivalents Greater than 500 PersonsEnvironmental Protection Agency

amended agricultural soils and by ingestion of fish that may have bioaccumulated fluoride in their tissues.

As previously discussed fluoride accumulates in soils and surface water sediments by release of the pollutant through leakages in the water distribution system, effluent releases from wastewater plants and treatment and disposal of human wastewater sludge or food processing sludge. Wastewater discharges of fluoride are considerably above the background level in the environment.[106] No environmental or ecological studies examining the potential of these exposure pathways have ever been undertaken in Ireland. The toxicity of soil dwelling organisms, freshwater or marine organisms to fluoride pollution has also not been examined. It has been documented, however, that fluorides tend to persist in most soils.[107] Research has also shown that several species of plants have the capacity to convert soluble fluoride obtained from the soil into carbon-fluorine compounds such as monofluoroacetic acid, ω-fluoro-oleic acid, ω-fluoropalmitic acid, and ω-fluoromyristic acid.[108],[109]

4.4 Fluoride in our Food

As reported by the EU Scientific Panel on Dietetic Products, Nutrition and Allergies, there is no convincing evidence that health and development of humans depends on the intake of fluoride.[110]

It is now known that the systemic application of fluoride through injection into drinking water supplies may impair normal development of enamel in the pre-eruptive tooth and cause fluorosis.[111]

Fluoride intake from food is generally low except when food is prepared with fluoridated water. The fluoride content of the water used in industrial production and home-cooking affects the fluoride content of the prepared food. The use of water containing up to 1 ppm/L has been estimated to

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[106] EPA Technical Support Document for the Round Two Sewage Sludge Pollutants, Office of Water, EPA-822-R-96-003, August 1996

[107] Brewer RF, 1966. Fluorine. In: Chapman HD, ed. Diagnostic criteria for plants and soils. Riverside, CA: Division of Agricultural Science, University of California, 180-195.

[108] Marais JSC. 1944. Monofluoracetic acid, the toxic principle of "grifblaar"Dichapetabum cymosum (Hook) Engl. Onderstepoort J Vet Sci Anim Indust 20:67-73.

[109] Ward PFV, Hall RJ, Peters RA. 1964. Fluoro-fatty acids in the seeds of Dichapetabum toxicarium. Nature 201:611-612.

[110] Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Fluoride, The EFSA Journal (2005) 192, 1-65

[111] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

increase the fluoride content of the food by 0.5 mg/kg compared to low-fluoride water.[112,113,114]

Several factors influence the level of fluorides in food. These include the locality in which the food is grown, the amount of fertilizer and pesticides applied, the type of processing the food receives and whether fluoridated water is used in food preparation.[115,116,117] People are naturally exposed to fluoride from a variety of sources, including air, water, soil and food. In addition, intake can occur from fluoridated water, beverages, dietary supplements and dental products. Most foods contain low levels of fluoride (in the range of 0.1 to 2.5 mg/Kg). There are many foods which contain higher concentrations, e.g. teas, marine fish, bananas, potatoes, etc. Brewed tea, for example, can contain up to 8.6 mg/L fluoride, and it has been reported that decaffeinated teas may contain twice the amount.[118]
It is known that certain foods will contain higher fluoride levels if they are processed with fluoride-containing water. It has been documented that the fluoride content of water used in industrial food production and home-cooking affects the fluoride content of ready-to-eat products.[119] Baby formula processed with fluoridated water can contain 1 mg/kg fluoride and cooking food in fluoridated water results in increased dietary fluoride levels. In England, where much more tea is consumed, a study found daily average intake of fluoride from tea to be 1.26 mg/day in children and 2.55 mg/day in adults.[120] It is also reported that sections of the population that have a greater daily intake of fluids (such as labourers or sports enthusasists) generally have a greater daily intake of fluorides because of a larger consumption of water.[121]

The UK Expert group on Vitamins and Minerals noted that foodstuffs processed with fluoridated water may contain a fluoride concentration of 0.6-1.0 mg/kg rather than the normal 0.2-0.3 mg/kg. They further observed that intake by infants depends mainly on whether they are fed breast milk or a formula milk diet.

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[112] Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Fluoride, The EFSA Journal (2005) 192, 1-65
[113] Becker W and Bruce A (1981). Fluoride intake from food. Vår Föda 33 (Suppl 3): 198-261
[114] Marier JR and Rose D (1966). The fluoride content of some food and beverages - a brief survey using a modified Zr-SPADNS method. J Food Sci 31: 941-946
[115] McClure FJ. 1949. Fluorine in foods. Public health reports. U.S. Public Health Service. Public Health Rep 64:1061-1074.
[116] Myers HM. 1978. Fluorides and dental fluorosis. Monogr Oral Sci 7:1-76. Naidu MR, Sastry KY, Reddy DR. 1986. Skeletal fluorosis, secondary to occult renal disease. Fluoride 19:166-168
[117] Waldbott GL. 1963b. Fluoride in food. Am J Clin Nutr 12:455-462.
[118] Review Of Fluoride, Expert Group On Vitamins And Minerals, UK Expert Group On Vitamins And Minerals, EVM/01/03/P, May 2001.
[119] Review Of Fluoride, Expert Group On Vitamins And Minerals, UK Expert Group On Vitamins And Minerals, EVM/01/03/P, May 2001.
[120] Cook HA. 1969. Fluoride in tea. Lancet 2:329.
[121] WHO. 1984. Fluorine and fluorides. Geneva, Switzerland: World Health Organization, Distribution and Sales Service, EHC Number 36.

The expert body further noted that prepared drinks, such as beers, colas, carbonated soft drinks, sports drinks, reconstituted juices and health food drinks would be expected to contain levels of fluoride which are similar to those of the water supplies in which the factories are located. The fluoride content of processed foods and beverages prepared from water containing fluoride at 1 ppm level will contain about 0.5 mg/kg more fluoride than those prepared with non-fluoridated water.[122] A consequence of this is that a subsection of the population that is sensitive to fluoride may exhibit variable degrees of clinically significant reactions following consumption of food products from these areas. Given that consumers with such hypersensitivity would naturally tend to avoid such food products the policy of fluoridating water supplies may be seen to be a further unnecessary risk for indigenous food companies in Ireland. It is also self-evident that as conscientious consumers worldwide become more aware of the risks posed by fluoride that they may avoid purchasing food products that they see as unnecessarily contaminated with fluoride.

One of the major health concerns remains the fluoride levels in infant foods[123] which have been examined in detail in the previous sections of this report and elsewhere. While the risks associated with this have also been extensively reported worldwide it is notable that very little if any information on this subject has been provided to parents, consumers and healthcare providers in Ireland.
The EU SCHER committee in their report[124] noted that bottled natural mineral water is increasingly being used as a major source of water for drinking (this is also the case in Ireland due in part to contaminated drinking water supplies). SCHER noted that a large variation in the level of fluoride has been observed in bottled mineral water reaching up to 8 mg/L (EFSA 2005). Risks to consumers from fluoride in drinking water are discussed in more detail in subsequent sections of this report.

Food chain bio-accumulation for fluorides is not well documented. The total human intake is of interest, since multiple sources, all of which are generally considered safe by themselves, could, under some circumstances, provide total intake that is considered to be above the "safe" level.[125] This is examined in greater detail in subsequent sections of this report addressing bio-accumulation of fluorides in soil and ecological receptors, the exposure risks

[122] Review Of Fluoride, Expert Group On Vitamins And Minerals, UK Expert Group On Vitamins And Minerals, EVM/01/03/P, May 2001
[123] Report Of The Expert Panel For Water Fluoridation Review Standing Committee on Operations and Environment, City of Calgary, and Calgary Regional Health Authority - March 1998
[124] Scientific Committee on Health and Environmental Risks, European Commission, Director General Health and Consumers, Critical Review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, May 2010
[125] Scientific Committee on Health and Environmental Risks, European Commission, Director General Health and Consumers, Critical Review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, May 2010

and pollutant pathways that may impact on human health and the environment.

Exposure to fatal levels of fluoride could occur were accidents to occur through operator error or equipment malfunction at wastewater treatment facilities. At least seven events of acute fluoride poisoning that are related to the fluoridation of drinking water have formally been reported in the USA.[126] Further human exposure incident data has been provided by the American Association of Poison Control Centre[127] on children with fluoride poisoning in Colorado from January 1st to December 31st, 1986. Among the 87 cases, 85 were of mistaken ingestion of fluoride tablets, fluoride drops and fluoridated mouth-rinsing water in children from 8 months to 6 years of age, the most common age being 2 to 3 years. One child of 8 and another of 9 developed symptoms after receiving a dental fluoride application and a fluoride mouth rinse at a dental clinic. In the same year (1986), 3,511 cases were reported in the USA of mistaken ingestion of fluoride products, of which 91% occurred in children under 6 years old.[128] A study by Akiniwa[94] in Japan has demonstrated that acute fluoride poisoning is shown to be caused by exposure to lower doses of fluoride than commonly suggested.

While no direct cases of fluoride poisoning have been reported to my knowledge in Ireland the EPA has reported that naturally elevated levels of fluoride are quite rare in Ireland. It is extremely alarming however that the Agency reported that any exceedances in chemical standards reported are due almost entirely to public water supplies being dosed with fluoride at levels in excess of the legally permitted dose.

The most recent EPA report[129] noted that one public water supply of the fifty one found to be non compliant exceeded the upper fluoride concentration of 1.5mg/L in 2010. The SCHER review[130] reported that fluoride levels in drinking water Ireland in excess of 5mg/l have been documented. No details are provided for the location of any non compliant water supplies, the period for which any exceedances occurred or the population numbers at risk from the chronic fluoride exposure as a consequence of the reported exceedances.

Alarmingly, the EPA further reported that in an audit of water supply facilities in 2010, twenty two per cent of facilities were observed to have inadequate chemical dosing arrangements and fifty six per cent had inadequate source protection measures in place to protect water supplies from potential sources

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[126] Akiniwa, Kenji, Re-examination of acute toxicity of fluoride, Fluoride Vol. 30 No. 2 89-104 1997. Research Review

[127] Augenstein WL, Spoerke DG, Kulig KW *et al*. Fluoride ingestion in children: a review of 87 cases. *Pediatrics* 88 (5) 907-912 1991.

[128] Whitford G M. Acute and chronic fluoride toxicity. *Journal of Dental Research* 71 (5) 1549-1254 1992.

[129] The Provision and Quality of Drinking Water in Ireland, A Report for the Year 2010, Environmental Protection Agency.

[130] Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, Scientific Committee on Health and Environmental Risks, Director General for Health & Consumers 2010

of pollution that could contaminate supplies.[131] The Agency also reported that eighty one separate boil-water notices were issued by local authorities in 2010.

While this was generally undertaken due to microbiological contamination, boiling water does have public health implications in that it results in increased fluoride concentration that may cause further exceedances of the recommended standards and impact on infant health and safety.

It is consequently a very serious and alarming development to observe that up to 8.1% of the water samples taken from public group water schemes in Ireland, as noted by the EPA, were reported[132] to have exceeded the legal standard for fluoride. The legal standard for fluoride coincidentally is twice the recommended fluoride concentration in drinking water.

Astonishingly, it is reported by the EPA that the population are regularly exposed to extremely high concentrations of this toxin and aluminium due to repeated accidental releases as a consequence of persistant operator errors at water treatment facilities. Incredibly to my knowledge the health or environmental implications of such incidences has never been examined or reported.

[131] The Provision and Quality of Drinking Water in Ireland, A Report for the Year 2010, Environmental Protection Agency.
[132] The Provision and Quality of Drinking Water in Ireland, A report for the Years 2006-2007, Environmental Protection Agency.

4.5 Ingested Fluoride

Fluoride is not essential for human growth and development[133], but has in the past forty years become one of the most widely available elements, present in artificially elevated concentrations in fluoridated drinking water, processed foods and drinks prepared with treated water as well as cooked foods prepared with fluoridated water. As a consequence the daily intake and exposure of the population to fluoride is now considerably higher than that of previous generations.

Fluoride content of the body is not under physiological control. In humans the dominant route of fluoride absorption is via the gastrointestinal tract. At the most basic level, consider the following; fluoride is added to water so that it may make contact with teeth in an attempt to reduce the incidence of dental disease. It is now known that upwards of forty % of the population may suffer from dental fluorosis, a condition demonstrating chronic long-term biological poisoning where the level of fluoride in blood plasma, as a consequence of ingesting fluoridated drinking water, is so high that it causes visible physical structural damage to people's teeth. One can only imagine regardless of the evidence provided later in this report how this highly reactive toxin may be affecting other organs and the general health, physical and mental wellbeing of the wider population.

The various mechanisms of biological interaction and toxicity of fluoride and disassociated silicafluoride compounds are illustrated in Figure 1 overleaf. The interaction of fluoride in each of these processes will be examined in this report.

Ingested fluoride converts to hydrofluoric acid in the highly acidic environment of our stomachs and small intestines.[134,135] Hydrofluoric acid in the stomach has the associated risk of causing gastrointestinal irritation and ulceration of gastric and duodenal tissue. Hydrofluoric acid is a caustic acid and can produce severe tissue damage.[136] Individuals with ulcers or heartburn are not good candidates for the long-term consumption of water containing fluoride.[137] It has been found that higher acidity of the stomach increases absorption of fluoride.[138]

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[133] Opinion of the EU Scientific Panel on Dietetic Products, Nutrition and Allergies related to the Tolerable Upper Intake Level of Fluoride, 2005

[134] J. Ekstrand, G. Alvani, O. Boreusl, and A. Norlin. "Pharmacokinetics of fluoride in man after single and multiple oral doses." *European Journal of Clinical Pharmacology* July 1977 12(4): 311-317.

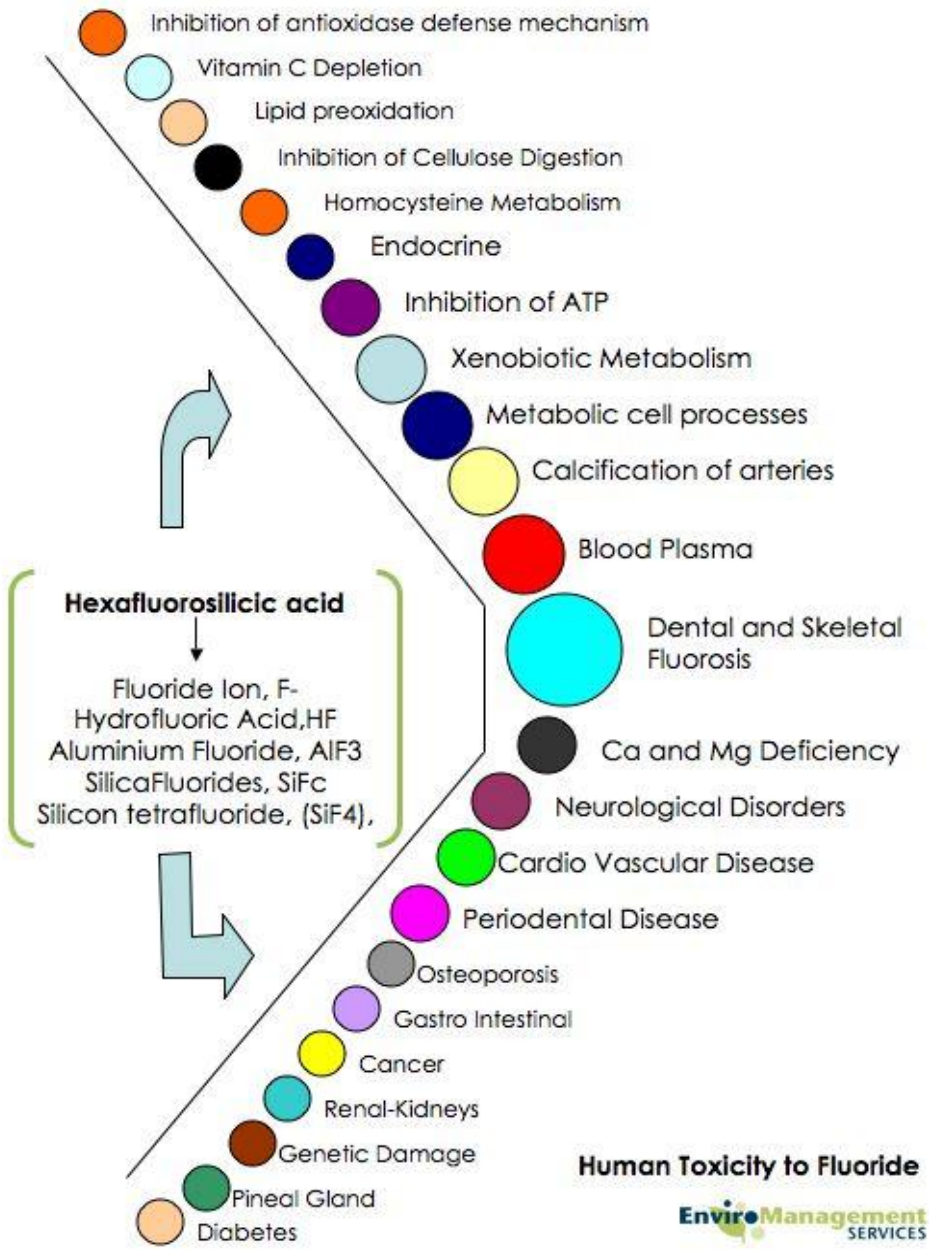
[135] Teitz, N., Clinical Chemistry, W.B. Saunders, Philadelphia, 1976.

[136] Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine, U.S. Agency for Toxic Substances and Disease Registry, Dept of Health & Human Services, 2003.

[137] Richard D. Sauerheber, Ph.D. (Chemistry, University of California, San Diego, 1976) On the Toxicity of Fluoridated Water.

[138] Opinion of the EU Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Fluoride, 2005

Fluoride in the body is retained in calcified tissue, bone and teeth as well as the pineal gland, kidneys and other tissues. Absorbed fluoride is partly excreted, predominantly via the kidney. In infants, retention in bone can be as high as 90% of the absorbed amount, whereas in adults, retention is 50% or less except for renally-impaired individuals where retention will be significantly more. In bone the substitution of fluoride for hydroxyl groups in apatite alters the mineral structure of the bone, reduces bone strength and increases risk of fracture and skeletal fluorosis (stiffness of joints, skeletal deformities).[139]



[139] Opinion of the EU Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Fluoride, 2005

There is limited evidence to suggest that water fluoridation may be a contributory factor in peptic ulcer disease through the formation of hydrofluoric (HF) acid in the stomach from the dissolution of silicafluoride compounds added to drinking water. A peptic ulcer is an inflammation of the stomach lining that develops when the lining of the stomach becomes corroded by stomach acid.

Acidic environments support the development of Helicobacter pylori, which is the major cause of peptic ulcer disease. It is medically plausible therefore that fluoridation of water supplies may be a contributory factor in the development of this disease. It is also known that a decrease in fluoride intake and reduction in blood plasma and urinary fluoride levels is associated with less gastrointestinal complaints.[140] Fluoride ions are released from fluorosilicic acid and almost completely absorbed by the body. The level of absorption depends in particular on the presence of calcium, magnesium and aluminium.[141],[142],[143],[144],[145] Aluminium has been found to increase absorption of fluoride while calcium and magnesium reduce absorption in the stomach.

Fluoride ions convert to hydrofluoric acid (HF) at pH 1.0-2.0 in the gut and are rapidly absorbed into the general circulation from the stomach and the intestine. [146] Once absorbed into the bloodstream fluoride is readily distributed throughout the body. Fluoride is retained in the bones, and teeth. Fluoride readily accumulates in the human pineal gland[147], by old age the pineal gland has a higher fluoride content than either bone, teeth or enamel.'[148]

In plasma, fluoride is transported as ionic fluoride and non-ionic fluoride. Ionic fluoride does not bind to plasma proteins and is easily excreted via urine.

[140] Susheela AK, Bhatnagar M. 2002. Reversal of fluoride induced cell injury through elimination of fluoride and consumption of diet rich in essential nutrients and antioxidants. Mol Cell Biochem 234/235:335-340.
[141] Harrison JE, Hitchman AJW, Hasany SA, Hitchman A, Tam CS (1984). The effect of diet calcium on fluoride toxicity in growing rats. Can J Physiol Pharmacol 62: 259-265.
[142] Kuhr J, Helbig J, Anders G, Munzenberg KJ (1987). Interactions between fluorides and magnesium. Magnesium-Bulletin 9: 110-113.
[143] Cerklewski FL (1997). Fluoride bioavailability – nutritional and clinical aspects. Nutr Res 17: 907-927.
[144] Spencer H, Osis D, Lender M (1981). Studies of fluoride metabolism in man. A review and report of original data. Sc Total Environ 17: 1-12.
[145] McClure FJ, Mitchell HH, Hamilton TS, Kinser CA (1945). Balances of fluorine ingested from various sources in food and water by five young men. Excretion of fluorine through the skin. J Ind Hyg Toxicol 27: 159-170.
[146] J. Ekstrand, G. Alvani, O. Boreusl, and A. Norlin. "Pharmacokinetics of fluoride in man after single and multiple oral doses." *European Journal of Clinical Pharmacology* July 1977 12(4): 311-317.
[147] Luke J. (1997). The Effect of Fluoride on the Physiology of the Pineal Gland. Ph.D. Thesis. University of Surrey, Guildford.
[148] Michotte Y, Lowenthal A, Knaepen L, Collard M, Massart DL: A morphological and chemical study of calcification of the pineal gland. J Neurol 1977;215:209-219.

It has been demonstrated that as the fluoride concentration in municipal water supplies increases with artificial fluoridation, the mean blood fluoride concentration of inhabitants can increase threefold from 0.014 to 0.04ppm.[149] This is confirmed by further research which demonstrated plasma fluoride concentration increased significantly with increased fluoride intake.[150]

When fluoride is in the form of HF, about 35-45%, is reabsorbed and returned to the systematic circulation.[151] When the kidneys are functioning properly, the body can excrete **up to** 50% of ingested fluoride. Malfunctioning kidneys can result in the body retaining up to 90% of ingested fluoride. Infants and toddlers can retain up to 85% of swallowed fluoride.

It is well-known that the metabolism and toxicity of fluoride may be modified by a great many factors, including xenobiotics (includes pharmaceutical drugs or environmental toxins that may be present in the body).[152],[153],[154]

Incorporation of fluoride ion into blood, bone and other tissues is variable[155] due in large part to wide ranges in water hardness and the ability of calcium to bind to the fluoride ion.

Fluoride also attaches to calcium anywhere this ion is concentrated throughout the body, including teeth, bones, ligaments, skeletal muscle and the pineal gland.[156],[157] Fluoride is a biologically active ion with demonstrable effects on bone cells, both osteoblasts and osteoclasts.[158]

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[149] Smith, Frank A.; Gardner, Dwight E.; Hodge, Harold C. Metabolism of fluoride. II. Fluoride content of blood and urine as a function of the fluoride in drinking water, Journal of Dental Research (1950), 29, 596-600 CODEN: JDREAF; ISSN: 0022-0345.

[150] Singer, Leon; Armstrong, W. D. Regulation of human plasma fluoride concentration, Journal of Applied Physiology (1948-1976) (1960), 15, 508-10 CODEN: JAPYAA; ISSN: 0021-8987

[151] Jha, S. K, Mishra K.V, Sharma K,D, Damodaran T, Fluoride in the Environment and its metabolism in Humans. Reviews of Environmental Contamination and Toxicology, Volume 211, March 2007. Springer-Verlag New York Inc.

[152] World Health Organization. Environmental Health Criteria, 227 Fluorides, IPS. Geneva: World Health Organization; 2002.

[153] Rao GS. Dietary intake and bioavailability of fluoride [review]. Annu Rev Nutr 1984;4:115-36.

[154] Trautner K, Einwag J. Influence of milk and food on fluoride bioavailability from NaF and Na2FPO3 in man. J Dent Res 1989;68:72-7.

[155] U.S. National Research Council, Report on Fluoride in Drinking Water, National Academy of Sciences,Washington, D.C., 2006

[156] Ongkana, Nutcharin; Zhao, Xiao-zhen; Tohno, Setsuko; High Accumulation of Calcium and Phosphorus in the Pineal Bodies with Aging Biological Trace Element Research, Volume 119, Number 2, November 2007 , pp. 120-127(8)

[157] Luke, Jennifer. "Fluoride Deposition in the Aged Human Pineal Gland". Caries Res 2991 (35): 125–28. Retrieved 2009-05-20

[158] U.S. National Research Council, Report on Fluoride in Drinking Water, National Academy of Sciences,Washington, D.C., 2006

The NRC Scientific Committee found[159] that fluoride accumulation is linear, rather than saturable, which suggests that fluoride accumulation in the bone is pathological, not physiological, where mineral nutrients always exhibit curved, saturable dose dependence and are fully reversible.

The NRC further reported how bone fluoride concentrations increase with age, in water fluoridated communities fluoride concentration have been shown to fall within the range of 4,000-12,000mg/kg. It was also observed that even if fluoride exposure is subsequently reduced the physiochemical effects are irreversible.

The NRC demonstrated that the levels of fluoride found in bone exceeded the ranges historically associated with Stage II skeletal fluorosis. Higher levels are expected for individuals with Type II diabetics.

The ATSDR observed[160] worryingly "*that although much is known about enzyme inhibition by fluoride, the human health significance remains to be determined.*" The ATSDR further documented "*that no data is available on the effects of intermediate-duration exposure to fluoride in humans while intermediate-duration exposure of animals to fluoride has resulted in effects on a number of organ systems, including bone, testes, kidney, neurobehavioral effects, and developmental effects.*" It was also noted in their report that "*effects on the bone are commonly reported, including decreased bone growth, alterations in tooth enamel, delayed bone healing, and increased bone formation rate.*"

A portion of the circulating inorganic fluoride also acts as an enzyme inhibitor because it forms metal-fluoride-phosphate complexes that interfere with the activity of those enzymes requiring a metal ion cofactor. In addition, fluoride may interact directly with the enzyme or the substrate. As far back as 2001 it is orth noting that the UK Expert Group on Vitamins and Minerals documended[161] how fluoride "*blocks normal cellular metabolism by inhibiting enzymes, in particular metalloenzymes involved in essential processes. Interference with functions controlled by calcium may be even more important, as the strong affinity for calcium results in hypocalcaemia, perhaps due to precipitation of fluorapatite. Other metal ions may be bound to fluoride as well, thereby blocking various biochemical mechanisms. Hyperkalaemia may result, with ventricular fibrillation*". [162]

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[159] U.S. National Research Council, Report on Fluoride in Drinking Water, National Academy of Sciences,Washington, D.C., 2006

[160] Interactive profile for Fluoride, U.S. Department of Health and Human Services Public Health Service Agency for Toxic Substances and Disease Registry, May 2004

[161] UK Expert Group on Vitamins and Minerals, Review of Fluoride, May 2001, EVM/01/03/, Page 34.

[162] Simpson E, Shankara Rao LG, Evans RM, Wilkie W, Rodger JC, Lakhani A (1980) Calcium metabolism in a fatal case of sodium fluoride poisoning. Ann Clin Biochem 17: 10-14.

The NRC reported[163] that a number of studies on rats, mice and guinea pigs have reported testicular effects, including reduced fertility, decreased sperm counts, and histologic alterations of the seminiferous tubules and Leydig cells.

There is a range of known or possible impacts of fluoride supplementation on health. Some of these effects accrue early such as dental fluorosis while others may occur later (eg any contributions to skeletal fluorosis and other aspects of bone health. Overall, there is general agreement that more robust information on the potential harms of fluoridation is needed to assess the long term human health impacts of water fluoridation.[164]

It is generally accepted that the effects on bone and teeth are the most sensitive effect of chronic long term exposure and that fluoride results in thickened bones and exostoses (skeletal fluorosis) when ingested in large doses for an extended period of time.

Little research exists on the interaction of fluoride with other toxins. Elsewhere in this report the interaction with aluminium will be examined. Another common interaction is with substances such as xenobiotics.

One of the most common such interactions is that between fluoride and aspirin. Aspirin (2- acetylsalicylic acid, ASA) is the most popular medicine in the world. ASA has been used as an analgesic, anti-inflammatory and antipyretic substance. In low doses taken for a long time, ASA will have anticoagulant activity.[165],[166]

Recent research[167] indicates that ASA counteracts the toxic effects of fluoride on body organs including the stomach but may also lead to increased accumulation of fluoride in bone, teeth or the pineal gland.

[163] U.S. National Research Council, Report on Fluoride in Drinking Water, National Academy of Sciences,Washington, D.C., 2006
[164] UK, Medical Research Council, Working Group Report Water Fluoridation and Health, Sept 2002
[165] Mastalerz L, Setkowicz M, Szczeklik A. Mechanism of chronic urticaria exacerbation by aspirin. Cerr Allergy Asthma Rep 2005;5:277-83.
[166] Radomski M W, Gordon P A, Radomski A, Armstrong P W. Modulation by aspirin of platelet aggregation pathways. Can J Cardiol 2000;16 Suppl F:S221.
[167] Inkielewicz, Czarnowski, Research report Oxidative stress parameters in rats exposed to fluoride and aspirin, Fluoride 41(1)76–82, January-March 2008

5.0 HUMAN CHEMISTRY: FLUORIDE INTERACTION WITH CALCIUM AND MAGNESIUM

The medical significance and importance of the interaction of fluoride with calcium and magnesium has profound implications for human health. Fluoride seeks out essential elements such as calcium and magnesium and binds with them, thereby interfering with their capacity to fulfil important metabolic processes in the body.[168] It is known that the level of fluoride absorption depends significantly on the presence of calcium, magnesium and aluminium.[169],[170],[171],[172],[173]

Calcium is necessary for bone mineralization and is an important cofactor for hormonal secretion in endocrine organs. At the cellular level, calcium is an important regulator of ion transport and membrane integrity. Calcium regulation is critical for normal cell function, neural transmission, membrane stability, bone structure, blood coagulation, and intracellular signalling.[174]

Calcium (Ca2+) is a substantial component of bones and teeth. In addition, it plays a role in neuromuscular excitability (i.e., decreases the proper function of the conducting myocardial system, heart and muscle contractility, intracellular information transmission and the coagulability of blood). Calcium intake is important at all ages,[175],[176] but the need for Ca2+ is higher during childhood, fetal growth, pregnancy, and lactation.[177] Epidemiological, animal and clinical studies support the existence of an inverse relation between Ca2+ intake and the occurrence of osteoporosis.[178],[179]

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[168] UK Medical Research Council Working Group Report: Water Fluoridation and Health, September 2002

[169] Harrison JE, Hitchman AJW, Hasany SA, Hitchman A, Tam CS (1984). The effect of diet calcium on fluoride toxicity in growing rats. Can J Physiol Pharmacol 62: 259-265.

[170] Kuhr J, Helbig J, Anders G, Munzenberg KJ (1987). Interactions between fluorides and magnesium. Magnesium-Bulletin 9: 110-113.

[171] Cerklewski FL (1997). Fluoride bioavailability – nutritional and clinical aspects. Nutr Res 17: 907-927.

[172] Spencer H, Osis D, Lender M (1981). Studies of fluoride metabolism in man. A review and report of original data. Sc Total Environ 17: 1-12.

[173] McClure FJ, Mitchell HH, Hamilton TS, Kinser CA (1945). Balances of fluorine ingested from various sources in food and water by five young men. Excretion of fluorine through the skin. J Ind Hyg Toxicol 27: 159-170.

[174] Suneja M, Muster H A, Batuman V, Arnold J L, Medscape Reference, Hypocalcemia, Oct 2011

[175] Heany RP. Nutritional factors in osteoporosis. *Annu Rev Nutr.* 1993;13:287–316

[176] Consensus Development Conference. Diagnosis, prophylaxis, and treatment of osteoporosis. *Am J Med.* 1993;94:646–50.

[177] Garzon P, Eisenberg MJ. Variation in the mineral content of commercially available bottled waters: implications for health and disease. *Am J Med.* 1998;105:125–30

[178] Heany RP, Gallagher JC, Johnston CC. Calcium nutrition and bone health in the elderly. *Am J Clin Nutr.* 1982;36:986–1013

[179] *Summary and recommendations.* Washington, DC: DHHS (PHS); 1988. The Surgeon General's Report on Nutrition and Health. Publication No. 88-50211;

A diet that is fortified in Ca2+ may reduce the rate of age-related bone loss and hip fractures, especially among adult women.[180] In spite of this knowledge, nutritional surveys indicate that a significant proportion of Irish men and women consume inadequate levels of Ca2+ and particularly women may have inadequate intake of this essential mineral.[181],[182]

Although many foods are now fortified with calcium (e.g., orange juice), naturally bio-available Ca2+ is found almost exclusively in milk, milk products, and water. Drinking water may be a significant source of Ca2+ for many consumers. It is logical therefore that one should not inject a substance into drinking water that may interact with calcium and further prevent its bio-availability for metabolism in the human body. Unfortunately this is exactly what the State is undertaking by fluoridating drinking water supplies with silicofluorides.

It is critically important therefore to note thatas far back as 1993 the U.S. *Agency for Toxic Substances and Disease Registry* reported[183] on the toxicological profile of fluorides and stated "*(b)ecause fluoride interacts with calcium ions needed for effective neurotransmission, fluoride can affect the nervous system.*"

It is known that fluoride not only inhibits enzymatic metabolism but that it also functions to prevent vital calcium and magnesium reactions as well as dramatically destabilising calcium binding in the body.[184]

Disorders in calcium metabolism give rise to many conditions including hypocalcaemia.

[180] McDowell LR. *Minerals in animal and human nutrition.* San Diego, Ca: Academic Press; 1992. pp. 26–73.pp. 78–95.pp. 98–137.

[181] SLÁN Survey of Lifestyle, Attitudes and Nutrition in Ireland Dietary Habits of the Population 2007, Department of Health and Children, 2008

[182] National Adult Nutrition Survey Summary report 2011, Irish Universities Nutrition Alliance.

[183] Toxicological Profile for Fluorides, Hydrogen Fluoride & Fluorine, U.S.Agency for Toxic Substances & Disease, Dept Of Health & Human Services, 1993, page 125.

[184] Alexander J. Murphy and Richard J. Coll. Fluoride Binding to the Calcium ATPase of Sarcoplasmic Reticulum Converts Its Transport Sites to a Low Affinity, Lumen-facing Form. The Journal of Biological Chemistry by the American Society for Biochemistry and Molecular Biology, Vol. 267, no. 24, issue of august 25, pp. 16990-16994, 1992

5.1 Hypocalcaemia

It is worth noting that the link between fluoride and hypocalcaemia was first reported by Simpson et al.[185] in 1980 and by the UK Expert Group on Vitamins and Minerals in 2001.[186]

The presentations of patients with hypocalcaemia vary widely, from asymptomatic to life-threatening situations. Hypocalcaemia is frequently encountered in patients who are hospitalized. Depending on the cause, unrecognized or poorly treated hypocalcaemic emergencies can lead to significant morbidity or death. Hypocalcaemia is an electrolyte imbalance and is indicated by a low level of calcium in the blood.

The hallmark of acute hypocalcaemia is neuromuscular irritability. Patients often complain of numbness and tingling in their fingertips, toes, and the perioral region. Paraesthesia of the extremities may occur, along with fatigue and anxiety.

Muscle cramps can be very painful and progress to carpal spasm or tetany. In extreme cases of hypocalcaemia, bronchospasm and laryngospasm with stridor may occur. Muscle symptoms can be as severe as to present as polymyositis with associated elevated muscle-associated isoenzymes. These symptoms are corrected by calcium replacement. Acute hypocalcaemia may also have cardiovascular manifestations.[187]

Patients with idiopathic hypoparathyroidism or pseudohypoparathyroidism may develop neurological complications, including calcifications of the basal ganglia and other areas of the brain, and extrapyramidal neurologic symptoms. If the patient has pre-existing subclinical epilepsy, hypocalcaemia may lower the excitation threshold for seizures. Epidermal changes are frequently found in patients with chronic hypocalcaemia. These include dry skin, coarse hair and brittle nails.

If hypocalcaemia has occurred prior to the age of 5, dental abnormalities may be present. Dental abnormalities include enamel hypoplasia, defects in dentin, shortened premolar roots, thickened lamina dura, delayed tooth eruption, and an increase in the number of dental caries.

Changes in smooth muscle function with low serum levels of calcium may cause irritability of the autonomic ganglia and can result in dysphagia,

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[185] Simpson E, Shankara Rao LG, Evans RM, Wilkie W, Rodger JC, Lakhani A (1980) Calcium metabolism in a fatal case of sodium fluoride poisoning. Ann Clin Biochem 17: 10-14.

[186] UK Expert Group on Vitamins and Minerals, Review of Fluoride, May 2001, EVM/01/03/, Page 34.

[187] Suneja M, Muster H A, Batuman V, Arnold J L, Medscape Reference, Hypocalcemia, Oct 2011

abdominal pain, biliary colic, wheezing, and dyspnea. In the elderly population, disorientation or confusion may be manifestations of hypocalcaemia.[188]

Hypocalcaemia is strongly associated with chronic kidney disease, inadequate PTH production and magnesium depletion; each of these conditions is regarded as a risks factor for developing hypocalcaemia. It is also known that fluoride toxicity and its interaction in the body specifically targets each of these conditions.

Therefore it is reasonable to suggest that fluoride may be a significant causative factor of hypocalcaemia. Hypocalcaemia is a common metabolic complication of malignant disease often requiring emergency intervention. Although it is more frequently associated with solid tumours, malignancy-associated hypocalcaemia (MAH) is seen in a significant number of patients with blood diseases.[189]

Of particular interest to Ireland are the studies by Christensson et al.[190],[191] who in a screen of apparently healthy adults in Sweden found an extraordinarily high incidence of 1.12% with hypocalcaemia among the wider population.

A further 2% of the apparently normal adults who harboured malignant disease were found to be hypocalcaemic during this health-screening programme. Depending on the cause, unrecognized or poorly treated hypocalcaemic emergencies can lead to significant morbidity or death.[192] It is also known that disorders of calcium metabolism such as hypocalcaemia are common in sarcoidosis.[193]

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[188] Schafer A L., Fitzpatrick L A.. Shoback D M, Hypocalcemia: Diagnosis and Treatment - Diseases of Bone and Mineral Metabolism

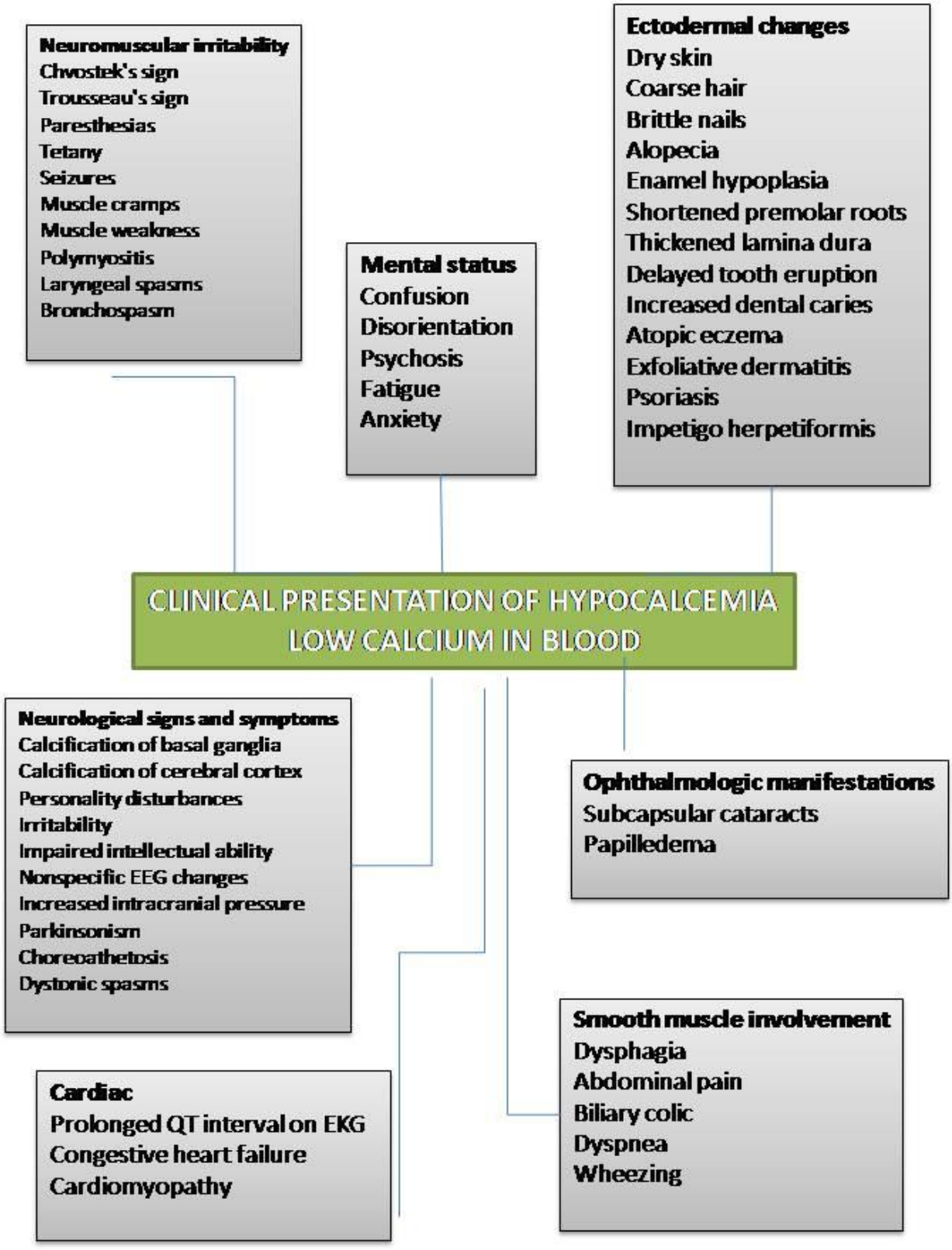
[189] Sargent, Jeremy T S, Smith, Owen P, Haematological emergencies managing hypercalcaemia in adults and children with haematological disorders. Br J Haematol. 2010 May;149(4):465-77. Epub 2010 Apr 4.

[190] Christensson, T., Hellstrom, K., Wengle, B., Alveryd, A. & Wikland, B. Prevalence of hypercalcaemia in a health screening in Stockholm. Acta MedScand 1976,200:131-137.

[191] Christensson, T., Hellstrom, K. & Wengle, B. Clinical and laboratory findings in patients with hypercalcaemia. Acta Med Scand 1976, 200: 355-360.

[192] Bikha Ram Devrajani, Syed Zulfiquar Ali Shah, Hypocalcemia in Acute Gastroenteritis(A Case-Control Study at Department of Internal Medicine) World Applied Sciences Journal 7 (6): 777-780,, 2009

[193] Sarcoidosis is a disease that leads to inflammation of the body's organs.



Source: Fitzpatrick, L.A.: The hypocalcemic states. Disorders of Bone and Mineral Metabolism., PA, pp. 568-588, 2002.

5.2 Sarcoidosis

Ireland has one of the highest incidences of sarcoidosis disease in the world.[194],[195]

This disease is a multi-system disorder, initially affecting young people in their 20s and 30s, which adversely affects quality of life. A number of spatial clusters of sarcoidosis have been indentified in Ireland which shows an incidence rate of this disease at rates up to twice or more than that of the European average. Interestingly, the incidence rate is Northern Ireland, where they do not fluoridate their drinking water, is less than half of the total national average for the Republic of Ireland and one quarter the incidence rate recorded in the highest incidence spatial locations found in Ireland.[196]

The geographic locations identified by Nicholson et al. of known spatial clusters for sarcoidosis mirror the geographic locations identifying vulnerable populations exposed to low calcium and magnesium in drinking water in this report. To my knowledge no study has ever been undertaken examining the potential link between drinking water quality, fluoridation and incidence of this disease in Ireland. This may explain why Northern Ireland, where drinking water is not fluoridated, has a much lower incidence rate of this disease compared to the ROI. It is noteworthy that the most at risk group of individuals to this disease are African Americans.[197],[198]

This racial group are also the most at-risk group from certain cancers[199] associated with lack of calcium bio-availability and potentially therefore, also the most at-risk from exposure to fluoride as noted elsewhere in this report. While sarcoidosis does not feature prominently in the media in Ireland, more Irish people suffer from sarcoidosis than they do from cystic fibrosis or cervical cancer. Extrathoracic sarcoidosis is generally associated with chronic progressive disease, particularly central nervous system, bone and skin involvement (e.g. lupus pernio). There may also be cardiac disease, renal disease or eye disease.[200]

While the specific cause for sarcoidosis is not known, one may observe from the pattern of disease progression that calcium bio-availability and fluoride

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[194] A. Saeed, M. Khan, S. Irwin and A. Fraser, Sarcoidosis presenting with severe hypocalcaemia. Irish Journal of Medical Science, Volume 180, Number 2, 575-577, DOI: 10.1007/s11845-009-0277-9

[195] Nicholson T. T., Plant, B.J.,Henry M.T., Bredin C.P., Sarcoidosis in Ireland: Regional differences in prevalence and mortality from 1996-2005. Sarcoidosis Vasculitis And Diffuse Lung Diseases 2010; 27; 111-120

[196] Nicholson T. T., Plant, B.J.,Henry M.T., Bredin C.P., Sarcoidosis in Ireland: Regional differences in prevalence and mortality from 1996-2005. Sarcoidosis Vasculitis And Diffuse Lung Diseases 2010; 27; 111-120

[197] Bresnitz EA, Strom BL. Epidemiology of Sarcoidosis. Epidemiol Rev 1983; 5: 1224-34.

[198] James DG, Hosoda Y. Epidemiology. In: James DG, Ed. Sarcoidosis and Other Granulomatous Disorders. Marcel Dekker, New York, 1994; 729-43.

[199] Merrill RM, Sloan A, Anderson AE, Ryker K. Unstaged cancer in the United States: a population-based study. BMC Cancer. 2011 Sep 21;11:402.

[200] A. Saeed, M. Khan, S. Irwin and A. Fraser, Sarcoidosis presenting with severe hypocalcaemia. Irish Journal of Medical Science, Volume 180, Number 2, 575-577, DOI: 10.1007/s11845-009-0277-9

exposure may have a vital role to play in its development, especially as it is now known that fluoride exposure is associated with each of the latter conditions. The geographic location identifying high clusters in this disease mirror the vulnerable populations identified in Figure 3 who may be exposed to low calcium and magnesium in drinking water. Death due to sarcoidosis occurs in 1 to 5% of patients with the disease[201] and mortality rates increase with age of patient. The association of fluoride or low calcium intake to sarcoidosis has to our knowledge never been tested. Perhaps the highest incidence of this disease recorded in the world was observed in a Galway/Mayo cluster where a prevalence of 96.55 per 100,000 was recorded.[202]

5.3 Relationship to Calcium Bio-availability in Drinking Water

It is likely that the incidence of these conditions would be most prevalent in geographic locations with low calcium and magnesium in drinking water. There are documented incidences of hypocalcemia in the southwestern region of Ireland which is known for its soft water.[203] This would be further exasperated by the artificially high level of fluoride in drinking water as a consequence of water fluoridation.

There is no doubt that the effects of fluoride in soft water such as found in the south of Ireland, particularly in Cork City, North, East and West Cork associated with the Rivers Blackwater, Lee, Bandon and Ilen may be subtle enough to go unnoticed by many health professionals and patients for long-time periods until clinical conditions present themselves. It is most likely that any association with drinking water low in calcium and magnesium and elevated in fluoride has never been investigated despite health risks being documented by the WHO.[204]

5.4 Fluoride Intake and Health Monitoring Programs

It is remarkable that to date, to my knowledge, no accurate risk assessment for fluoride has been undertaken to determine if it may be a contributory factor to disease or ill health within the wider community in Ireland.

Due to health concerns and increased incidences of hypersensitivity to certain foods the U.S. EPA have restricted the use of certain fluoride pesticides/herbicides due to food chain contamination and over-exposure of the population, especially infants, to fluoride. The U.S. EPA Office of Pesticides Programmes (OPP) undertook a risk assessment[205] of sulphuryl fluoride, a

[201] Hunninghake GW, Costabel U, Ando M, et al. Statement on sarcoidosis. Am J Respir Crit Care Med 1999; 160 (2): 736-55

[202] Nicholson T. T., Plant, B.J.,Henry M.T., Bredin C.P., Sarcoidosis in Ireland: Regional differences in prevalence and mortality from 1996-2005. Sarcoidosis Vasculitis And Diffuse Lung Diseases 2010; 27; 111-120

[203] Elamin and de Buyl. A novel mutation in the calcium-sensing receptor gene in an Irish pedigree showing familial hypocalciuric hypercalcemia: a case report. Journal of Medical Case Reports 2010, 4:349

[204] Calcium and Magnesium in Drinking-water Public health Significance, World Health Organization, 2009.

[205] U.S. EPA Office of Pesticide Programme Fluoride Risk Assessment and Relative Source Contribution

pesticide that breaks down into fluoride and is commonly used in the USA. The OPP found significant residues of fluoride in food that contributed to over-exposure of humans to fluoride. When combined with fluoride in drinking water and toothpaste the tolerance levels exceeded the safe tolerance standards for humans. No accurate total fluoride intake risk assessment has ever been undertaken in Ireland, despite this it is known that for many individuals (in particular infants and diabetics) the tolerable daily intake levels for fluoride are exceeded.[206],[207],[208],[209]

5.5 Calcium Disorders and Gastroenteritis

It is also known that elderly patients are frequently affected with disorders of calcium metabolism.[210],[211] Low serum calcium level (hypocalcemia) was identified in patients with acute gastroenteritis.[212] In the latter study hypocalcemia was identified in 94% of gastroenteritis patients similar to the recent findings published in the Journal of Gastroenterology[213] and consistent with further research undertaken by Westblom et al.[214]

Gastroenteritis is an infection of the gut and a leading cause of morbidity and mortality worldwide.[215] The peak incidence of infectious gastroenteritis is found in younger age groups (< 5years), while severe disease leading to hospitalization and resulting in death is most frequently observed in elderly patients (> 60 years).[216]

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[206] Critical review of new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, Scientific Committee on Health and Environmental Risks, Director General for Health & Consumers 2010

[207] U.S. National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C.

[208] Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Fluoride, The EFSA Journal (2005) 192, 1-65

[209] Review Of Fluoride, Expert Group On Vitamins And Minerals, UK Expert Group On Vitamins And Minerals, EVM/01/03/P, May 2001.

[210] Holick MF: Vitamin D deficiency. N Engl J Med 2007, 357:266-281.

[211] Abboud H, Henrich WL: Stage IV chronic kidney disease. N Engl J Med 2010, 362:56-65.

[212] Bikha Ram Devrajani, Syed Zulfiquar Ali Shah, Hypocalcemia in Acute Gastroenteritis(A Case-Control Study at Department of Internal Medicine) World Applied Sciences Journal 7 (6): 777-780,, 2009

[213] Bovee-Oudenhoven, I.M., M.L. Lettink-Wissink, Van W. Doesburg, B.J. Witteman and R. Van Der Meer, 2003. Diarrhoea caused by enterotoxigenic Escherichia coli infection of humans is inhibited by dietary calcium. Gastroenterol., 125: 469-76.

[214] Westblom, T.U. and TW. Milligan, 1992. Acute Bacterial Gastroenteritis Caused by Hafnia alvei. Clinical Infectious Diseases, 14(6): 1271-72

[215] Musher, D.M. and B.L. Musher, 2004. Contagiousacute gastrointestinal infections. N. Engl. J. Med. 351: 2417-27.

[216] Gangarosa, R.E., R.I. Glass, J.F. Lew and J.R. Boring 1992. Hospitalizations involving gastroenteritis in the United States, 1985: the special burden of the disease ***a***mong the elderly. Am. J. Epidemiol., 135: 281-90

5.6 Additional Health Risks from Low Calcium and Magnesium

Magnesium (Mg^{2+}) plays an important role as a cofactor and activator of more than 350 enzymatic reactions many of which are involved in energy metabolism including glycolysis, ATP metabolism, and transport of elements such as sodium, potassium and calcium through membranes, neuromuscular excitability and muscle contraction. It is also involved in protein and nucleic acid synthesis and is needed for normal vascular tone and insulin sensitivity.

Epidemiological studies suggest that an inverse relation exists between Mg^{2+} intake and the occurrence of ischaemic heart disease, cardiac arrhythmias and sudden death.[217],[218] Studies also suggest that an inverse relation exists between Mg^{2+} levels in drinking water and the occurrence of cardiac disease.[219] Surveys in Ireland demonstrate that no population group within the Irish population consumes the daily recommended Mg^{2+} requirement of 375 mg/day set by the EU Scientific Committee for Food. [220]

Certain population groups may be at a particular risk of not meeting recommended intakes for magnesium. The elderly may be vulnerable due, for example, to lower appetite and problems with eating. Ageing is also associated with an increase in the urinary excretion and a decrease in absorption of magnesium. Diuretics, often used by the elderly, may also lead to higher excretion rates.[221,222]

Pregnant women may be at risk of deficiency due to reduced serum magnesium levels. An increase in lean tissue during pregnancy, minimised urinary magnesium excretion and elevated bone resorption during lactation, may lead to increased requirements during and after pregnancy.[223]

Mg^{2+} depletion sometimes develops during periods of high physical activity leading to muscle cramps. Recovery capacity may be reduced due to insufficient magnesium intake.[224]

It is uncertain therefore given the enormous significance of both essential elements to human health why monitoring of these elements is not undertaken for monitoring drinking water quality. It is certain that the addition of fluoride compounds to drinking water would have a certain and immediate negative implication for both calcium and magnesium bio-availability.

[218] Eisenberg MJ. Magnesium deficiency and cardiac arrhythmias. *NY State J Med.* 1986;86:133–6.

[219] Marx A, Neutra RR. Magnesium in drinking water and ischemic heart disease. *Epidemiol Rev*. 1999;19:258–72.

[220] Irish Universities Nutrition Alliance (IUNA), The North-South Ireland Food Consumption Survey (2001).

[221] Rude RK, Magnesium deficiency: A cause of heterogeneous disease in humans. Journal of bone and mineral research 13, 4 (1998) 749-758.

[222] Institute of Medicine, Dietary Reference Intakes for calcium, phosphorus, magnesium, vitamin D and fluoride. National Academy Press, Washington, (2001) 190-249.

[223] Institute of Medicine, Dietary Reference Intakes for calcium, phosphorus, magnesium, Vitamin D and fluoride. National Academy Press, Washington, (2001) 190-249.

[224] Biesalski HK et al.: Vitamine, Spurenelemente und Mineralstoffe. Thieme Verlag, Stuttgart, 6 (2002) 132-237, 274, 333-334, 426, 485-486, 561.

While the concentrations of Ca^{2+} and Mg^{2+} in drinking water vary markedly from one drinking water supply to another, mineral-rich drinking waters may provide substantial contributions to total intakes of these nutrients in some populations or population subgroups. Water treatment processes can affect mineral concentrations and, hence, the total intake of calcium and magnesium for some individuals.[225] Fluoridation of drinking water will result in complexes of fluoride ions with calcium or magnesium ions.

The presence of calcium and magnesium will decrease the fluoride concentration in solution through the formation of soluble as well as insoluble calcium fluoride and magnesium fluoride complexes.[226,227] Gal et al.[228] regarded this phenomenon of particular public health significance as the precipitation of any calcium fluoride complexes could be responsible for the creation of more harmful hydrated forms. Pimentel et al. reported similarly in the Journal *Environmental Toxicology and Chemistry,* that systemic calcium and magnesium levels are depleted as a result of fluoride intoxication[229]. Calcium and, to a lesser extent, magnesium in water and food are known to have antitoxic activity. They can help prevent the absorption of some toxic elements such as lead and cadmium from the intestine into the blood, either via direct reaction leading to formation of an unabsorbable compound or via competition for binding sites.[230,231,232,233,234,235]

Since the early 1960's, epidemiological studies in many countries all over the

225 Calcium and Magnesium in Drinking-water Public health Significance, World Health Organization, 2009.

226 J.Y. Gal, N. Gache, Y. Fovet, Formation constants β_2 of calcium and magnesium fluorides at 25°C, Talanta, The International Journal of Pure and Applied Analytical Chemistry, Volume 53, Issue 3, 4 December 2000, Pages 617–626

227 J.Y. Gal, J.C. Bollinger, H. Tolosa, N. Gache, Calcium carbonate solubility: a reappraisal of scale formation and inhibition. Talanta 43 (1996) 1497.

228 J.Y. Gal, J.C. Bollinger, H. Tolosa, N. Gache, Calcium carbonate solubility: a reappraisal of scale formation and inhibition. Talanta 43 (1996) 1497.

229 Pimentel, R., Bulkley, R.V., 1983. Influence of water hardness on fluoride toxicity to rainbow trout. Environ. Toxicol. Chem. 2, 381–386.

230 Thompson, D.J. (1970) *Trace element in animal nutrition*. 3rd ed. Int. Minerals and Chem. Corp., Illinois.

231 Levander, O.A. (1977). Nutritional factors in relation to heavy metal toxicants. *Fed. Proc.* 36, 1683-1687.

232 Hopps, H.C. and Feder, G.L. (1986) Chemical qualities of water that contribute to human health in a positive way. *Sci. Total Environ.* 54, 207-216.

233 Nadeenko, V.G., Lenchenko, V.G. and Krasovskii, G.N. (1987) Combined effect of metals during their intake with drinking water. (In Russian.) *Gig. Sanit.* No.12 /1987 9-12.

234 Durlach, J., Bara, M. and Guiet-Bara, A. (1989) Magnesium level in drinking water: its importance in cardiovascular risk. In *Magnesium in Health and Disease* (ed. Y.Itokawa and J.Durlach), pp. 173-182, J.Libbey & Co Ltd, London.

235 Plitman, S.I., Novikov, Yu.V., Tulakina, N.V., Metelskaya, G.N., Kochetkova, T.A. and Khvastunov, R.M. (1989) On the issue of correction of hygenic standards with account of drinking water hardness. (In Russian.) *Gig. Sanit.* No. 7/1989 7-10.

world have reported that soft water (i.e., water low in calcium) and water low in magnesium are associated with increased morbidity and mortality from cardiovascular disease (CVD) compared to hard water and water high in magnesium.

An overview of epidemiological evidence is provided by recent review articles.[236],[237],[238],[239] Recent studies also suggest that the intake of soft water may be associated with higher risk of fracture in children[240], certain neurodegenerative diseases[241], pre-term birth and low weight at birth[242], some types of cancer[243],[244] as well as cardiovascular mortality from cardiovascular, ischaemic heart and hypertensive heart disease.[245] In addition to an increased risk of sudden death[246],[247],[248] the intake of water low in magnesium seems to be associated with a higher risk of motor neuronal disease[249], pregnancy disorders

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[236] Sauvant, M-P. and Pepin, D. (2002) Drinking water and cardiovascular disease. *Food* Chem. Toxicol. 40, 1311-1325.

[237] Donato, F., Monarca, S., Premi, S., and Gelatti, U (2003) Drinking water hardness and chronic degenerative diseases. Part III. Tumors, urolithiasis, fetal malformations, deterioration of the cognitive function in the aged and atopic eczema. (In Italian.) *Ann. Ig.* **15**, 57-70.

[238] Monarca, S., Zerbini, I., Simonati, C. and Gelatti, U. (2003) Drinking water hardness and chronic degenerative diseases. Part II. Cardiovascular diseases. *Ann. Ig.*15, 41-56.

[239] Nardi, G., Donato, F., Monarca, S., and Gelatti, U. (2003) Drinking water hardness and chronic degenerative diseases. Part I. Analysis of epidemiological research. (In Italian.) Annali di igiene - medicina preventiva e di comunita 15, 35-40.

[240] Verd Vallespir, S., Domingues Sanches, J., Gonzales Quintial, M., Vidal Mas, M., Mariano Soler, A.C., de Roque Company, C. and Sevilla Marcos, J.M. (1992) Association between calcium content of drinking water and fractures in children (in Spanish). *An. Esp. Pediatr.* 37, 461-465.

[241] Jacqmin, H., Commenges, D., Letenneur, L., Barberger-Gateau, P. and Dartigues, J.F. (1994) Components of drinking water and risk of cognitive impairment in the elderly. *Am. J. Epidemiol.* 139, 48-57.

[242] Yang, Ch.Y., Chiu, H.F., Chang, Ch. Ch., Wu, T.N. and Sung, F.Ch. (2002) Association of very low birth weight with calcium levels in drinking water. *Environ. Research*, Section A 89, 189-194.

[243] Yang, Ch.Y., Tsai, S.S., Lai, T.Ch., Hung, Ch.F. and Chiu, H.F. (1999c) Rectal cancer mortality and total hardness levels in Taiwan's drinking water. *Environ. Research*, Section A 80, 311-316.

[244] Yang, Ch.Y., Cheng, M.F., Tsai, S.S. and Hsieh, Y.L. (1998) Calcium, magnesium, and nitrate in drinking water and gastric cancer mortality. *Jpn. J. Cancer Res.* 89, 124-130.

[245] Crawford M.D. GARDNER. M.J. Morris J .N. Cardio Vascular Disease and the mineral content OF drinking Water. British Medical Bulletin, VOL 27 No 1. Pp 21-24

[246] Eisenberg, M.J. (1992) Magnesium deficiency and sudden death. *Am. Heart J.* 124, 544- 549.

[247] Bernardi, D., Dini, F.L., Azzarelli, A., Giaconi, A., Volterrani, C. and Lunardi, M. (1995) Sudden cardiac death rate in an area characterized by high incidence of coronary artery disease and low hardness of drinking water. *Angiology* 46, 145-149.

[248] Garzon, P. and Eisenberg, M.J. (1998) Variation in the mineral content of commercially available bottled waters: implication for health and disease. *Am. J. Med.* 105, 125- 130.

[249] Iwami, O., Watanabe, T., Moon, Ch.S., Nakatsuka, H. and Ikeda, M. (1994) Motor neuron disease on the Kii Peninsula of Japan: excess manganese intake from food coupled with low magnesium in drinking water as a risk factor. *Sci. Total Environ.* 149, 121-135.

(so-called pre-eclampsia)[250] and some types of cancer.[251],[252],[253],[254]

Low magnesium levels are associated with endothelial dysfunction, increased vascular reactions, elevated circulating levels of C-reactive protein and decreased insulin sensitivity. Low magnesium status has been implicated in hypertension, coronary vascular disease (CDV), Type 2 diabetes mellitus and metabolic syndrome.[255] It has been demonstrated by Chamberlain et al[256] that fluoride interferes with magnesium and manganese ions with consequences for rumen microorganisms.

A study by Machan et al.[257] examining manganese fluoride interaction processes relating to enamel and its effect on caries found that the fluoride ion affects enamel hardening and prevents its annealing, but this effect diminishes after administration of manganese.[258],[259]

Since pathologists and anatomists first began examining the heart, they realized that a connection existed between deposits of calcium and disease. In an early study of autopsy findings in 2,500 patients who died of coronary artery disease, they were found to have 2-5 times as much calcium as those who died of other causes. This observation is of great significance, because atherosclerotic

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[250] Melles Z, Kiss AS: Influence of the magnesium content of drinking water and of magnesium therapy on the occurrence of preeclampsia. *Magnesium Res* ***5*** :277–279,1992 .

[251] Yang, Ch.Y., Chiu, H.F., Cheng, M.F., Tsai, S.S., Hung, Ch.F. and Lin, M.Ch. (1999a) Esophageal cancer mortality and total hardness levels in Taiwan's drinking water. *Environ. Research*, Section A 81, 302-308.

[252] Yang, Ch.Y., Chiu, H.F., Cheng, M.F., Tsai, S.S., Hung, Ch.F. and Tseng, Y.T. (1999b) Pancreatic cancer mortality and total hardness levels in Taiwan's drinking water. *J.* Toxicol. Environ. Health A 56, 361-369.

[253] Yang, Ch.Y., Tsai, S.S., Lai, T.Ch., Hung, Ch.F. and Chiu, H.F. (1999c) Rectal cancer mortality and total hardness levels in Taiwan's drinking water. *Environ. Research*, Section A 80, 311-316.

[254] Yang, Ch.Y., Chiu, H.F., Cheng, M.F., Hsu, T.Y., Cheng, M.F. and Wu. T.N. (2000) Calcium and magnesium in drinking water and the risk of death from breast cancer. J. Toxicol. Environ. Health, Part A 60, 231-241.

[255] Calcium and Magnesium in Drinking-water Public health Significance, World Health Organization, 2009.

[256] C. C. Chamberlain and Wise Burroughs, Effect of Fluoride, Magnesium and Manganese Ions on in Vitro *Journal of Animal Science* 1962, 21:428-432.

[257] A Machoy-Mokrzynska, Fluoride-Magnesium Interaction, Institute of Pharmacology and Toxicology, Pomeranian Medical Academy, Szczecin, Poland. *Fluoride Vol. 28 No. 4; November, 1995, pp 175-177*

[258] Collys K, Slop D, Coomans D. Interaction of magnesium and fluoride in the rehardening and acid resistance of surface-softened bovine enamel in vitro. *Magnesium Trace Element* 9 (1) 47- 53, 1990

[259] Luoma A R, Luoma H, Raisanen J, Hausen H. Effect of magnesium and fluoride on the fermentative dissolution of enamel by a streptococcal layer as measured by mircrohardness tester and a proton probe microanalysis. *Caries Research* 17 430-438. 1983.

coronary artery disease is the number one cause of death in the Western world.[260]

Sufficient evidence is now available[261] to confirm the health risk from drinking water deficient in calcium or magnesium. Many studies show that higher water magnesium is related to decreased risks for CVD and especially for sudden death from CVD. This is of particular significance as it has been reported that one young Irish person under the age of 35 dies suddenly each week from cardiac diseases such as Cardiomyopathy or Sudden Adult Death Syndrome (SADS).[262]

This relationship has been independently described in epidemiological studies with different study designs, performed in different areas (with different populations), and at different times. The consistent epidemiological observations are supported by data from autopsy, clinical and animal studies.[263]

It is important to note the association of fluoride to SADS, it has been documented that fluoride inhibits calmodulin, loss of calmodulin activity has been linked to contractile dysfunction[264] which causes congestive heart failure as characterized by a high incidence of SADS.[265] This is further examined in section 7.12 of this report.

[260] Bayne Selby Jr, MD Professor of Radiology, Co-Director, Division of Interventional Radiology, Department of Radiology, Medical University of South Carolina, Pathophysiology of Calcium in the Coronary Arteries, Medscape Reference, April 2011

[261] Calcium and Magnesium in Drinking-water Public health Significance, World Health Organization, 2009.

[262] Dr Joe Galvin Consultant Cardiologists Mater Hospital, Dublin RTE News 12th February 2009

[263] Kozisek, F. Health risks from drinking demineralised water, Rolling revision of the WHO Guidelines for drinking-water quality, World Health Organization Geneva, 2004

[264] Turla, M, B. Gnegy,. Margaret, E,. Epps, S,. Shlafer, M. Loss of calmodulin activity in cardiac sarcoplasmic reticulum after ischemia. Biochemical and Biophysical Research Communications Volume 130, Issue 2, 31 July 1985, Pages 617–620

[265] Pogwizd SM, Bers DM. Na/Ca exchange in heart failure: contractile dysfunction and arrhythmogenesis. Ann N Y Acad Sci. 2002 Nov;976:454-65.

Recent studies demonstrating an association between intake of soft water, and higher risk of fracture in children, certain neurodegenerative diseases, pre-term birth and low weight at birth as well as some types of cancer cannot be dismissed.

> Given the obvious importance of both essential elements to general health and the hugely significant interactive properties of fluoride with both calcium and magnesium, the potential health consequences of adding fluorine compounds to soft water should be of paramount importance for the protection of public health and the provision of safe potable drinking water for human consumption.

This is of particular importance in communities with soft water and in communities dependent on groundwater aquifers for drinking water supplies. Approximately 26 % of the public and private drinking water supply in Ireland is provided by groundwater or spring sources with the latter supplying approximately 9 % of the total.[266]

In certain counties, e.g. Roscommon, the percentage is significantly higher, with groundwater providing approximately 75 % of the drinking water.[267]

5.7 Vulnerable Populations: Disease Clusters in Ireland

Those individuals living in communities where drinking water has low levels of calcium and magnesium would be at a risk of calcium and magnesium deficiency and at higher risk of developing neurological or cardiovascular disease as well as hypocalcemia and sarcoidosis in addition to certain cancers.[268] The WHO have also reported that concern for calcium deficiency would be directed mostly at individuals who consume little or no milk and rely on drinking water for their main source of the mineral. It is most likely that clusters of disease would develop in geographic locations with non-calcareous bedrock resulting in naturally low calcium concentrations in drinking and groundwater.

Logically the risk would be increased significantly by artificial fluoridation of water, as fluoride ions would further reduce the bio-availability of calcium for consumers. It is alarming therefore that data on drinking water quality for calcium and magnesium is not published in EPA drinking water reports.[269] It is apparent from the published reports that neither element is monitored consistently by either the EPA or local authorities. This is a matter that should

[266] Environmental Protection Agency (2008a) The Provision and Quality of Drinking Water in Ireland - A Report for the Years 2006-2007. EPA, Wexford

[267] Environmental Protection Agency (2007) Water Quality in Ireland 2006: Key Indicators of the Aquatic Environment. EPA, Wexford.

[268] Calcium and Magnesium in Drinking-Water Public Health Significance, World Health Organization, 2009

[269] The Quality of Drinking Water in Ireland Reports for the Years 2000-2010 inclusive, Office of Environmental Enforcement Environmental Protection Agency

require urgent attention by the EPA and HSE because of the growing concern that individuals may not be receiving clinically important levels of calcium and magnesium from drinking water as it is now known that many drinking water sources available to consumers in Ireland may contain insufficient levels of Ca2+and Mg2+. It is recommended that physicians should encourage patients to check the mineral content of their drinking water, whether tap or bottled, and choose water most appropriate for their needs.[270]

In spite of the knowledge of the health importance of $Ca^{2+}$and Mg^{2+} in drinking water there is no apparent health surveillance and bio-monitoring of the population in high risk areas to ensure that individuals consume the recommended dietary intake in accordance with WHO recommendations.

It is most likely that local authorities would have difficulty meeting the interim recommended limits for both $Ca^{2+}$and Mg^{2+} in most geographic areas of the country. This may itself explain why no data is readily available. It is imperative that the concentrations of both these substances are known prior to fluoridating drinking water. It is also essential to be aware of how the addition of fluoride to water may impact negatively on overall water quality.

The EPA in Ireland found that groundwater is generally low in both calcium and magnesium levels.[271] Calcium levels range from 17-89mg/l and magnesium levels range from 3.3 to 23mg/l. In both instances the lower concentrations are associated with Devonian bedrock geology found principally in the Southern part of Ireland. Devonian bedrock geology is principally located in the environs of Cork city, West Cork, East Cork, North Cork, South Limerick, South Kerry, North Waterford and parts of Clare, Tipperary, Roscommon, Leitrim and Cavan.

The communities living in these geographic areas would be considered the most vulnerable and at risk to the reported health risks and diseases from consuming drinking water low in $Ca^{2+}$and Mg^{2+} as reported by the WHO.[272] Individuals living in such areas, with low background concentrations of Ca2+and Mg2+ in drinking water, are exposed to a further cumulative risk with increased fluoride concentrations in the drinking water.

As fluoride actively seeks out calcium and magnesium, it further reduces the availability of these important ions for metabolism in the body. In areas with low $Ca^{2+}$and Mg^{2+}, the toxicity of the fluoride ion and any silicafluoride derivative compounds will be more aggressive on the body. This may have further negative implications for health due to the enhanced toxic effect of the fluoride ion on the body.

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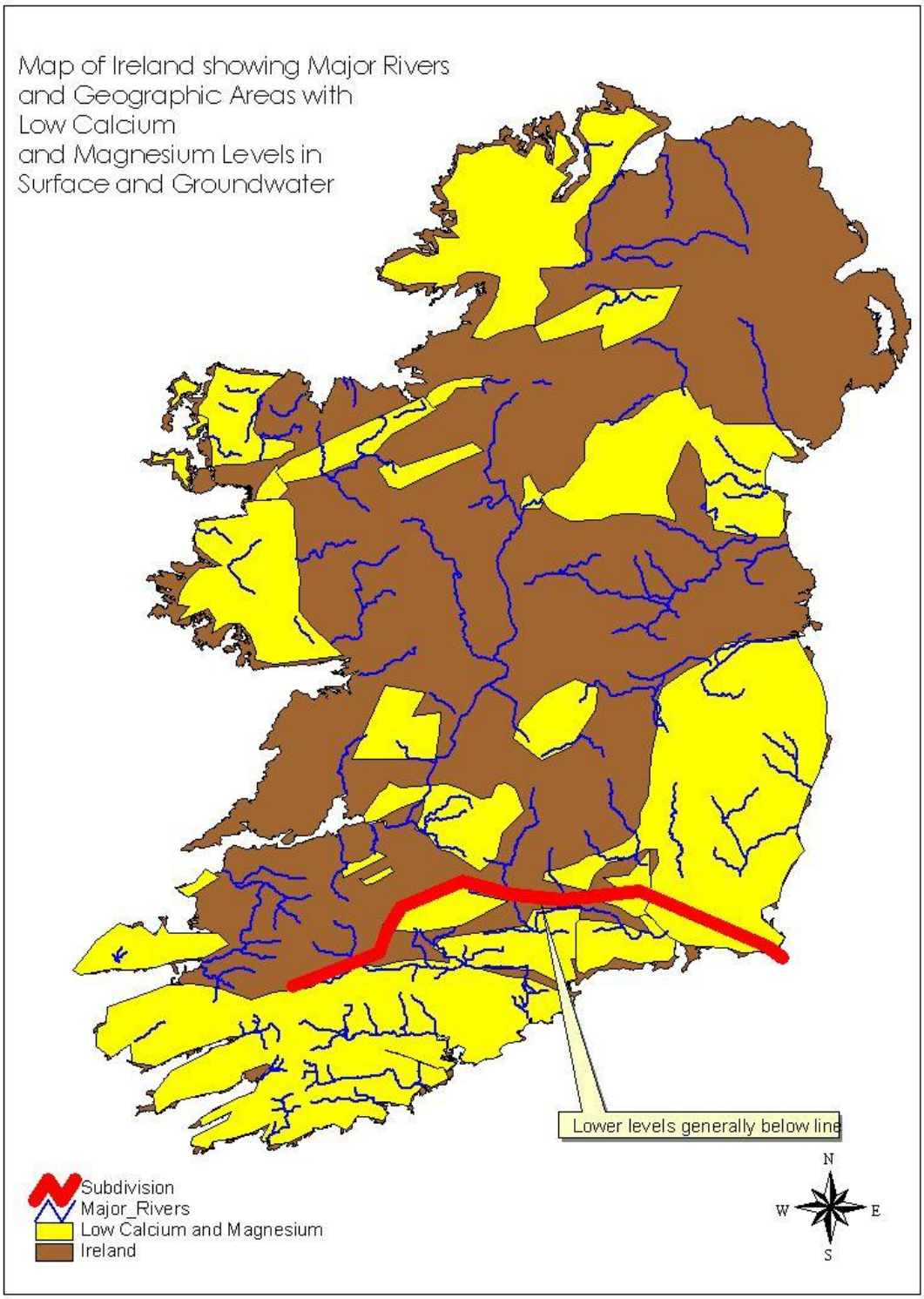
[270] Azoulay, A., Garzon, P., Eisenberg, M, J,. Comparison of the Mineral Content of Tap Water and Bottled Waters, J Gen Intern Med. 2001 March; 16(3): 168–175.

[271] Environmental Protection Agency (2005) *A Methodology for the Determination of Natural Background Quality of Groundwaters (2002-W-DS/7)*, Environmental RTDI Programme 2000-2006, EPA, Wexford.

[272] Calcium and Magnesium in Drinking-Water Public Health Significance, World Health Organization, 2009

Figure 3. Vulnerable geographic locations and high risk communities to water fluoridation in Ireland. All areas idenfied as yellow are low calcium waters with the softest water located below red line due to geological bedrock found in County Cork and South Kerry.



It is likely therefore that the risk of developing health complications would increase, especially when the reactive ion fluoride is added to drinking water in low $Ca^{2+}$and Mg^{2+} areas. As fluoride interacts with both calcium and magnesium ions, the likely consequence would be acute calcium and magnesium deficiencies in sensitive groups of the population. In a recent study of the Irish population aged between 18-64 year olds, it was found that there was a significant prevalence of calcium deficiency particularly amongst women.[273]

The bio-availability of fluoride and the role of calcium were also reported as a matter of some concern by the UK Medical Research Council (MRC) in 2002. [274] The MRC raised serious questions as to the health risks of water fluoridation in geographic areas with low calcium and magnesium levels.

It is noteworthy therefore that the highest incidence of cancer and mortality for prostrate, pancreatic and colorectal cancer in Ireland, as noted in the National Cancer Register, was found to be in County Cork.[275] A similar finding was found for incidence and mortality from cardiovascular disease.[276]

It is truly astonishing despite the known risks and high incidences of these diseases in populations consuming low levels of $Ca^{2+}$and Mg^{2+} in drinking water, that the Health Service Executive would appear to be unaware of the health risks posed to the population. If they were aware they would ensure that both elements were monitored in drinking water and more especially they would not support the addition of a known Ca^{2+}andMg^{2+} inhibitor being added to drinking water via water fluoridation. It is extremely alarming and disturbing that to my knowledge there is no reference in any public health reports for Ireland highlighting the health risk of low calcium and magnesium. Nor has any concern previously been raised regarding the impact of fluorine compounds being added to 'soft' drinking water or the increased risk of developing serious if not fatal diseases in the population as a consequence of calcium or magnesium deficiencies.

It is not surprising therefore that it has been found that the Southern region of Ireland, representing Cork City, West Cork and West Kerry, has the highest incidence of premature mortality from certain cancers and cardiovascular disease in Ireland. It is even more disturbing that the cumulative additive risk of injecting silicafluorides, containing the most reactive known chemical element fluoride, as well as its derivative compounds, into public water supplies and the inherent health risks that may be associated with this practice, have not been examined to date. Given the risks to human health this is not only unacceptable but entirely incomprehensible.

[273] Irish Universities Nutrition alliance (2008), National Teens Food Survey
[274] UK Medical Research Council Working Group Report: Water Fluoridation and Health, September 2002
[275] National Cancer Registry Ireland: 1994-2005.
[276] Ireland Take Heart Progress on the implementation of building Healthier Hearts 1999-2005, Health Service Executive.

Kidney and bladder cancer incidences are relatively high for women in Ireland (9th & 4th highest respectively of 31 countries surveyed). It is known that age-standardised mortality rates from cancer of the kidney have been increasing steadily for men (by 1.9% a year) and women (by 1.2% a year) since at least the 1960's (note Ireland commenced fluoridation in 1967). There has been a dramatic increase in the percentage of Stage I kidney cancers, from 5% of the total in 1994-1998 to 37% in 2004-2008 while Stage II cancers increased from 0% in 1994-1998 to 12% in 2004-2008. For bladder cancer, there has been a small increase in Stage III and IV cancers while the percentage of unstaged cancers remained high, at over forty per cent.[277]

In a recent study[278], undertaken in the U.S.A., researchers analysed data from 1,040,381 male and 1,011,355 female incident cancer cases diagnosed between 2000-2007 and collected by population-based cancer registries in the National Cancer Institute's Surveillance, Epidemiology and End Results Program. They found that the level of unstaged disease was greater in more lethal cancers (e.g., liver, oesophagus, and pancreas) compared with less deadly cancers (i.e., colon, urinary bladder and female breast).

It was reported that unstaged disease increased with age and is greater among non-married patients. Blacks compared with whites experienced significantly higher levels of unstaged cancers of the stomach, rectum, colon, skin (melanoma), urinary bladder, thyroid, breast, corpus, cervix, and ovaries, but lower levels of unstaged liver, lung and bronchial cancers. Males compared with females experienced significantly lower levels of unstaged cancers of the liver, pancreas, oesophagus and stomach, but significantly higher levels of unstaged lung and bronchial cancer and thyroid cancer.

There is no reason not to believe, given the size of the database in this study, that a similar pattern of cancer incidence may exist in Ireland. This being the case, given that the racial and ethnic demographics of Ireland have changed significantly in the past twenty years; it is to be expected that cancer incidence rates will increase significantly in southern regions, particularly County Cork.

This is likely to occur as the number of high risk individuals (including Black African ethnicity and people of any other Black background) increase in a geographic location where the incidences of such cancers is already significantly above normal. In additiont to cancer incidences increasing there is a risk that the incidence of cardiovascular disease in this region will also increase due to the interaction of fluoride with low calcium and magnesium levels in drinking water and its increased toxic bio-availability in the body.

It is reasonable to hypothesise that the addition of silicafluoride compounds to drinking water with low calcium and magnesium levels only acts a cumulative additive risk of initiating such cancers, as fluoride interacts with both elements reducing their bio-availability in the human body.

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[277] National Cancer Registry, Cancer of the Kidney, Ureter and Bladder, Nov 2011
[278] Merrill RM, Sloan A, Anderson AE, Ryker K. Unstaged cancer in the United States: a population-based study. BMC Cancer. 2011 Sep 21;11:402.

That the toxic effect of the fluoride ion plays a key role in acute magnesium deficiency is well known.[279],[280],[281]

The amount of fluoride assimilated by living organisms constantly increases as Magnesium absorption diminishes. The same principle applies to Ca^{2+}. The median fluoride natural background level in groundwater is estimated to be 0.1mg/l and less than 0.2mg/l in surface waters. In fluoridated drinking water it varies from 0.7-1.5mg/l.

Previous research findings have suggested that serum concentrations of fluoride influence the rate of calcification. It is now known that higher blood fluoride concentrations have shown greater enhancement of calcification.[282]

It has further been demonstrated that as concentration in municipal water supplies increased with artificial fluoridation the mean blood fluoride concentration of the inhabitants increased threefold from 0.014 to 0.040 p.p.m.[283] This is confirmed by further research demonstrating that plasma fluoride concentration increased significantly with increased fluoride intake.[284]

The significance of any association between fluoride and calcium and magnesium deficiencies and other critical diseases cannot be underestimated. These findings are of major significance demanding, in the interests of public health and safety, an immediate response from the State requiring the cessation of the water fluoridation policy without delay.

[279] A Machoy-Mokrzynska, Fluoride-Magnesium Interaction, Institute of Pharmacology and Toxicology, Pomeranian Medical Academy, Szczecin, Poland. *Fluoride Vol. 28* No. 4; November, 1995, pp 175-177

[280] Marier J R. Observations and implications of the (Mg F) interrelations in bio-systems: a review and comments on magnesium intake and fluoride intake in the modern-day world. Proceedings of the Finnish Dental Society 76. 82-92, 93-102, 1980. (Abstracted in *Fluoride* 14, 142 1981).

[281] Guminska M. The effect of magnesium on metabolism in living organisms and medical consequences of its deficiency in man. *Folia Medica Cracoviensia* 26 1-2, 5-28, 1985.

[282] Donald R. Taves, W.F. Neuman, From the Department of Radiation Biology, University of Rochester School of Medicine and Dentistry, Rochester, New York, USA, Factors controlling calcification *in vitro*: Fluoride and magnesium Archives of Biochemistry and Biophysics, Volume 108, Issue 3, December 1964, Pages 390–397

[283] Smith, Frank A.; Gardner, Dwight E.; Hodge, Harold C. Metabolism of fluoride. II. Fluoride content of blood and urine as a function of the fluoride in drinking water, Journal of Dental Research (1950), 29, 596-600 CODEN: JDREAF; ISSN: 0022-0345.

[284] Singer, Leon; Armstrong, W. D. Regulation of human plasma fluoride concentration, Journal of Applied Physiology (1948-1976) (1960), 15, 508-10 CODEN: JAPYAA; ISSN: 0021-8987

6.0 HUMAN CHEMISTRY: FLUORIDE INTERACTIVITY WITH ALUMINIUM

Due to its very strong electronegativity, fluorine has a tremendous effect on its neighbouring functional groups in a molecule. In many cases, the fluorine-containing compounds are more soluble than their counterparts. As a result, their solubility characteristics are also affected.[285] This is particularly the case with aluminium. The fluorine compounds used in water fluoridation disassociates to form fluoride ions. Fluoride Ions have been shown to affect the activity of a variety of functional proteins.[286] It is now known that a number of these effects are due to aluminofluoride ions.[287] The active species is thought to be AlF; which, under some circumstances, can act as a phosphate analogue.[288] Since trace amounts of aluminium are found in drinking water, as residual contaminants from water treatment with aluminium sulphate (ALUM) chemicals, it is likely this effect will also be observed in the human body.

It is for such reasons that the U.S. *Agency for Toxic Substances and Disease Registry* (ATSDR) recommended that additivity of fluoride to other compounds, that may lead to additive interactions, should be assumed as a public health protective measure in exposure-based assessments of health hazards from exposure to mixtures of fluoride compounds.[289] To my knowledge no such protective measures have been undertaken in Ireland. Humans are exposed to aluminium from a variety of environmental sources. Because aluminium sulphate (alum) is used as a flocculating agent in the purification of municipal water supplies, drinking water may contain high levels of aluminium.[290] Aluminium is a potent neurotoxic agent in humans.[291] It has been reported that aluminium by itself may not exert toxic affects on the nervous system, however, it may become a toxin after joined together with a fluoride to become an aluminium fluoride.[292] Alarmingly, it has been reported that in drinking water treated with aluminium compounds the addition of fluoride at a level of 1 mg/L was found to increase the amount of soluble aluminium by a factor of 10.[293]

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[285] Fluorine-Containing Peptidomimetics as Inhibitors of Aspartyl ProteasesBiomedical, Frontiers of Fluorine Chemistry. Chapter 14, pp 184–195 Chapter DOI: 10.1021/bk-1996

[286] Wiseman, A. (1970) Handb. EX^. Pharmacal. 20/2,48-97

[287] Chabre, M (1990) Trends BioChem Sci 15, 6-10

[288] Marc Paulais anRd . James Turne, Activation of the Na'-K'-2Cl- Cotransporter in Rat Parotid Acinar Cells by Aluminum Fluoride and Phosphatase Inhibitors, The Journal of Biological Chemistry, Vol. 267, No. 30, Issue of October 25, pp. 21558-21563,1992

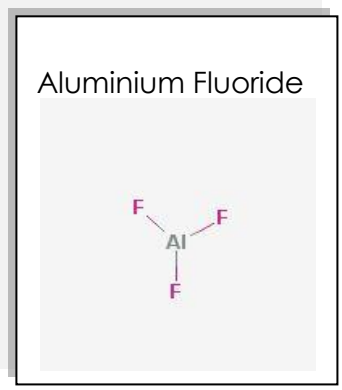
[289] Interactive Profile for Cyanide, Fluoride, Nitrate and Uranium. U.S Department of Health and Human Services, Public Health Service Agency for Toxic Substances and Disease Registry. May 2004.

[290] Aluminum Toxicity in Infants and Children, Committee on Nutrition PEDIATRICS, Vol. 97 No. 3 March 1996

[291] Aluminum Compounds Review of Toxicological Literature Abridged Final Report, USA National Institute of Environmental Health Sciences, October 2000

[292] Professor Robert L. Isaacson, My Views on the Fluoridation of Water Fluoridation, Department of Psychology and Center for Developmental and Behavioral Neuroscience, Binghamton University, NY

[293] Integrated Laboratory Systems, Aluminum Compounds Review of Toxicological



It is known that in aluminium-treated drinking water, fluorides can exist as fluoroaluminates.[294] The increased bio-availability of aluminium as a consequence of its interaction with fluoride in fluoridated drinking water is clearly therefore a major healthcare concern. The targeted risk for bottle-fed infants using formula reconstituted with fluoridated water is particularly alarming. It is known that bone and liver are the tissues most frequently affected by increased absorption of aluminium. It is also reported that intake of aluminium is a factor in certain brain, neurological and renal diseases.

Elevated plasma aluminium levels have been reported in healthy infants while in patients with chronic renal failure total body aluminium can be markedly increased.[295] The presence of aluminium-fluoride in infant formula contaminated from fluoridated compounds in drinking water is therefore of enormous concern.

It is remarkable that despite concerns and warnings issued by the Committee on Nutrition in the peer-reviewed journal Pediatrics, no health impact studies or risk reduction recommendations have been provided by either the Food Safety Authority or the Health Service Executive in Ireland. It is obvious that the cumulative risk of adding fluoride to drinking water poses serious health concerns for the population, in particular when the interaction of fluoride with other harmful substances, such as aluminium or other xenobiotic compounds, occurs.

Fluoride is one of the most reactive biological and chemical elements available and is used as a basic binding element for that purpose in a considerable number of pesticides and insecticides, an example being cryolite, the insecticide, that happens to be an aluminium fluoride compound. This is especially so given the findings of a study published in the peer-reviewed *Brain Research Journal*.[296] The authors of this study found that *"Fluoride has diverse actions on a variety of cellular and physiological functions, including the inhibition of a variety of enzymes, a corrosive action in acid mediums, hypocalcemia, hyperkalemia, and possibly cerebral impairment."*

Literature Abridged Final Report, U.S National Institute of Environmental Health Sciences, October 2000

[294] Integrated Laboratory Systems, Aluminum Compounds Review of Toxicological Literature Abridged Final Report, U.S National Institute of Environmental Health Sciences, October 2000

[295] Aluminum Toxicity in Infants and Children, Committee on Nutrition PEDIATRICS, Vol. 97 No. 3 March 1996

[296] Verner J A, Jensen K F, Horvath W, Isaacson R L, Chronic adminstration of aluminum-fluride or sodium-fluoride to rats in the drinking water: alterations in neuronal and cerebrovascular integrity, Brain Research 1998 Feb 16;784(1-2):284-98

Aluminium-based products, particularly those for water treatment (e.g., aluminium sulfate [alum], polyaluminium chloride [PAC], and sodium aluminate), are commonly used as compounds in water treatment in Ireland. It is also known that the relative risk of having Alzheimer's disease is increased when individuals have a high amount of alumimum in the brain coupled with low levels of fluoride.[297] Concern over the use of aluminium compounds as coagulants in drinking water treatment plants using fluoridation (as well as in effluent wastewater treatment plants) exists, since their addition usually results in an increase in the amount of aluminium in finished water.

Further important details on the toxicological profile of aluminium fluorides have been documented in a number of important studies that demonstrated serious kidney abnormalities and blood vessel deteriorations.[298],[299],[300] One group of investigators has proposed that the aluminium present in infant formulas played a role in the development of aluminium toxicity in two neonates with renal fai1ure.[301] The Varner et al. research team concluded that the alterations of the blood vessels from exposure to aluminium fluoride may be a primary event, triggering neuro-degenerative diseases. It has been reported that in respect to the study and cause of Alzheimer´s disease, the long-term action of aluminium fluoride also represents "*a serious and potent risk factor for the development of this new epidemic threat to human civilization*".[302,303.,304]

Professor Strunecká (Department of Physiology and Developmental Biology, Charles University, Prague), found that "*aluminium fluoride might induce the alterations of homeostasis, metabolism, growth and differentiation of the living organism. The long-term synergistic effects of these ions in living environments and their hidden danger for human health are not yet fully recognized*".[305]
Fluorides and fluoride-aluminium compounds have been demonstrated to

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[297] Belovjovic, G., Jakovlevic, B. (1999) Aluminum and Alzheimer's disease. Spr. ArArh. Celok 126: 283-289.

[298] Varner JA, Jensen KF, Horvath W, Isaacson RL, Chronic administration of aluminum-fluoride or sodium-fluoride to rats in drinking water: alterations in neuronal and cerebrovascular integrity, *Brain Res*. 1998 Feb 16;784(1-2):284-98

[299] Isaacson RL, Varner JA, Jensen KF, Toxin-induced blood vessel inclusions caused by the chronic administration of aluminum and sodium fluoride and their implications for dementia, *Ann N Y Acad Sci.* 1997 Oct 15;825:152-66..

[300] Varner JA, Horvath WJ, Huie CW, Naslund HR, Isaacson RL, Chronic aluminum fluoride administration, I. Behavioral observations, *Behav Neural Biol.* 1994 May;61(3):233-41

[301] Freundlich M, zilleruelo C, Ahitbol C, Strauss J, Faugere MC, Malluche NH. Infant formula as a cause of aluminum toxicity in neonatal uraemia. *Lancet.* 1985;2:527-529

[302] STRUNECKÁ A: Aluminium plus fluoride: a new deadly duo in Alzheimer´s disease. *The News (A Hoechst Marion Roussel Report of International Medical Opinion)* 1: 10-11, 1999.

[303] STRUNECKÁ A, PATOČKA J: Pharmacological and toxicological effects of aluminofluoride complexes. *Fluoride* 32: 230 -242, 1999a.

[304] STRUNECKÁ A, PATOČKA J: Re-evaluation of the participation of aluminium in Alzheimer´s disease (in Czech). *Čs fyziol* 48**:** 9-15, 1999b

[305] STRUNECKÁ A et al: Fluoride Plus Aluminum: Messengers of False Information, *Physiological Research 51: 557-564, 2002.*

affect guanine nucleotide-binding proteins (G proteins).[306] G proteins are on/off switches, which regulate cellular communication, relaying information received from outside the cell to the inside or from one cell to another. G proteins are metabolic enzymes, ion channels, transporters, and other parts of the cell machinery, controlling transcription, motility, contractility, and secretion, which in turn regulate systemic functions such as embryonic development, learning and memory, and homeostasis.[307] They are called G proteins because they bind to guanine nucleotides, a major component of the DNA and RNA. G protein defects have serious implications for autistic children.[308] Autism is a neuro-developmental disorder that affects the development of the brain. It is not beyond reasonable doubt to suggest therefore that in the absence of any detailed scientific risk assessments, fluoride could plausibly have an effect on the functioning of the central nervous system and development of toxicity in children.

It is reasonable to assume that in the absence of detailed risk assessments, and given that fluoride is regarded as a developmental neurotoxin, that there is an unproven risk of fluoride being a contributory risk factor to the rise in autism. It is very alarming to find therefore, despite the known risk to human health, that the interactivity of fluoride or silicafluoride compounds and its implications for human health have never been examined either in Ireland or in any country where water fluoridation exists. It is worth noting also that in a study[309] examining the association between fluoride, magnesium, aluminium and bone quality that bone aluminium was found to be positively associated with bone pain and proximal myopathy in patients. It was further found that the effects of fluoride and magnesium on bone quality may be exacerbated by their interaction with aluminium.

It is of some concern therefore that the EPA has reported[310] that regulatory compliance with the aluminium standard in drinking water has been as low as 85% in some locations such as Skibbereen in West Cork. Even more alarming is the fact that compliance figures of 66% have been reported for Donegal in previous years. In both cases, exceedances for fluoride were also noted presenting a very disturbing health concern for the communities residing in both counties.

[306] Margit Jeschke,Gesche J. R. Standke, Mira Susa, Fluoroaluminate Induces Activation and Association of Src and Pyk2 Tyrosine Kinases in Osteoblastic MC3T3-E1 Cells, The Journal of Biological Chemistry, 1998.

[307] Susana R. Neves, Prahlad T. Ram, Ravi Iyengar, G Protein Pathways, *Science 31 May 2002: 1636-1639.DOI:10.1126/science.1071550*

[308] The Mechanism of Aluminum-independent G-protein Activation by Fluoride and Magnesium. The Journal of Biological Chemistry by The American Society for Biochemistry and Molecular Biology, Vol. 268, No. 4, Issue of February 5, pp. 2393-2402,1993

[309] Ng AH, Hercz G, Kandel R, Grynpas MD. Association between fluoride, magnesium, aluminum and bone quality in renal osteodystrophy. Bone. 2004 Jan;34(1):216-24.

[310] The Quality of Drinking Water in Ireland Reports for the Years 2000-2010 inclusive, Office of Environmental Enforcement Environmental Protection Agency

7.0 HUMAN TOXICITY TO FLUORIDE

Fluoride is a toxin and its mode of action occurs at both the cellular and molecular levels causing significant enzyme inhibition involved in biochemical, cellular and molecular processes. Along with other toxic effects, fluoride is known to induce oxidative stress leading to excessive generation of reactive oxygen species (ROS), lipid peroxidation, a decrease in the glutathione(GSH)/oxidized glutathione (GSSH) ratio and alterations in activities of antioxidant enzymes, as well as an inhibition of glycolysis thus causing the depletion of cellular ATP and disturbances in cellular metabolism.

Furthermore fluoride changes the expression profile of apoptosis-related genes and cause endoplasmic reticulum stress leading to inhibition of protein synthesis.[311] The fluoride ion is a potent nucleophile in its desolvated state, fluorine's high redox potential precludes the haloperoxidase-type mechanism used in the metabolic incorporation of chloride and bromide ions.[312]

The toxicity of fluoride is associated with its high chemical and biological activity. Fluoride freely and rapidly migrates across the biological membranes, primarily in the form of HF via passive non-ionic diffusion in response to differences in the acidity of adjacent body fluid compartments.

After ingestion, fluoride is rapidly and virtually totally absorbed into the blood and distributes between organs and tissues. On an average, approximately 50% of the fluoride ingested by our body each day is excreted through the kidneys while the remaining fluoride accumulates in the bones, teeth, pineal glands and other tissues.[313]

The toxic fluoride effects include an induction of inflammatory reactions, cell contractile responses, inhibition of protein synthesis and cell cycle progression, oxidative stress and DNA damage.[314]

Fluoride induced apoptosis (cell death) was demonstrated in the cells from different organs and tissues including lungs,[315],[316], kidneys,[317],[318] liver,[319],[320]

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[311] Natalia Ivanovna Agalakova, Gennadii Petrovich Gusev, Molecular mechanisms of cytotoxicity and cell death induced by inorganic fluoride, Sechenov Institute of Evolutionary Physiology and Biochemistry, Russian Academy of Sciences.

[312] Dong C, Huang F, Deng H, Schaffrath C, Spencer JB, O'Hagan D, Naismith JH. Crystal structure and mechanism of a bacterial fluorinating enzyme. Nature. 2004 Feb 5;427(6974):561-5.

[313] G. M. Whitford, "Intake and metabolism of fluoride", *Advanced Dental Research*, vol. 8, pp. 5-14, 1994.

[314] O. Barbier, L. Arreola-Mendoza, L. M. Del Razo, "Molecular machanisms of fluoride toxicity", *Chemico-Biological Interactions*, vol. 188, pp. 319-333, 2010.

[315] E. V. Thrane, M. Refsnes, G. H. Thoresen, M. Lag, P. E. Schwarze, "Fluoride-induced apoptosis in epithelial lung cells involves activation of MAP kinases p38 and possibly JNK", *Toxicological Sciences*, vol. 61, pp. 83-91, 2001.

[316] M. Refsnes, P. E. Schwarze, J. A. Holme, M. Lag, "Fluoride-induced apoptosis in human epithelial lung cells (A549 cells): role of different G protein-linked signal systems", *Human and Experimental Toxicology*, vol. 22, pp. 111-123, 2003.

brain,[321],[322] pancreas,[323],[324] thymus,[325] endometrium,[326],[327] bone marrow,[328] hair follicles,[329] erythrocytes,[330],[331] as well as and leukemic cells. [332],[333]

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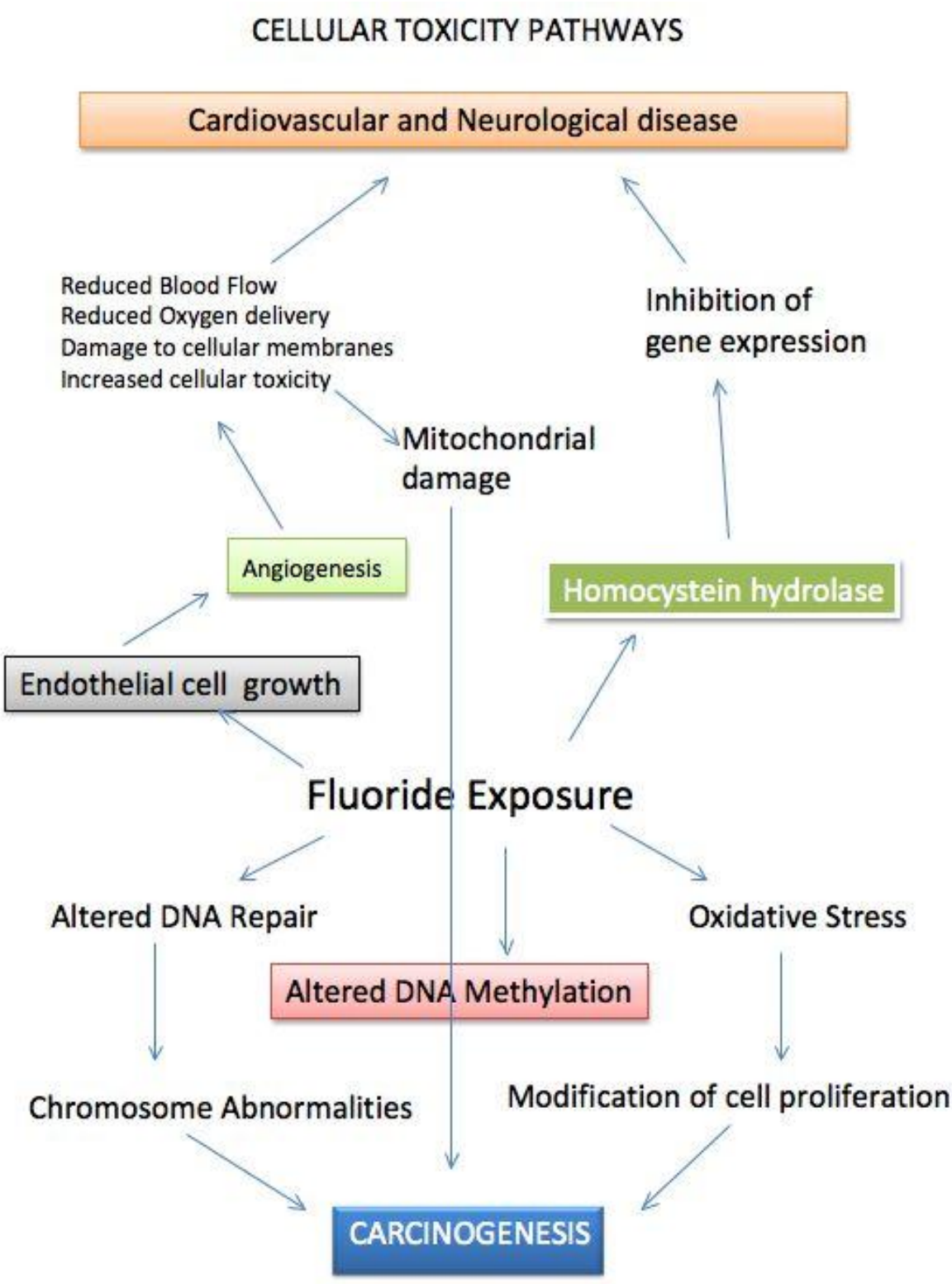
[317] H. Xu, X.-Q. Jin, L. Jing, G.-S. Li, "Effect of sodium fluoride on the expression of Bcl-2 family and osteopontin in rat renal tubular cells", *Biological Trace Element Research*, vol. 109, pp. 55-60, 2006.
[318] C. Bai, T. Chen, Y. Cui, T. Gong, X. Peng, H.-M. Cui, "Effects of high fluoride on the cell cycle and apoptosis of renal cells in chickens", *Biological Trace Element Research*, vol. 138, pp. 173-180, 2010.
[319] A. G. Wang, T. Xia, Q. L. Chu, M. Zhang, F. Liu, X. M. Chen, K. D. Yang, "Effects of fluoride on lipid peroxidation, DNA damage and apoptosis in human embryo hepatocytes", *Biomedical and Environmental Sciences*, vol. 17, pp. 217-222, 2004.
[320] L. F. He, J. G. Chen, "DNA damage, apoptosis and cell cycle changes induced by fluoride in rat oral mucosal cells and hepatocytes", *World Journal of Gastroenterology*, vol. 21, pp. 1144-1148, 2006
[321] Y. Ge, H. Ning, C. Feng, H. Wang, X. Yan, S. Wang, J. Wang, "Apoptosis in brain cells of offspring rats exposed to high fluoride and low iodine", *Fluoride*, vol. 39, pp. 173-178, 2006
[322] Y.-J. Liu, Z.-Z. Guan, Q. Gao, J.-J. Pei, "Increased level of apoptosis in rat brains and SH-SY5Y cells exposed to excessive fluoride – a mechanism connected with activation of JNK phosphorylation", *Toxicology Letters*, vol. 204, pp. 183-189, 2011.
[323] A C. Loweth, G. T. Williams, J. H. Scarpello, N. C. Morgan, "Heterotrimeric G-proteins are implicated in the regulation of apoptosis in pancreatic beta-cells", *Experimental Cell Research*, vol. 229, pp. 69-76, 1996.
[324] J. Elliot, J. H. Scarpello, N. G. Morgan, "Effects of tyrosine kinase inhibitors on cell death induced by sodium fluoride and pertussis toxin in the pancreatic β-cell line, RINm5F", *British Journal of Pharmacology*, vol. 132, pp. 119-126, 2001.
[325] H. Wang, B. Zhou, J. Cao, X. Gu, C. Cao, J. Wang, "Effects of dietary protein and calcium on thymus apoptosis induced by fluoride in female rats (Wistar rats)", *Environmental Toxicology*, vol. 24, pp. 18-224, 2009.
[326] M. Guney, B. Oral, G. Take, S. C. Giray, T. Mungan, "Effect of fluoride intoxication on endometrial apoptosis and lipid peroxidation in rats: role of vitamins E and C", *Toxicology*, vol. 231, pp. 215-223, 2007.
[327] M. Guney, B. Oral, H. Demirin, N. Karahan, T. Mungan, N. Delibas, "Protective effects of vitamins C and E against endometrial damage and oxidative stress in fluoride intoxication", *Clinical and Experimental Pharmacology and Physiology*, vol. 34, pp. 467–474, 2007.
[328] A. Machalinska, A. Machoy-Mokrzynska, W. Marlicz, I. Stecewicz, B. Machalinski, "NaF-induced apoptosis in human bone marrow and cord blood CD34 positive cells", *Fluoride*, vol. 34, pp. 258-263, 2001
[329] Z.-H. Wang, X.-L. Li, Z.-Q. Yang, M. Xu, "Fluoride-induced apoptosis and lipid peroxidation in human hair follicles in vitro", *Biological Trace Element Research*, vol. 137, pp. 280-288, 2010.
[330] S. Shouhan, S. J. Flora, "Effects of fluoride on the tissue oxidative stress and apoptosis in rats: biochemical assays supported by IR spectroscopy data", *Toxicology*, vol. 254, pp. 61–67, 2008.
[331] N. I. Agalakova, G. P. Gusev, "Fluoride-induced death of rat erythrocytes in vitro", *Toxicology In Vitro*, vol. 25, pp. 1609-1618, 2011.
[332] S. Otsuki, S. R. Morshed, S. A. Chowdhury, F. Takayama, T. Satoh, K. Hashimoto, K. Sugiyama, O. Amano, T. Yasui, Y. Yokote, K. Akahane, H. Sakagami, "Possible link between glycolysis and apoptosis induced by sodium fluoride", *Journal of Dental Research*, vol. 84, pp. 919-923, 2005.
[333] C. D. Anuradha, S. Kanno, S. Hirano, "Oxidative damage to mitochondria is a preliminary step to caspase-3 activation in fluoride-induced apoptosis in HL-60 cells", *Free Radical Biology and Medicine*, vol. 1, pp. 367-373, 2001.

Apoptosis, or programmed cell death, is a complex and highly regulated phenomenon playing a key role in the elimination of unnecessary or damaged cells and in a variety of normal biological processes such as cell proliferation and differentiation, tissue homeostasis and aging.[334],[335] The morphological changes typical for apoptosis include condensation of nuclear chromatin, DNA fragmentation, disintegration of mitochondria, cell shrinkage, membrane blebbing (bulge in plasma membrane) and formation of apoptotic bodies.

The biochemical features include a delicate regulation of intracellular signaling pathways via gene expression and/or protein activity. The molecular mechanisms underlying fluoride-induced apoptosis are different by nature and include the stimulation of G protein-dependent signaling systems, oxidative stress, ATP depletion, activation of the cell surface death receptors, disruption of outer mitochondria membrane, activation of caspases, alterations in the ratio of anti-apoptotic Bcl-2 proteins, upregulation of p53 expression[336], expression of apoptosis-related genes, endoplasmic reticulum stress and disturbances in protein synthesis.[337]

[334] A. H. Wyllie, "Where, o death, is thy sting?" A brief review of apoptosis biology", *Molecular Neurobiology*, vol. 42, pp. 4-9, 2010.
[335] E. F. Mason, J. C. Rathmell, "Cell metabolism: an essential link between cell growth and apoptosis", *Biochimica et Biophysica Acta*, vol. 1813, pp. 645-654, 2011.
[336] p53 is the most commonly mutated tumor suppressor gene in human cancers.
[337] Natalia Ivanovna Agalakova, Gennadii Petrovich Gusev, Molecular mechanisms of cytotoxicity and cell death induced by inorganic fluoride, Sechenov Institute of Evolutionary Physiology and Biochemistry, Russian Academy of Sciences.


CELLULAR TOXICITY PATHWAYS
Cardiovascular and Neurological disease
Reduced Blood Flow
Reduced Oxygen delivery
Damage to cellular membranes
Increased cellular toxicity
Inhibition of gene expression
Mitochondrial damage
Angiogenesis
Homocystein hydrolase
Endothelial cell growth
Fluoride Exposure
Altered DNA Repair
Oxidative Stress
Altered DNA Methylation
Chromosome Abnormalities
Modification of cell proliferation
CARCINOGENESIS

7.1 Cellular Oxidative Stress and Fluoride

Generally, this term describes an imbalance between oxidative damage in the cell, produced by reactive oxygen species (ROS), and the status of intracellular antioxidant systems. Chronic or acute ROS overproduction, exceeding the capacity of cellular antioxidant defence systems, causes oxidative damage to macromolecules such as DNA and proteins, peroxidation of membrane phospholipids, mitochondrial depolarization, thus initiating apoptosis and organ lesions. The mechanism of action of fluoride and fluoride compounds is to assist in the activation of superoxide production.

It has been found that fluoride is a toxic anion that stimulates oxygen consumption.[338] That can lead to the generation of highly destructive free radicals such as superoxide radicals that can damage cell membranes and lead to oxidative stress. The fluoride ion reacts with an active site nucleophile[339] causing superoxide production in neutrophils. Reactive oxygen species (such as fluoride) have been found to contribute to neutrophil activation and the development of acute inflammatory processes in which neutrophils play a central role.[340] It has been found that the fluoride ion (F-) is an effective activator of the respiratory burst in neutrophils, as indicated by its ability to induce O2- production by these cells. F- stimulated O2- production showed a requirement for Ca++.[341]

Fluoride depletes the energy reserves and the ability of white blood cells to properly destroy foreign agents by the process of phagocytosis.[342] It has been found that as little as 0.2ppm fluoride stimulates superoxide production in resting white blood cells, virtually abolishing phagocytosis. Even micro-molar amounts of fluoride, below 1 ppm, may seriously depress the ability of white blood cells to destroy pathogenic agents.[343],[344],[345],[346]

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[338] Sbarra AJ, Karnovsky ML. The biochemical basis of phagocytosis. I. Metabolic changes during the ingestion of particles by polymorphonuclear leukocytes. J Biol Chem 1959;234:1355–1362

[339] A nucleophile is a chemical species that is electron-rich.

[340] Lorne E, Zmijewski J W, Zhao X, Liu G, Tsuruta Y, Park Y J, Dupont H, and Abraham E, Role of extracellular superoxide in neutrophil activation: interactions between xanthine oxidase and TLR4 induce proinflammatory cytokine production. Am J Physiol Cell Physiol April 2008 vol. 294 no. 4 C985-C993

[341] Curnutte JR, Babior BM, Karnovsky ML. Fluoride-mediated activation of the respiratory burst in human neutrophils. A reversible process. J Clin Invest1979;63:637–64

[342] Phagocytosis is process the human body uses to destroy dead or foreign cells.

[343] John Curnette, et al, " *Fluoride-mediated Activation of the Respiratory Burst in Human Neutrophils*," Journal of Clinical Investigation, Vol. 63, pp. 637-647 (1979).

[344] W. L. Gabler and P. A. Leong, ., " *Fluoride Inhibition of Polymorphonumclear Leukocytes*," Journal of Dental Research, Vol. 48, No. 9, pp. 1933-1939 (1979);

[345] W. L. Gabler, et al., " *Effect of Fluoride on the Kinetics of Superoxide Generation by Fluoride*," Journal of Dental Research, Vol. 64, p. 281 (1985);

[346] A. S. Kozlyuk, et al., " *Immune Status of Children in Chemically Contaminated Environments*," Zdravookhranenie, Issue 3, pp. 6-9 (1987)

There is increasing evidence to suggest that mitochondrial dysfunction and interrelated intramitochondrial generation of the superoxide anion and other reactive oxygen species (ROS) are also implicated in the pathophysiological processes associated with aging, cancer, neurodegenerative and inflammatory disorders, diabetes and diabetic complications.[347],[348],[349],[350],[351]

Oxidative stress is also a common mechanism by which chemical toxicity can occur in the liver. The results of animal tests suggest that fluoride reduces the efficiency of the enzymatic antioxidative system in the liver.[352] Oxidative stress is a recognized mode of fluoride action in some cell types. An involvement of increased ROS production and lipid peroxidation in the fluoride-induced apoptosis has been proven *in vitro* in many cultured cell types, as well as *in vivo* in experimental animals and in people living in areas of endemic fluorosis.[353],[354] The main ROS implicated in the tissue injury include the superoxide anion (O2) generated in mitochondria and the hydroxyl radical (OH-) produced from the decomposition of hydroperoxides. Fluoride may induce the generation of superoxide anion (O2-) as well as its downstream consequences such as hydrogen peroxide, peroxynitrile, hydroxyl radical (OH-). The resultant altered thermal instability from superoxide activation may result in enzyme-activated irreversible inhibition and decreased enzymatic activity resulting in elevated S-ADENOSYLHOMOCYSTEINE HYDROLASE (AdoHcyase) in the blood. That is fluoride induces free radicals that lead to cellular oxidative stress resulting in increased AdoHcyase. AdoHcyase is an enzyme responsible for homocysteine metabolism. In addition to its association with cerebrovascular disease, homocysteine may play a role in neurodegenerative disorders.[355] It is known that increased levels of AdoHcy may play a role in the development of cardiovascular diseases and numerous other conditions associated with hyperhomocysteinaemia.[356],[357]

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[347] Shigenaga MK, Hagen TM, Ames BN. Oxidative damage and mitochondrial decay in aging. Proc Natl Acad Sci USA. 1994;91:10771–10778.

[348] Green K, Brand MD, Murphy MP. Prevention of mitochondrial oxidative damage as a therapeutic strategy in diabetes. Diabetes. 2004;53

[349] Csiszar A, Pacher P, Kaley G, Ungvari Z. Role of oxidative and nitrosative stress, longevity genes and poly(ADP-ribose) polymerase in cardiovascular dysfunction associated with aging. Curr Vasc Pharmacol. 2005;3:285–291.

[350] Pacher P, Beckman JS, Liaudet L. Nitric oxide and peroxynitrite in health and disease. Physiol Rev. 2007;87:315–424.

[351] Partha Mukhopadhyay,a Mohanraj Rajesh,a Kashiwaya Yoshihiro,b György Haskó,c and Pál Pache, Simple quantitative detection of mitochondrial superoxide production in live cells, Biochemical and Biophysical Research Communications Volume 358, Issue 1, 22 June 2007, Pages 203–208.

[352] Błaszczyk I, Birkner E and Kasperczyk S, Influence of Methionine on Toxicity of Fluoride in the Liver of Rats Biological Trace Element Research Volume 139, Number 3, 325-331, DOI: 10.1007/s12011-010-8664-7

[353] O. Barbier, L. Arreola-Mendoza, L. M. Del Razo, "Molecular machanisms of fluoride toxicity", *Chemico-Biological Interactions*, vol. 188, pp. 319-333, 2010.

[354] D. Chlubek, "Fluoride and oxidative stress", *Fluoride*, vol. 36, pp. 217-228, 2003.

[355] Ramon Diaz-Arrastia, MD, PhD, Homocysteine and Neurologic Disease, ARCH NEUROL/VOL 57, OCT 2000

[356] Ksenija Fumi , Robert Belui , Mario uk, Tea Pavkov, Doris Kloor, Ivo Bari, Ivana Miji and Oliver Vugrek, Functional analysis of human S-adenosylhomocysteine hydrolase isoforms SAHH-2 and SAHH-3, European Journal of Human Genetics (2007) 15,347–351.

7.2 Homocysteine Metabolism Cardiovascular Disease and Fluoride

Over the last 10 years, there has been an explosion of interest in homocysteine, a sulphur-containing amino acid that occupies a central location in metabolic pathways.

This interest is primarily because of the realization that hyperhomocysteinaemia is an important risk factor for vascular disease, including stroke, independent of long-recognized factors such as hyperlipidaemia, hypertension and diabetes mellitus. In addition to its association with cerebrovascular disease, homocysteine may play a role in neurodegenerative disorders.[358]

Homocysteine is an intermediate product of methionine metabolism.[359],[360],[361],[362] Any substance that may inhibit secondary metabolism of methionine would result in increased homocysteine levels resulting from less homocysteine being metabolized into (L)-Cystathionine. Fluoride is known to be an inhibitor of enzymatic activity and research has identified fluoride as an inhibitor of homocysteine hydrolase.[363] Inhibition of homocysteine hydrolase would result in cellular accumulation of homocysteine.[364]

Wang et al.[365] in their recent meta-analysis of 8 intervention trials with supplemental folic acid, which involved a total of almost 17,000 subjects, concluded that reduction in blood homocysteine levels (Hcy) significantly reduced the risk of stroke by 29%. The *European Journal of Clinical Investigation* likewise reported that 40% of all stroke victims have elevated Hcy levels compared to only 6% of controls.[366] In their commentary on

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[357] Trabeti e. Homocysteine, MTHFR gene polymorphisms and cardio-cerebrovascular risk. J Appl Genet 2008; 49:267-282.

[358] Ramon Diaz-Arrastia, MD, PhD, Homocysteine and Neurologic Disease, ARCH NEUROL/VOL 57, OCT 2000

[359] Palmer JL, Abeles RH, The mechanism of action of S-adenosylhomocysteinase. The Journal of biological chemistry 1979 Feb 25;254(4):1217-26

[360] Hershfield MS, Aiyar VN, Premakumar R, Small WC, S-Adenosylhomocysteine hydrolase from human placenta. Affinity purification and characterization. The Biochemical journal 1985 Aug 15;230(1):43-52

[361] Ueland PM, Helland S, S-adenosylhomocysteinase from mouse liver. Catalytic properties at cellular enzyme level. The Journal of biological chemistry 1980 Aug 25;255(16):7722-7

[362] Fumi K, Beluzi R, Cuk M, Pavkov T, Kloor D, Bari I, Miji I, Vugrek O, Functional analysis of human S-adenosylhomocysteine hydrolase isoforms SAHH-2 and SAHH-3. European journal of human genetics : EJHG 2007 Mar;15(3):347-51

[363] Mehdi S, Jarvi ET, Koehl JR, McCarthy JR, Bey P. The mechanism of inhibition of S-adenosyl-L-homocysteine hydrolase by fluorine-containing adenosine analogs. J Enzyme Inhib. 1990;4(1):1-13.

[364] Liu S, Wnuk S F, Yuan C, Robins M J, Borchardt R T, Adenosine-5'-carboxaldehyde: a potent inhibitor of S-adenosyl-L-homocysteine hydrolase, *J. Med. Chem.*, 1993, *36* (7), pp 883–887

[365] Wang X, Qin X, Demirtas H, et al. Efficacy of folic acid supplementation in stroke prevention: a meta-analysis. Lancet 2007;369:1876–82.

[366] Andersson, A., Brattstrom, L., Israelsson, B., Isaksson, A., Hamfelt, A., and Hultberg, B. (1992) Plasma homo- cysteine before and after methionine loading with regard

"judging causality in the face of inconclusive trial evidence," Wald et al.[367] examined evidence from the meta-analysis of cohort studies, the genetic polymorphism studies and the randomized intervention trials. They concluded that "the summary estimate from the trials is consistent with a short-term protective effect [of homocysteine lowering] of 12% on ischaemic heart disease events and 22% on stroke, or a larger long-term effect."

Another study by Burke et al.[368] comparing post-mortem serum Hcy levels of men who died suddenly of severe CAD and controls showed that serum Hcy levels were elevated in men who died with coronary thrombus. This study found that the risk was increased if there was concomitant diabetes mellitus and the presence of fibrous plague atheromas.

Several other studies[369] have identified moderate Hcy as an independent risk factor for atherosclerotic disease (atherosclerosis is a chronic inflammatory disease of the arteries, in which deposits of fatty acid substances, cholesterol, calcium and other substances build up in the endothelial layer of the arteries). Current research has also identified the link between fluoride and atherosclerosis. [370]

It has been hypothesised that advanced vascular disease is accompanied by depletion of thioretinaco ozonide from cellular membranes.[371] It has been documented that this substance is also implicated in oxidative metabolism.[372] Fluoride has been known as a metabolic inhibitor for many years, causing inhibition of oxidative metabolism and decreased ATP synthesis.[373],[374],[375],[376]

Fluoride is believed to inhibit the activity of many enzymes by disrupting the

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to age, gender, and menopausal status. *European Journal Clinical Investigation*, 22(2), 79-87

[367] Wald DS, Morris JK, Law M, Wald NJ. Folic acid, homocysteine, and cardiovascular disease: judging causality in the face of inconclusive trial evidence. BMJ 2006;333:1114–7

[368] Burke AP, Fonseca V, Kolodgie F, Zieske A, Fink l, Virmani R. Increased serium homocysteine and sudden death resulting from coronary atheroscerosis with fibrous plaques. Arterioscler Thromb Vasc Biol 2002; 22:1936-1941.

[369] Weiss N, Keller C, Hoffman U, Loscalzo j, Endothelial Dysfuntion And Atherothrombosis In Mild Hyperhomocysteinemia. Vasc Med 2002; 7:227-239.

[370] Li Y, Berenji G R, Shaba W F, Tafti B, Yevdayev E, Dadparvar S. Association of vascular fluoride uptake with vascular calcification and coronary artery disease. Nucl Med Commun. 2012 Jan;33(1):14-20.

[371] McCully KS. Chemical pathology of homocysteine. I. Atherogenesis Ann Clin Lab Sci 1993;23:477–93

[372] McCully KS. Chemical pathology of homocysteine. II. Carcinogenesis and homocysteine thiolactone metabolism Ann Clin Lab Sci. 1994;24:27

[373] Yiamouyiannis J. Fluoride: the Aging Factor. Health Action Press, Delaware, Ohio, 1993; pp 26–33, 94–99.

[374] W. L. Gabler and P. A. Leong, ., " *Fluoride Inhibition of Polymorphonumclear Leukocytes*," Journal of Dental Research, Vol. 48, No. 9, pp. 1933-1939 (1979);

[375] A. S. Kozlyuk, et al., " *Immune Status of Children in Chemically Contaminated Environments*," Zdravookhranenie, Issue 3, pp. 6-9 (1987)

[376] D. J. Newell, "Fluoridation of Water Supplies and Cancer - An Association?," Applied Statistics, Vol. 26, No. 2, pp. 125-135 (1977)

molecular structure of proteins by interfering with normal hydrogen bonding. In addition, the effects of fluoride on chromosomal structure, genetic damage and carcinogenesis may be attributable to inhibition of DNA repair enzyme structure.[377] Current research examining the chemical pathology of homocysteine in atherogenic heart disease[378], oxidative metabolism, and carcinogenesis[379] has found that depletion of thioretinaco ozonide from cellular membranes is suggested to underlie the carcinogenic and atherogenic effects of fluoride and other electrophilic carcinogens.[380]

It is clear based on recent scientific findings that the ability of fluoride to inhibit homocystein metabolism in the body may have very significant implications for preventative healthcare and public expenditure on healthcare in Ireland. No study has been undertaken in Ireland to assess the impact of water fluoridation on the incidence of atherosclerotic disease in high risk communities. This is especially critical in geographic areas with low calcium and magnesium levels in drinking water where the concentration of fluoride ions and fluoridation derivative compounds would have a markedly increased toxicity and bio-availability. It is not surprising that it has been found that the southern region of Ireland representing Cork city, West Cork and West Kerry, has the highest incidence of premature mortality from cardiovascular disease as well as certain cancers in Ireland. It is extremely alarming that there is no reference in any public health reports in Ireland highlighting the association and risk factor of low calcium and magnesium in drinking water with development of these diseases. It is even more disturbing that the cumulative additional risk of injecting silicafluorides, containing the most active and reactive chemical element fluoride, as well as its derivative compounds, into public water supplies and the inherent health risks that may be associated with this practice have not been examined.

[377] Yiamouyiannis J. Fluoride: the Aging Factor. Health Action Press, Delaware, Ohio, 1993; pp 26–33, 94–99.

[378] Atherogenic heart disease involves the formation of plaques in arterial walls that narrow the arterial passage, restricting blood flow and increasing the risk of heart attack, as well as stroke.

[379] Carcinogenesis is the process of initiating and promoting cancer. Mosby's Medical Dictionary, 8th edition. 2009.

[380] Kilmer S. McCully , Chemical Pathology of Homocysteine. IV. Excitotoxicity, Oxidative Stress, Endothelial Dysfunction, and Inflammation, *Annals of Clinical & Laboratory Science* 39:219-232 (2009)

7.3 Homocysteine Metabolism and Fluoride: Significance for Congenital Heart Defects, Down Syndrome, Neurological and Angiogenic Diseases

There is little dietary intake or excretion of Homocysteine (Hcy); virtually all of the Hcy in the body is formed and removed by endogenous metabolism.[381] Homocysteine metabolism has been identified as a risk factor in congenital heart defects. Meta-analysis research has demonstrated that maternal Hcy was significantly associated with an increased risk of having a child with a Congenital Heart Defect (CHD).[382],[383,]

Homocysteine metabolism is associated with neurological diseases including neural tube defect.[384],[385,386] Neural tube defects (NTD) are among the most common birth defects worldwide.

Homocysteine metabolism has been documented as a risk factor of Down Syndrome (DS). An elevated risk for DS has been observed with irregular homocysteine metabolism.[387] Research has demonstrated a positive relationship between homocysteine levels and increased hostile behaviour in schizophrenia.[388]

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[381] Yap S. Classical homocystinuria: Vascular risk and its prevention. J Inherit Metab Dis. 2003;26:259-265

[382] Verkleij-Hagoort A, Bliek j, Sayed-Tabatabaei F, Ursem n, Steegers E, Steegers-Theunissen R. Hyperhomocysteine-mia and MTHFR polymorphisms in association with orofacial clefts and congenital heart defects: a meta-analysis. Am J Med Genet A 2007; 143A:- 952-960.

[383] Botto LD, Correa , Erickson JD. Racial and temporal variations in the prevalence of heart defects. Pediatrics 200; 107:E32.

[384] Steegers-Theunissen RP, Boers GH, Trijbels fj. Finkelstein jd, Blom hj, Thomas CM, et al. Maternal hyperhomocysyeinemia: a risk factor for neural-tube defects? Metabolism 1994; 43: 1475-1480

[385] Ratan SK, Rattan KN, Pandey RM, Singhal S, Kharab S, Bala M, Singh V, Jhanwar A, Evaluation of the levels of folate, vitamin B12, homocysteine and fluoride in the parents and the affected neonates with neural tube defect and their matched controls. Pediatr Surg Int. 2008 Jul;24(7):803-8. Epub 2008 May 8.

[386] Brustolin S, Guigliana R, Felix T.T, Genetics of homocysteine metabolism and associated disorders. Brazialian Journal of Medical and Biological Research (2010) 43: 1-7

[387] Bosco P, Gueant-Rodriguez RM, Anello G, Barone C, Namour F, Caraci F, et al. Methionine synthase (MTR) 2756 (A --> G) polymorphism, double heterozygosity methionine synthase 2756 AG/methionine synthase reductase (MTRR) 66 AG, and elevated homocysteinemia are three risk factors for having a child with Down syndrome. Am J Med Genet A. 2003 Sep 1;121A(3):219-24.

[388] Panagiotakos DB, Pitsavos C, Chrysohoou C, Tsetsekou E, Papageorgiou C, Christodoulou G, Stefanadis C. Increased plasma homocysteine concentrations in healthy people with hostile behavior: the ATTICA study. Med Sci Monit. 2004 Aug;10(8):CR457-62. Epub 2004 Jul 23.

Young male schizophrenic patients were found to have elevated homocysteine levels as were young male patient with bipolar disorder.[389] Elevated homocysteine levels have also been found to be associated with depression. Recent research demonstrated that major depression in patients with moderate Alzheimer's disease (AD) was associated with higher plasma homocysteine levels.[390] Three recent case-control studies, from the United Kingdom[391],[392] and Sweden[393], have reported a correlation between AD and high homocysteine levels. Homocysteine may also be an important factor in Parkinson's disease. Kuhn et al.[394],[395] found elevated of plasma homocysteine in Parkinsonian patients. Furthermore, it has been found that a high plasma concentration of homocysteine may contribute to epilepsy.[396] This is of critical importance as Ireland has one of the highest incidences of epilepsy in the world.[397],[398]

It has been found that homocysteine (Hcy) inhibits DNA methylation and endothelial cell (EC) growth, resulting in a disturbance of the angiogenic balance of the body and reduced formation of new blood vessels essential for fetal development, tissue regeneration and wound healing. Angiogenesis (the formation of new blood vessels) also plays a supporting role in pathological conditions such as solid tumour growth, rheumatoid arthritis, and diabetic retinopathy.399,400 Inhibition of angiogenesis reduces blood vessel

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[389] Levine J, Sela BA, Osher Y, Belmaker RH. High homocysteine serum levels in young male schizophrenia and bipolar patients and in an animal model. Prog Neuropsychopharmacol Biol Psychiatry. 2005 Sep;29(7):1181-91.

[390] Sheng C C, Chuan M C, Chun Y Y, Han Y Y, Lian L C, Fang Y C, Kuan L C, Cheng L Y, Plasma Homocysteine Levels and Major Depressive Disorders in Alzheimer Disease, American Journal of Geriatric Psychiatry: November 2010 - Volume 18 - Issue 11 - pp 1045-1048.

[391] Clarke R, Smith AD, Jobst KA, Refsum H, Sutton L, Ueland PM. Folate, vitamin B12, and serum total homocysteine levels in confirmed Alzheimer disease. Arch Neurol. 1998;55:1449-1455.

[392] McCaddon A, Davies G, Hudson P, Tandy S, Cattell H. Total serum homocysteine in senile dementia of Alzheimer type. Int J Geriatr Psychiatry. 1998;13:235-239.

[393] Lehmann M, Gottfries CG, Regland B. Identification of cognitive impairment in the elderly: homocysteine is an early marker. Dement Geriatr Cogn Disord. 1999; 10:12-20.

[394] Kuhn W, Roebroek R, Blom H, et al. Elevated plasma levels of homocysteine in Parkinson's disease. Eur Neurol. 1998;40:225-227.

[395] Muller T, Werne B, Fowler B, Kuhn W. Nigral endothelial dysfunction, homocysteine, and Parkinson's disease. Lancet. 1999;354:126-127.

[396] Schwarz S, Zhou G-Z. N-methyl-D-aspartate receptors and CNS symptoms of homocystinuria. Lancet. 1991;337:1226-1227.

[397] Linehan, C,. Walsh, P A,. The prevlance of Epilepsy in Ireland, Brainwave The Irish Epilepsy Association. 2009

[398] L Forsgren, E Beghi, A Oun, M Sillanpää. The epidemiology of epilepsy in Europe - a systematic review. European Journal of Neurology Volume 12. Issue: 4, Pages 245-253

[399] Jamaluddin, M,S,. Chen,I,. Yang,F,. Jiang, X., Jan, M., Liu, X., Schafer, A,I,. Durante, W., Yang, X,. Wang, H. Homocysteine inhibits endothelial cell growth via DNAhypomethylation of the*cyclin A* gene, Blood Journal 2007 110: 3648-3655

[400] Joseph F. Murphy and Desmond. J. Fitzgerald, Vascular endothelial cell growth factor (VEGF) induces cyclooxygenase (COX)-dependent proliferation of endothelial

growth and collateral arteries formation reducing blood flow and oxygen delivery within the body that can lead to cardiac ischaemia, as well as peripheral artery disease. The absence of blood vessels in a repairing or otherwise metabolically active tissue may inhibit repair or other essential functions. The damage is the result of the build-up of metabolic waste products, inability to maintain cell membranes, mitochondrial damage, and eventual leakage of autolyzing proteolytic enzymes into the cell and surrounding tissues. Angiogenesis is also important in leukocyte extravasation and thus the pathogenesis of inflammatory rheumatoid arthritis.[401]

DNA methylation is an important epigenetic mechanism that selectively regulates gene expression, and is associated with cancer development and cardiovascular disease.[402],[403] Regulation of gene expression whereby neurons and neuronal networks adapt their short- and long-term responses to environmental stimuli represents a major component of neurological disease.[404] A limited number of genes, estimated to be in the range of 15 to 300, show activity-dependent upregulation in the nervous system, whereas the number of genes down-regulated is much lower, suggesting that gene induction is the favoured process for long-term neuronal adaptations.[405]

Activation of gene expression was shown to be involved in a large variety of processes in both the developing and mature nervous system, including proliferation of neuronal precursors, outgrowth of neuronal processes, learning and memory in invertebrates and vertebrates, induction of neurotrophic and neuroprotectant cellular programs, and regulation of circadian rhythms.[406],[407],[408],[409],[410]

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cells (EC) via the VEGF-2 receptor, *The FASEB Journal* express article 10.1096/fj.00-0757fje. Published online May 29, 2001.

[401] Z Szekanecz; G Szegedi; A E Koch, Angiogenesis in rheumatoid arthritis: pathogenic and clinical significance. Journal of investigative medicine : the official publication of the American Federation for Clinical Research 1998;46(2):27-41.

[402] Hiltunen MO, Yla-Herttuala S. DNA methylation, smooth muscle cells, and atherogenesis. Arterioscler Thromb Vasc Biol. 2003;23:1750-1753.

[403] Dong C, Yoon W, Goldschmidt-Clermont PJ. DNA methylation and atherosclerosis. J Nutr. 2002; 132:2406S-2409S.

[404] Tardito D, Perez J, Tiraboschi E, Musazzi L, Racagni G, Popoli, M. Signalling Pathways Regulating Gene Expression, Neuroplasticity, and Neurotrophic Mechanisms in the Action of Antidepressants: A Critical Overview, PHARMACOLOGICAL REVIEWS Vol. 58, No. 1

[405] West AE, Griffith EC, and Greenberg ME (2002) Regulation of transcription factors by neuronal activity. *Nature Rev Neurosci* **3:**921–931.

[406] Kandel ER (2001) The molecular biology of memory storage: a dialogue between genes and synapses. *Science (Wash DC)* **294:**1030–1038.

[407] Mabuchi T, Kitagawa K, Kuwabara K, Takasawa K, Ohtsuki T, Xia Z, Storm D, Yanagihara T, Hori M, and Matsumoto M (2001) Phosphorylation of cAMP response element-binding protein in hippocampal neurons as a protective response after exposure to glutamate in vitro and ischemia in vivo. *J Neurosci* **21:**9204– 9213

[408] Reppert SM and Weaver DR (2001) Molecular analysis of mammalian circadian rhythms. *Annu Rev Physiol* **63:**647–676.

[409] Kida S, Josselyn SA, de Ortiz SP, Kogan JH, Chevere I, Masushige S, and Silva AJ

Fluoride exposure may therefore be seen to play a leading role in the development of both neurological and angiogenic diseases.

Undoubtedly however the most significant finding is that fluoride inhibits AdoHydrae and homocysteine metabolism which is linked to cardiovascular disease, atherosclerotic disease, congenital heart defects, Down Syndrome, neurodegenerative disorders including depression, schizophrenia, bi-polar disorder, epilepsy, behavioural disorders, Alzheimer's disease and carcinogenesis. Given the enormous healthcare, financial and social impact implications, the significance of any association between fluoride and homocysteine metabolism and increased risk of genetic abnormalities, neurological or angiogenic disease cannot be underestimated. These findings are of major significance demanding, in the interests of public health, an immediate response from the State requiring the cessation of the water fluoridation policy without delay.

7.4 Lipid Peroxidation and Fluoride

Another important, although indirect, mechanism of fluoride-induced cytotoxicity demonstrated in the cells of many tissues is lipid peroxidation (LPO). Lipid peroxides occur in enzymatic or non-enzymatic reactions involving activated chemical species known as "reactive oxygen species" (ROS).

A direct link between fluoride-induced apoptosis and elevated lipid peroxidation (LPO) has been demonstrated in both human and animal studies.[411,412,413,414,415,416,417,418,419,420,421,422]

(2002) CREB required for the stability of new and reactivated fear memories. *Nat Neurosci* **5:**348–355

[410] Pittinger C, Huang YY, Paletzki RF, Bourtchouladze R, Scanlin H, Vronskaya S, and Kandel ER (2002) Reversible inhibition of CREB/ATF transcription factors in region CA1 of the dorsal hippocampus disrupts hippocampus-dependent spatial memory. *Neuron* **34:**447–462.

[411] Y. M. Shivarajashankara, A. R. Shivashankara, B. P. Gopalakrishna, S. H. Rao, "Oxidative stress in children with endemic skeletal fluorosis", *Fluoride*, vol. 34, pp. 108–113, 2001

[412] P. Kalyanalakshmi, M. Vijayabhaskar, M.D. Naidu, "Lipid peroxidation and antioxidant enzyme status of adult males with skeletal fluorosis in Andhra Pradesh, India", *Fluoride*, vol. 40, pp. 42-45, 2007.

[413] Y. M. Shivarajashankara, A. R. Shivashankara, P. G. Bhat, S. H. Rao, "Effect of fluoride intoxication on lipid peroxidation and antioxidant systems in rats", *Fluoride*, vol. 34, pp. 108-113, 2001.

[414] I. Inkielewicz, J. Krechniak, "Fluoride effects on glutathione peroxidase and lipid peroxidation in rats", *Fluoride*, vol. 37, pp. 7-12, 2004.

[415] I. Blaszczyk, E. Grucka-Mamczar, S. Kasperczyk, E. Birkner, "Influence of methionine upon the concentration of malondialdehyde in the tissues and blood of rats exposed to sodium fluoride", *Biological Trace Element Researc*h, vol. 129, pp. 229-238, 2009.

7.5 Antioxidant Enzymes Activity, Muscle, Brain Function and Fluoride

Alongside aforementioned oxidative stress-related events, fluoride is proven to inhibit many enzymes such as those involved in the pentose pathway and antioxidant defense system.[423],[424],[425]

A decrease in blood free radical scavenging enzymes was found in the blood of people living in areas of high fluoride exposure.[426],[427],[428]

A similarly significant decrease was observed in animals after long-term high-fluoride intake.[429],[430],[431],[432] It has also been demonstrated that fluoride affects

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[416] E. Karaoz, M. Oncu, K. Gulle, M. Kanter, F. Gultekin, S. Karaoz, E. Mumcu, "Effect of chronic fluorosis on lipid peroxidation and histology of kidney tissues in first- and second-generation rats", *Biological Trace Element Researc*h, vol. 102, pp. 199-208, 2004.

[417] A. X. Zhan, M. Wang, Z. R. Xu, W. F. Li, J. X. Li, "Evaluation of caspase-dependent apoptosis during fluoride-induced liver lesion in pigs", *Archives of Toxicology*, vol. 80, pp. 74–80, 2006

[418] L. F. He, J. G. Chen, "DNA damage, apoptosis and cell cycle changes induced by fluoride in rat oral mucosal cells and hepatocytes", *World Journal of Gastroenterology*, vol. 21, pp. 1144-1148, 2006.

[419] Z.-H. Wang, X.-L. Li, Z.-Q. Yang, M. Xu, "Fluoride-induced apoptosis and lipid peroxidation in human hair follicles in vitro", *Biological Trace Element Research*, vol. 137, pp. 280-288, 2010.

[420] Inkielewicz I, Rogowska M, Krechniak J. Lipid peroxidation and antioxidant enzyme activity in rats exposed to fluoride and ethanol. Fluoride 2006;39:53-9.

[421] Guan ZZ, Xiao KQ, Zeng XY, Long YG, Cheng YH, Jiang SF, et al. Changed cellular membrane lipid composition and lipid peroxidation of kidney in rats with chronic fluorosis. Arch Toxicol 2000;74:602-8

[422] Shanthakumari D, Srinivasalu S, Subramanian S. Effect of fluoride intoxication on lipid peroxidation& antioxidant status in experimental rats. Toxicology2004;204:219-28.

[423] Carlson, J. R., and Suttie, J. W. (1966). Pentose phosphate pathway enzymes and glucose oxidation in fluoride-fed rats. *Am. J. Physiol.* 210, 79–83.

[424] Park, S., Ajtai, K., and Burghardt, P. (1999). Inhibition of myosin ATPase by metal fluoride complexes. *Biochem. Biophys. Acta* 1430, 127–140.

[425] Vani, M. L., and Reddy, K. P. (2000). Effects of fluoride accumulation on some enzymes of brain and gastrocnemius muscle of mice. *Fluoride* 33,17–26.

[426] Y. M. Shivarajashankara, A. R. Shivashankara, B. P. Gopalakrishna, S. H. Rao, "Oxidative stress in children with endemic skeletal fluorosis", *Fluoride*, vol. 34, pp.108–113, 2001

[427] P. Kalyanalakshmi, M. Vijayabhaskar, M.D. Naidu, "Lipid peroxidation and antioxidant enzyme status of adult males with skeletal fluorosis in Andhra Pradesh, India", *Fluoride*, vol. 40, pp. 42-45, 2007.

[428] J. Li, S. Ca, "Recent studies on endemic fluorosis in China", *Fluoride*, vol. 27, pp. 125-128, 1994.

[429] Y. M. Shivarajashankara, A. R. Shivashankara, P. G. Bhat, S. H. Rao, "Effect of fluoride intoxication on lipid peroxidation and antioxidant systems in rats", *Fluoride*, vol. 34, pp. 108-113, 2001.

[430] Y. M. Shivarajashankara, A. R. Shivashankara, P. G. Bhat, S. H. Rao, "Lipid peroxidation and antioxidant systems in the blood of young rats subjected to chronic fluoride toxicity", *Indian Journal of Experimental Biology*, vol. 41, pp. 857-860, 2003.

[431] Y. M. Shivarajashankara, A. R. Shivashankara, P. G. Bhat, S. H. Rao, "Lipid peroxidation and antioxidant systems in the blood of young rats subjected to chronic

the brain and muscle by inhibiting some enzymes associated with energy production and transfer, membrane transport, and synaptic transmission.[433],[434],[435]

7.6 ATP Depletion and Fluoride

Fluoride has been known as a metabolic inhibitor for many years, causing inhibition of cellular respiration (oxidative metabolism) and decreased ATP synthesis.[436] Inhibition of ATP synthesis would result in increased ATP synthase. Increased ATP synthase is associated with tumour tissues, breast cancer cells and has been reported to induce many biological effects like angiogenesis.[437],[438] Angiogenesis has been demonstrated to be essential to the development of a tumour.[439]

Fluoride is a well-known inhibitor of enzymes of glycolytic pathway.[440] ATP is generated by glycolysis and oxidative phosphorylation. ATP depletion can arrest many crucial cellular functions like transmembrane ion transport and protein phosphorylation, disturb membrane potential and membrane-cytoskeleton interactions, what may finally lead to cell death.[441],[442]

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fluoride toxicity", *Indian Journal of Experimental Biology*, vol. 41, pp. 857-860, 2003.

[432] M. Sinha, P. Manna, P. C. Sil, "A 43kDa protein from the herb, *Cajanus indicus* L., protects against fluoride induced oxidative stress in mice erythrocytes", *Pathophysiology*, vol. 14, pp. 47-54, 2007.

[433] Krishnamachari, K. A. V. R., and Krishnaswamy, K. (1973). Genu valgum and osteoporosis in an area of endemic fluorosis. *Lancet* **2,** 877–879

[434] Shashi, A., Singh, J. P., and Thaper, S. P. (1994). Effect of long-term administration of fluoride on levels of protein, free amino acids, and RNA in rabbit brain. *Fluoride* **27,** 155–159.

[435] Vani, M. L., and Reddy, K. P. (2000). Effects of fluoride accumulation on some enzymes of brain and gastrocnemius muscle of mice. *Fluoride* **33,** 17–26.

[436] Yiamouyiannis J. Fluoride: the Aging Factor. Health Action Press, Delaware, Ohio, 1993; pp 26–33, 94–99.

[437] Ma, Z,. Cao, M,. Liu, Y,.He, Y,.Wang, Y, Yang, C,. Wang, W,.Du,Y,. Zhou, M,. and Feng Gao, Mitochondrial F1Fo-ATP synthase translocates to cell surface in hepatocytes and has high activity in tumor-like acidic and hypoxic environment. Acta Biochim Biophys Sin (2010) 42 (8): 530-537

[438] S M Rumjahn, N Yokdang, K A Baldwin, J Thai and I L O Buxton. Purinergic regulation of vascular endothelial growth factor signaling in angiogenesis. British Journal of Cancer (2009) 100, 1465–1470.

[439] Chan, D,A,. Kawahara,T,A, .Sutphin P,D, Chang, H, Y,. Chi,J,T,. Giaccia1, A, J. Tumor Vasculature Is Regulated by PHD2-Mediated Angiogenesis and Bone Marrow-Derived Cell Recruitment, Cancer Cell 15, 527–538, June 2, 2009

[440] J. Qin, G. Chai, J. M. Brewer, L. L. Lovelace, L. Lebioda, "Fluoride inhibition of enolase: crystal structure and thermodynamics", *Biochemistry*, vol. 45, pp. 793-800, 2006.

[441] O. Barbier, L. Arreola-Mendoza, L. M. Del Razo, "Molecular machanisms of fluoride toxicity", *Chemico-Biological Interactions*, vol. 188, pp. 319-333, 2010.

[442] E. F. Mason, J. C. Rathmell, "Cell metabolism: an essential link between cell growth and apoptosis", *Biochimica et Biophysica Acta*, vol. 1813, pp. 645-654, 2011.

Agalakova and Gusev[443] suggested that the role of fluoride in ATP depletion might be the primary process in the chain of events impairing Glutathione (GSH) replenishment and antioxidant defence in the human body. These findings support the hypothesis that inhibition of cellular ATP formation is a crucial event in the progression of irreversible cell injury.

It has been reported that ATP depletion may act to alter endothelial barrier function.[444] Endothelial dysfunction, or the loss of proper endothelial function, is a hallmark for vascular diseases, and is often regarded as a key early event in the development of atherosclerosis.

Impaired endothelial function, causing hypertension and thrombosis, is often seen in patients with coronary artery disease, diabetes mellitus, hypertension, and hypercholesterolaemia. Endothelial dysfunction has also been shown to be predictive of future adverse cardiovascular events, and is also present in inflammatory disease such as rheumatoid arthritis and systemic lupus erythematosus. Systemic lupus erythematosus (SLE) is a long-term auto-immune disorder that may affect the skin, joints, kidneys, brain and other organs. SLE is an auto-immune disease, which means the body's immune system mistakenly attacks healthy tissue. This leads to long-term (chronic) inflammation.[445]

The significance of fluoride as a metabolic inhibitor of ATP, with resultant increased risk of developing tumours and cancers and well as its importance in endothelial dysfunction in developing coronary artery disease, diabetes mellitus and other critical neurological complications like hypertension cannot be underestimated. These findings are of major significance demanding, in the interests of public health and safety, an immediate response from the State requiring the cessation of the water fluoridation policy without delay.

[443] Agalakova, N, I, Gusev, G, P,. Molecular mechanisms of cytotoxicity and cell death induced by inorganic fluoride, Sechenov Institute of Evolutionary Physiology and Biochemistry, Russian Academy of Sciences.

[444] Wilson, J,.Winter, M,. Shasby M,D,. Oxidants, ATP Depletion, and Endothelial Permeability to Macromolecules Blood, Vol 76, No 12 (December 15). 1990: pp 2578-2582

[445] U.S National Library of Medicine

7.7 Fluoride Inhibition of Cellulose Digestion

Chamberlain et al.[446] demonstrated that fluoride inhibits cellulose digestion and that it would be possible even for a low level of fluoride to result in partial blocking of normal cellulose degradation. This study also demonstrated that fluoride interferes with magnesium and manganese ions with consequences for rumen microorganisms.

Fluoride has also been documented by the U.S. Agency for Toxic Substances and Disease 2004 (ATSDR) as a general inhibitor of the energy production of the human cell, and of glycolysis in particular[447]. The ATSDR further reported that *"although much is known about enzyme inhibition by fluoride, the human health significance remains to be known."*

Alarmingly, it has been reported that inhibition of ATP and glycolysis are common characteristics of malignant cells.[448]

7.8 Vitamin C Deficiency and Fluoride

It is also worth noting the link between fluoride and other micronutrients; in particular Vitamin C. Vitamin C is an essential nutrient for humans and is required by the body to form collagen in bones, cartilage, muscle and blood vessels. It also aids in the absorption of iron.[449]

It has been documented that in the absence of sufficient Vitamin C, water fluoridation will lead to Vitamin C depletion, dental fluorosis and to abnormal levels of metabolites in blood tissues.[450] Animal studies have also demonstrated the link between Vitamin C and fluoride intake.[451] The findings of this latter study support the previous observations noted by Yiamouyiannis. This fact should be of particular interest to the Irish health authority as a national health survey has documented the inadequacy of Vitamin C intake in boys and girls aged from 5 to 12 years in Ireland.[452] A more recent study among adults aged 65 years and over, similarly showed a low intake of Vitamin C in addition to inadequate intakes of calcium, Vitamin A and Vitamin D.[453]

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[446] C. C. Chamberlain and Wise Burroughs, Effect of Fluoride, Magnesium and Manganese Ions on in Vitro *Journal of Animal Science* 1962, 21:428-432.
[447] U.S. Agency for Toxic Substances and Disease 2004.
[448] BISWAS S, RAY M, MISRA S, DUTTA D P, RAY S. Selective inhibition of mitochondrial respiration and glycolysis in human leukaemic leucocytes by methylglyoxal, Biochem. J. (1997) 323, 343±348
[449] Mayo Foundation for Medical Education and Research
[450] Vitamin C and Fluoridation- John A. Yiamouyiannis Ph.D.
[451] Verma RJ, Sherlin DM , Fluoride and Vitamin C, Human & Experimental Toxicology, 2001 Dec;20(12):619-23.
[452] Irish Universities Nutrition Alliance (IUNA), 2001, Adequacy of micronutrient Intakes in Ireland, Results from the National Food Consumption Surveys,
[453] Irish Universities Nutrition alliance (2008), National Teens Food Survey

Alarmingly, it is possible that water fluoridation may itself be contributing to Vitamin C depletion as well as dental fluorosis in Ireland. Interestingly, it has also been observed that in low fluoride areas, dietary supplementation with Vitamin C leads to fluoride deposition in teeth equal to that of higher fluoride areas with water fluoridation.[454,455] It is plausible therefore, to assume, that exposure to fluoride compounds in drinking water may increase the associated risk of Vitamin C depletion, a contributory factor associated in the development of both dental fluorosis and periodontal disease.

Indeed the coexistence of fluorosis and signs of Vitamin C deficiency have been reported[456,457] and it has been suggested that low concentrations of Vitamins A, C and D tend to worsen the symptoms of fluorosis and accelerate the development of fluoride toxicosis.[458]

7.9 Periodontal Disease and Fluoride

Periodontal disease is one of the two major dental diseases that affect human populations worldwide at high prevalence rates.[459,460] Periodontal diseases constitute one of the major global oral health burdens and periodontitis remains a major cause of tooth loss in adults worldwide. The World Health Organization recently reported that severe periodontitis exists in 5-20% of adult populations.[461]

A recent University of Southern California research study[462] indicates that having periodontal disease before age 35 increases the risk of developing Alzheimer's disease in later life.
Periodontal disease is associated with bone loss around the alveolar bone (housing the teeth) and tooth loss. It is present in 90% of individuals over the

454 D. J. Thompson, P. H. Phillips, Journal of Dental Research. 45, 845 (1966).
455 D. Triers, C.G. Elliott, M.D. Smith, J. K. Dent. Res. 47, 1171 (1968).
456 Krishnamachari K. A. V. R, and Laxmaiah N., Lack of effect of massive dose of vitamin C on fluoride excretion in fluorosis during a short clinical trial, The American Journal of Clinical Nutrition 28: November 1975, pp. 1234-1236.
457 Pandit, C. G., I. N. S. Raghavachari, D. S. Rao And V. Krishnamurthy. Endemic fluorosis in South India. A study of the factors involved in the production of mottled anamel in children and severe bone manifestations in adults. Indian J. Med. Res. 28: 559, 1940.
458 Suttie JW, Phillips PH: Fluoride ingestion and vitamin metabolism. In Fluorine and Dental Health: the Pharmacology and Toxicology of Fluorine. JC Muhler and MK Hine eds. Bloomingtonl, N: Indiana University Press, 1959, pp 70-77.
459 Petersen PE. The World Oral Health Report 2003: Continuous improvement of oral health in the 21st century - The approach of the WHO Global Oral Health Programme. Community Dent Oral Epidemiol 2003;31(Suppl. 1):3-24.
460 Papapanou PN. Epidemiology of periodontal diseases: An update. J Int Acad Periodontol 1999;1:110-116.
461 Jin LJ, Armitage GC, Klinge B, Lang NP, Tonetti M, Williams RC. Global oral health inequalities: task group--periodontal disease. Adv Dent Res. 2011 May;23(2):221-6.
462 Gatz M, Mortimer J A, Fratiglioni L, Johansson B, Berg S, Reynolds C A, Pedersen N L. Potentially modifiable risk factors for dementia in identical twins , Alzheimer's & Dementia: The Journal of the Alzheimer's Association Volume 2, Issue 2 , Pages 110-117, April 2006

age of 65. It has been reported that the potential mechanisms by which host factors may influence onset and progression of periodontal disease directly or indirectly include underlying low bone density in the oral cavity, bone loss, genetic susceptibility and exposure to risk factors. [463]

It has been reported that fluoride decreases cortical bone mineral density and increases skeletal fragility.[464],[465] Further studies by Philips et al.[466] reported a negative association between drinking fluoridated water and cortical bone mass and concluded that *"increased fluoride may in fact be detrimental to cortical bone mass.*" Consequently, as the jaw bone is composed of cortical bone, it is evident that exposure to fluoride may be considered a risk factor for the onset of periodontal disease.

It is alarming therefore that the European Food Safety Authority (EFSA) has stated that an infant's retention of fluoride in bone can be as high as 90% of the absorbed amount.[467]

Several studies have shown that osteoporosis is also associated with jaw bone quality, bone density,[468] the condition of the inferior mandibular cortex,[469][470] and the alveolar bone condition.[471] There is increasing evidence also that osteoporosis, and the underlying loss of bone mass characteristic of this disease, is associated with periodontal disease and tooth loss. Current evidence, including several prospective studies, supports an association of osteoporosis with the onset and progression of periodontal disease in humans. [472] Systemic loss of bone density in osteoporosis, including that of the oral cavity, may provide a host system that is increasingly susceptible to infectious

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[463] Wactawski-Wende J. Periodontal diseases and osteoporosis: association and mechanisms. Ann Periodontol. 2001 Dec;6(1):197-208.

[464] Riggs BL, et al. (1990). Effect of Fluoride treatment on the Fracture Rates in Postmenopausal Women with Osteoporosis. New England Journal of Medicine. 322:802-809.

[465] Sogaard CH, et al. (1995). Effects of fluoride on rat vertebral body biomechanical competence and bone mass. Bone. 16(1): 163-9.

[466] Phipps KR, Burt BA. (1990). Water-borne fluoride and cortical bone mass: A comparison of two communities. Journal of Dental Research 69: 1256-1260.

[467] Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Fluoride. The EFSA Journal (2005) 192, 1-65

[468] Jacobs R, Ghyselen J, Koninckx P, van Steenberghe D. Long-term bone mass evaluation of mandible and lumbar spine in a group of women receiving hormone replacement therapy. *Eur J Oral Sci* 1996; 104:10-16.

[469] Lee K, Taguchi A, Ishii K, Suei Y, Fujita M, *et al*. Visual assessment of the mandibular cortex on panoramic radiographs to identify postmenopausal women with low bone mineral densities. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2005;100:226-31

[470] Nishida M, Grossi SG, Dunford RG, Ho AW, Trevisan M. Calcium and the risk for periodontal disease. *J Periodontol* 2000; 71:1057-66.

[471] Taguchi A, Tsuda M, Ohtsuka M, Kodama I, Sanada M, *et al*. Use of dental panoramic radiographs in identifying younger postmenopausal women with osteoporosis. *Osteoporosis Int* 2006; 17:387-94.

[472] Wactawski-Wende J. Periodontal diseases and osteoporosis: association and mechanisms. Ann Periodontol. 2001 Dec;6(1):197-208.

destruction of periodontal tissue.[473] Both periodontal disease and osteoporosis are serious public-health concerns. The association between fluoride and osteoporosis is well documented. Understanding the association between fluoride exposure and bone quality and the mechanisms underlying those associations is critically important to prevent, diagnose and treat these very common diseases.

As fluoride has been demonstrated to effect bone health and density negatively, it is plausable therefore to assume that exposure to fluoride through ingesting fluoridated water or foods is therefore likely to increase the associated risk of developing periodontal disease.

It is accepted that at least 50% of ingested fluoride is taken up by bone, and that fluoridation of water can increase normal dietary intake by about 50%.[474] It is known that fluoride affects bone in at least two ways—fluoride ions can replace hydroxyl ions in the hydroxyapatite lattice, and increased fluoride concentrations in plasma directly increase osteoblastic differentiation and activity. Such changes could have an important effect on risk of fracture.[475]

The Hillier et al.[476] epidemiological study examining fluoride in drinking water found that patients exposed to fluoridated water had increased amounts of fluoride in the cortical bone, supporting not only the importance of water fluoride in interfering with bone metabolism, but fluoridation of water to osteoporosis and periodontal disease. It is notable that the link between fluoride exposure and weakening bone strength was also accepted by the USA Scientific Committee of the National Research Council[477].

Other direct and indirect associations which exist, that increase the risk or probability of developing periodontal disease, include Vitamin C deficiency and diabetes. The relationship between fluoride exposure and both Vitamin C deficiency and diabetes is also well-documented providing further strength of association between fluoride exposure and periodontal disease. The WHO reported that severe Vitamin C deficiency may result in aggravated periodontal disease conditions.[478]

[473] Wactawski-Wende J. Periodontal diseases and osteoporosis: association and mechanisms. Ann Periodontol. 2001 Dec;6(1):197-208.
[474] Whitford GM. The metabolism and toxicity of fluoride. Basel: Karger, 1996: 1–141
[475] Hillier S, Cooper C, Kellingray S, Russell G, Hughes H, Coggon D, Fluoride in drinking water & risk of hip fracture in the UK: a case-control study, Lancet 2000; 355: 265–69.
[476] Hillier S, Cooper C, Kellingray S, Russell G, Hughes H, Coggon D, Fluoride in drinking water & risk of hip fracture in the UK: a case-control study, Lancet 2000; 355: 265–69.
[477] National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C. p. 146.
[478] Petersen E P. Ogawa H., Strengthening the Prevention of Periodontal Disease: The WHO Approach, Global Oral Health Program, World Health Organization, Geneva, Switzerland, J Periodontol • December 2005

In regard to diabetes, it is widely documented that subjects with diabetes mellitus have a higher risk of periodontal disease, and indeed periodontal disease has been considered the sixth complication of diabetes.[479],[480]

Extensive studies have reported significant associations between diabetes and severity of periodontal disease.[481],[482] Taylor et al.[483] concluded from a literature review of severe periodontal disease and diabetes mellitus that not only was there a greater prevalence of periodontal symptoms amongst diabetics but that the progression of periodontal disease was also more aggressive or rapid.

The significance of any association between fluoride, cortical bone mass, periodontal disease, Vitamin C and other critical diseases cannot be underestimated. These findings are of major significance demanding, in the interests of public health and safety, an immediate response from the State requiring the cessation of the water fluoridation policy without delay.

7.10 Diabetes and Fluoride

The effects of fluoride exposure on endocrine organs were examined by the USA National Research Committee (NRC) in their review of water fluoridation. The NRC concluded that "*fluoride exposure appears to bring about increases in blood glucose or impaired glucose tolerance in some individuals and to increase the severity of some types of diabetes*".[484] Impaired glusose intolerance (IGT) is a prediabetic state dysglycemia that is associated with insulin resistance and increased cardiovascolar heart disease. IGT is also a risk factor for mortality.[485]

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[479] Grossi SG, Skrepcinski FB, DeCaro T, Zambon JJ, Cummins D, Genco RJ. Response to periodontal therapy in diabetics and smokers. J Periodontol 1996; 67:1094-1102.
[480] Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: An epidemiological perspective. Ann Periodontol 2001;6:99-112.
[481] Grossi SG, Genco RJ. Periodontal disease and diabetes mellitus: A two-way relationship. Ann Periodontol 1998;3:51-61.
[482] Soskolne WA, Klinger A. The relationship between periodontal diseases and diabetes: An overview. Ann Periodontol 2001;6:91-98.
[483] Taylor GW, Burt BA, Becker MP, et al. Severe periodontitis and risk for poor glycemic control in subjects with non-insulin-dependent diabetes mellitus. J Periodontol 1996;67(Suppl.):1085-1093.
[484] National Research Council of the National Academies, Fluoride In Drinking Water, A Scientific Review of EPA's Standards, Ch. 8. Effects on the Endocrine System, Pages 259-260
[485] National Research Council of the National Academies, Fluoride In Drinking Water, A Scientific Review of EPA's Standards, Ch. 8. Effects on the Endocrine System, Pages 259-260:
[485] Barr EL, Zimmet PZ, Welborn TA, *et al.* (2007). "Risk of cardiovascular and all-cause mortality in individuals with diabetes mellitus, impaired fasting glucose, and impaired glucose tolerance: the Australian Diabetes, Obesity, and Lifestyle Study (AusDiab)". *Circulation* 116 (2): 151–7

The NRC noted that, in general, impaired glucose metabolism appears to be associated with serum or plasma fluoride concentrations of about 0.1 mg/L or greater in both animals and humans.[486],[487],[488],[489] The NRC also highlighted the increased health risk to diabetic individuals who will often have higher than normal water intake, and consequently, will have higher than normal fluoride intake for a given concentration of fluoride in drinking water.

As noted by the UK Expert Group on Vitamins and Minerals[490], several studies have dealt with the effects of fluoride on people with manifest kidney diseases. Fluoride excretion is decreased in patients with kidney failure, and the plasma ionic fluoride concentration is higher than normal.[491],[492],[493] The capacity of the skeleton to store fluoride may provide a sufficient safety margin, but it is also possible that an increased plasma fluoride concentration may result from fluoride liberation from the bone resorption processes involved in certain kidney diseases.

Patients with diabetes insipidus may absorb excess amounts of fluoride because of the large quantities of fluids ingested. Patients with chronic renal failure who are dialysed with fluoridated water receive an additional load of fluoride from the dialysate. In comparison with the average gastrointestinal uptake, the fluoride absorption increases by 20- to 30-fold during a single pass of dialysis.[494]

Undoubtedly this represents an enormous health management risk in Ireland where up to 200,000 patients are diagnosed with diabetes (and a further 200,000 undocumented that may have the condition).[495] It is extremely alarming that Type 2 diabetes mellitus (T2DM) is expected to increase by 37% over the next decade in Ireland.[496]

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[486] Rigalli, A., J.C. Ballina, E. Roveri, and R.C. Puche. 1990. Inhibitory effect of fluoride on the secretion of insulin. Calcif. Tissue Int. 46(5):333-338.

[487] Rigalli, A., R. Alloatti, I. Menoyo, and R.C. Puche. 1995. Comparative study of the effect of sodium fluoride and sodium monofluorophosphate on glucose homeostasis in the rat. Arzneimittel-Forsch. 45(3):289-292.

[488] Trivedi, N., A. Mithal, S.K. Gupta, and M.M. Godbole. 1993. Reversible impairment of glucose tolerance in patients with endemic fluorosis. Fluoride Collaborative Study Group. Diabetologia 36(9):826-828.

[489] de la Sota, M., R. Puche, et al Changes in bone mass and in glucose homeostasis in subjects with high spontaneous fluoride intake. Medicina 57(4):417-420. 1997.

[490] Review Of Fluoride, Expert Group On Vitamins And Minerals, UK Expert Group on Vitamins and Minerals, EVM/01/03/P, May 2001. (Appendix 4)

[491] Juncos LI, Donadio JV (1972) Renal failure and fluorosis. JAMA 222: 783- 785

[492] Berman LB, Taves D (1973) Fluoride excretion in normal and uremic humans. Clin Res 21: 100.

[493] Hanhijaervi H (1974) Comparison of free ionized fluoride concentrations of plasma and renal clearance in patients of artificially fluoridated and nonfluoridated drinking water areas. Proc Finn Dent Soc 70: 21.

[494] Review Of Fluoride, Expert Group On Vitamins And Minerals, UK Expert Group on Vitamins and Minerals, EVM/01/03/P, May 2001

[495] Diabetes Federation of Ireland.

[496] Marar O, Senturk S, Agha A, Thompson C, Diarmuid Smith D. The prevalence of vitamin B12 deficiency in patients with type 2 diabetes mellitus on metformin, *Royal College of Surgeons in Ireland Student Medical Journal 2011;4(1):*16-20

Diabetes is the most common cause of kidney failure accounting for nearly 44% of new cases.[497] The kidney is the organ responsible for excreting most of the fluoride. It is exposed to concentrations of fluoride about five times higher than in other organs. Human kidneys, nevertheless, have to concentrate fluoride as much as 50-fold from plasma to urine. Portions of the renal system may therefore be at higher risk of fluoride toxicity than most soft tissues. One has to consider that subjects who already have impaired kidney function and are unable to excrete fluoride efficiently will retain more fluoride.

Several investigators have shown that patients with impaired renal function, or on haemodialysis, tend to accumulate fluoride much more quickly than normal. It has been found that patients with renal osteodystrophy can have higher fluoride concentrations in their serum.[498] The NRC review raised specific concerns regarding this susceptible sub-population. To this Author's knowledge neither the Health Service Executive nor the Food Safety Authority in Ireland have ever provided any public announcement or health policy statements warning people with diabetes not to consume fluoridated water.

It is generally accepted, as noted by the U.S. NRC[499], that more research is needed on bone concentrations of fluoride in people with altered renal function, as well as other potentially sensitive populations (e.g., the elderly, postmenopausal women and people with altered acid balance), in order to better understand the risks of musculoskeletal effects in these populations.

Alarmingly, it has also been found that fluoride inhibits homocysteine (Hcy) metabolism. It is now known that Homocysteine inhibits DNA methylation and endothelial cell (EC) growth, resulting in a disturbance of the angiogenic balance of the body and reduced formation of new blood vessels essential for fetal development, tissue regeneration and wound healing. Inhibition of angiogenesis reduces blood vessel growth and collateral arteries formation descreasing blood flow and oxygen delivery within the body that can lead to cardiac ischaemia, as well as peripheral artery disease. It has also been found that angiogenesis plays a supporting role in pathological conditions such as diabetic retinopathy, which leads to rapid blindness in persons with diabetics.[500,501]

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[497] U.S. National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), National Institutes of Health (NIH).

[498] National Research Council of the National Academies, Fluoride In Drinking Water, A Scientific Review of EPA's Standards, Ch. 8. Effects on the Endocrine System, Pages 259-260

[499] National Research Council of the National Academies, FLUORIDE IN DRINKING WATER, A Scientific Review of EPA's Standards, Ch. 8. Effects on the Endocrine System, Pages 259-260

[500] Jamaluddin, M,S,. Chen,I,. Yang,F,. Jiang, X., Jan, M., Liu, X., Schafer, A,I,. Durante, W., Yang, X,. Wang, H. Homocysteine inhibits endothelial cell growth via DNAhypomethylation of the*cyclin A* gene, Blood Journal 2007 110: 3648-3655

[501] Joseph F. Murphy and Desmond. J. Fitzgerald, Vascular endothelial cell growth factor (VEGF) induces cyclooxygenase (COX)-dependent proliferation of endothelial cells (EC) via the VEGF-2 receptor, *The FASEB Journal* express article 10.1096/fj.00-0757fje. Published online May 29, 2001.

A further risk factor must also be examined as reported by the U.S. NRC when they reported how it has long been suspected that fluoride, even at concentrations below 1.2 mg/L in drinking water, over the years can increase the risk for renal calculi (kidney stones). [502]

This observation is supported by Singh et al.[503] who carried out extensive medical examinations of more than 18,700 people. It was found that patients with clear signs and symptoms of skeletal fluorosis were 4.6 times more likely to develop kidney stones. The NRC further observed and recommended that if fluoride in drinking water may be considered as a risk factor for kidney stones, future studies should be directed toward examining the risk of kidney stone formation and the corresponding safe level to be achieved in drinking water to protect against this contaminant.

It is scientifically impossible to determine the accurate safe level without undertaking adequate detailed risk assessments yet alarmingly it has been accepted that the toxicological properties of silicafluorides or their derivatives have not been investigated. Furthermore no short-term or sub-chronic exposure, chronic exposure, cytotoxicity, reproductive toxicity, teratology, carcinogenicity, or initiation/promotion studies are available.

The significance of any association between fluoride, diabetes, kidney failure, skeletal fluorosis and other critical diseases cannot be underestimated. These findings are of major significance demanding, in the interests of public health and safety, an immediate response from the State requiring the cessation of the water fluoridation policy without delay.

[502] U.S. National Research Council, Fluoride and Drinking Water: A scientific review of EPA's Standards. 2006.
[503] Singh, P.P., M.K. Barjatiya, S. Dhing, R. Bhatnagar, S. Kothari, and V. Dhar. 2001. Evidence suggesting that high intake of fluoride provokes nephrolithiasis in tribal populations. Urol. Res. 29(4):238-244

7.11 Calcification of Major Arteries and Fluoride

Fluoride is known to be capable of inhibiting a number of important enzymes in the human body, including preglycolytic enzymes, phosphatases, and cholinesterase. Glycolytic enzymes play a central role in dementia[504] as well as other critical processes. In addition, it is reported that inhibition of one or more enzymes controlling cellular glycolysis may result in binding or precipitation of calcium as calcium fluoride,[505] in a mechanism similar to that observed by Li Y et al.[506] It is also known that certain species of fluoride interfere with both contractile power of the heart and the mechanism of beat in a way that cannot be ascribed to hypocalcaemia. [507]

Perhaps one of the most alarming potential consequences of water fluoridation, as highlighted in recent research, is that a significant correlation exists between fluoride uptake and calcification of the major arteries, including coronary arteries.[508]

A similar process was observed by Mertz et al. when they reported how fluoride acted to increase intracellular calcium concentration by releasing Ca2+ from intracellular stores and stimulating extracellular Ca2+ entry.[509]

It is known that plasma fluoride levels increase with age and with increasing fluoride content of bone, and as a consequence of renal insufficiency such as diabetes.[510],[511],[512]

Since pathologists and anatomists first began examining the heart, they realized that a connection existed between deposits of calcium and

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[504] Iwangoff, P, Armbruster,R, Enz,A, Ruge W. M, Glycolytic enzymes from human autoptic brain cortex: Normal aged and demented cases. Mechanisms of Ageing and Development, Volume 14, Issues 1–2, September–October 1980, Pages 203–209

[505]United States National Library of Medicine, Hazardous substances databank, Biomedical effect and Toxicity of Fluorides.

[506] Li Y, Berenji GR, Shaba WF, Tafti B, Yevdayev E, Dadparvar S. Association of vascular fluoride uptake with vascular calcification and coronary artery disease. Nucl Med Commun. 2012 Jan;33(1):14-20.

[507]United States National Library of Medicine, Hazardous substances databank, Biomedical effect and Toxicity of Fluorides.

[508] Li Y, Berenji GR, Shaba WF, Tafti B, Yevdayev E, Dadparvar S. Association of vascular fluoride uptake with vascular calcification and coronary artery disease. Nucl Med Commun. 2012 Jan;33(1):14-20.

[509] Mertz, L. M., Horn, V. J., Baum, B. J., and Amhudkar, I. S. (1990) *Am. J.- Physiol.* 258, C654-C66

[510] Ekstrand J and Whitford GM (1988). Fluoride metabolism. In: Fluoride in Dentistry. Ekstrand J, Fejerskov O, Silverstone LM (eds) Munksgaard, Copenhagen, pp 150-170.

[511] Ekstrand J, Ehrnebo M, Boreus LO (1978). Fluoride bioavailability after intravenous and oral administration: importance of renal clearance and urine flow. Clin Pharmacol Ther 23: 329- 337

[512] Singer L and Ophaug RH (1979). Concentrations of ionic, total and bound fluoride in plasma. Clin Chem 25: 523-525

disease. In a study of autopsy findings in 2,500 patients who died of coronary artery disease, they were found to have 2-5 times as much calcium as those who died of other causes.[513] In June 2000, the American College of Cardiology (ACC) and American Heart Association (AHA) Consensus Panel wrote in the *Journal of the American College of Cardiology*: "*Coronary calcium is part of the development of atherosclerosis; …it occurs exclusively in atherosclerotic arteries and is absent in the normal vessel wall*." Simply put, the presence of calcification in the epicardial coronary arteries indicates that the patient has coronary atherosclerosis.[514]

This latter observation is of great significance because atherosclerotic coronary artery disease is the number one cause of death in the western world. It is obvious that any associated risk that may accelerate the formation of plaque within the walls of the coronary arteries is of major concern to public health.

In a separate study by Oshnishi et al.[515] examining the extent of element accumulation in the arteries ad cardiac valves the average content of calcium was the highest in the site of the abdominal aorta ramifying into the common iliac arteries, and it decreased in the order: internal iliac, coronary, abdominal aorta, common iliac, external iliac, superior mesenteric, inferior mesenteric, thoracic aorta, brachial, radial, common carotid, subclavian, ulnar, axillary, renal and internal thoracic arteries. Regarding elements in the cardiac valves, the average content of calcium was the highest in the aortic valve. It was found that there were extremely significant direct correlations among the contents of calcium, phosphorus and magnesium.

Any association between fluoride and increased calcification and plaque formation in the arteries is of enormous significance demanding, in the interests of public health and safety, an immediate response from the Government of Ireland requiring the cessation of the water fluoridation policy without delay.

[513] Selby J B, Morris P B, Pearlman J D., Coronary Artery Calcification on CT Scanning, MedScape Ref April 2011.

[514] O'Rourke RA, Brundage BH, Froelicher VF. American College of Cardiology/American Heart Association Expert Consensus document on electron-beam computed tomography for the diagnosis and prognosis of coronary artery disease. *Circulation*. Jul 4 2000;102(1):126-40.

[515] Ohnishi, Y,. Tohno, S., Mahakkanukrauh, R,. Tohno, Y,. Vaidhayakarn, P,. Azuma, C,. Satoh, H,. Moriwake, Y,. Chomsung R,. Minami, T. Accumulation of elements in the arteries and cardiac valves of Thai with aging, Biological Trace Element Research, Volume 96, Numbers 1-3, 71-92, DOI: 10.1385/BTER:96:1-3:71

There is now general agreement that the presence of calcification in the epicardial coronary arteries indicates that the patient has coronary atherosclerosis.[516] Coronary artery atherosclerosis is the principal cause of coronary artery disease (CAD), in which atherosclerotic changes are present within the walls of the coronary arteries. CAD is a progressive disease process that generally begins in childhood and manifests clinically in middle to late adulthood. Cardiovascular disease is the single biggest cause of death in Ireland accounting for over 40% of all deaths and 37% of deaths under 65 years in Ireland. Interestingly the available comparable data on ischaemic heart disease[517] incidence and attack rates suggest a decline in most countries.[518] Up to the year 2000 this trend was not observed in Ireland.

It is alarming that the WHO reported that Ireland has the highest mortality rate from ischaemic heart disease for males in the European Union.[519] In 1998, 13,352 people died from vascular diseases in Ireland. In 1950, 11,887 people died from the same group of diseases[520] representing a 12.3 % mortality increase. This is an astonishing statistic given the improvements in medical healthcare including diagnosis and preventative medicine in the same period up to the year 2000.

More recent statistics[521] published by the HSE report a recent decline in deaths from circulatory system disease including Ischaemic heart disease between 2000 and 2009. While this decline now appears to have plateaued, at the same time there has been an astonishing two- to four-fold increase in primary care for cardiovascular conditions (2000-2005) with a two- to three-fold increase in angiography and angioplasty procedures.[522]

The southern region, in particular Cork city and County, continues to have the highest incidence of cardiovascular disease nationally. It is medically plausable that this is directly associated to the quality of drinking water as has been documented by the WHO. It is known that geographic areas with low calcium and magnesium levels in the water supply have increased incidence of cardiovascular disease. It is likely that this would increase further in populations consuming drinking water low in calcium and magnesium due to the increased bio-availability of fluoride and its derivative compounds in the body.

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[516] O'Rourke RA, Brundage BH, Froelicher VF. American College of Cardiology/American Heart Association Expert Consensus document on electron-beam computed tomography for the diagnosis and prognosis of coronary artery disease. *Circulation*. Jul 4 2000;102(1):126-4

[517] Ischaemic heart disease is a disease characterized by reduced blood supply to the heart.

[518] *Giampaoli S. Ischaemic heart disease*, EUPHIX, European Union Public Health Information System

[519] 1996 World Health Statistics Annual, World Health Organisation, Geneva, 1998

[520] 50 Years of Heart Disease in Ireland Mortality, Morbidity and Health Services Implications, Irish Heart Foundation, 2001.

[521] Health trends in Ireland, Key Trends 2010. Department of Health and Children

[522] Ireland Take Heart Progress on the implementaiton of building Healthier Hearts 1999-2005, Health Service Executive.

It is interesting that the one known variable between the 1950's and 1990's is the fluoridation of drinking water supplies, which commenced in the late 1960's and early 1970's and could therefore not have been a contributory factor to coronary disease in the group related to 1950. It is likely that the reduction in fluoride levels in drinking water, enacted in 2007 as a result of recommendations by the Forum on Fluoridation on grounds of reducing occurrence of dental fluorosis, may result in a further drop in mortality levels from cardiovascular disease. However, it is apparent that the incidence of coronary disease amongst under 60s may also be increasing as much higher numbers of people are now being diagnosed with diabetes and other medical complications. Given the association between fluoridation of water supplies and coronary artery disease incidence rates and the correlation between fluoride uptake and calcification in major arteries, this suggests that exposure of the population to fluoride through fluoridation of water supplies may play an important role in cardiovascular disease and strokes in Ireland. The WHO have previously reported[523] how low Ca and Mg levels in drinking water impact on cardiovascular disease as well as many other health conditions.

Research findings have previously suggested that serum concentrations of fluoride influence the rate of calcification. Higher fluoride concentrations have shown greater enhancement of calcification.[524]

It has further been demonstrated that as concentration in municipal water supplies increased with artificial fluoridation, the mean blood fluoride concentration of the inhabitants increased threefold from 0.014 to 0.040 ppm.[525]

This observation was confirmed by further research which demonstrated that plasma fluoride concentration increased significantly with increased fluoride intake.[526]

Simply put, higher fluoride intake contributes to higher blood plasma fluoride levels which contribute to higher calcification in the arteries and higher risk of coronary disease resulting in higher health costs and mortality within the community.

[523] Calcium and Magnesium in Drinking-Water Public Health Significance, World Health Organization, 2009

[524] Donald R. Taves, W.F. Neuman, From the Department of Radiation Biology, University of Rochester School of Medicine and Dentistry, Rochester, New York, USA, Factors controlling calcification *in vitro*: Fluoride and magnesium Archives of Biochemistry and Biophysics, Volume 108, Issue 3, December 1964, Pages 390–397

[525] Smith, Frank A.; Gardner, Dwight E.; Hodge, Harold C. Metabolism of fluoride. II. Fluoride content of blood and urine as a function of the fluoride in drinking water, Journal of Dental Research (1950), 29, 596-600 CODEN: JDREAF; ISSN: 0022-0345.

[526] Singer, Leon; Armstrong, W. D. Regulation of human plasma fluoride concentration, Journal of Applied Physiology (1948-1976) (1960), 15, 508-10 CODEN: JAPYAA; ISSN: 0021-8987

It is imperative that public health policy protects the most vulnerable in our society, as it is supposed to do; consequently it is logical that continuation of any policy which is putting the most vulnerable at risk must end immediately.

It is interesting that a public health recommendation exists for adults to take low-dose aspirin for its anti-coagulant effect on the blood.

Research by Inkielewicz et al.[527] has suggested that one of the main benefits of aspirin is that it counteracts the toxic effects of fluoride on antioxidant parameters, increases the absorption of fluoride in bone and other tissues while reducing the blood fluoride plasma level. Inkielewic et al. also found that that consumption of artificial fluoridated water had various adverse direct effects on heart function.

Similarly Japanese researchers[528] found that children with dental fluorosis have a higher incidence of heart damage than those without fluorosis.

This observation was also found by Chinese researchers[529] who recorded an increase in abnormal heart rhythm in patients with dental fluorosis.

[527] Inkielewicz, Czarnowski, Research report Oxidative stress parameters in rats exposed to fluoride and aspirin, Fluoride 41(1)76–82, January-March 2008
[528] The Lancet, Jan. 28, 1961, p. 197, Tokushima J. Exper., Med. 3-50-53, 1956
[529] Wang et al, "Toxicity From Water Containing Arsenic and Fluoride in Xinjiang" Fluoride Vol. 30 No. 2 81-84 1997

7.12 Calmodulin Activity Sudden Death Syndrome, Neurological Disease and Fluoride.

Calmodulin mediates critical signalling pathways responsible for divergent functions in the heart including calcium cycling, hypertrophy and apoptosis. Dysfunction in the Calmodulum signalling pathway occurs in heart disease and is associated with increased susceptibility to life-threatening arrhythmia.[530] The medical term for an abnormal rhythm of the heart is arrhythmia.

Research on fluoride inhibition by Yoria et al[531] demonstrated that fluoride interfered with calcium and calmodulin activity preventing its activation of phosphodiesterase. Fluoride is already known as an inhibitor of a number of enzymes, including phosphatases, preglycolytic enzymes and cholinesterase.[532] Phosphates metabolism plays a critical role in cellular function, physiology and disease[533], including allergy, asthma, obesity, myocardial hypertrophy and Alzheimer's disease.[534]

Glycolytic enzymes also play a central role in dementia.[535] It is reported that inhibition of one or more enzymes controlling cellular glycolysis may result in binding or precipitation of calcium as calcium fluoride (calcification of the arteries). Furthermore, some species of fluoride are known to interfere with both the contractile power of the heart and the mechanism of beat in a way that cannot be ascribed to hypocalcaemia.[536]

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[530] Christensen MD, Dun W, Boyden PA, Anderson ME, Mohler PJ, et al. (2009) Oxidized Calmodulin Kinase II Regulates Conduction Following Myocardial Infarction: A Computational Analysis. PLoS Comput Biol 5(12): e1000583. doi:10.1371/journal.pcbi.1000583

[531] Yorio T, Sinclair R, Henry S. Fluoride inhibition of the hydro-osmotic response of the toad urinary bladder to antidiuretic hormone. J Pharmacol Exp Ther. 1981 Nov;219(2):459-63.

[532] United States National Library of Medicine, Hazardous substances databank, Biomedical effect and Toxicity of Fluorides.

[533] Hong Zheng, Shawn Alter, and Cheng-Kui Qu, SHP-2 tyrosine phosphatase in human diseases Int J Clin Exp Med. 2009; 2(1): 17–25.

[534] Bottini N, Bottini E, Gloria-Bottini F, Mustelin T. Low-molecular-weight protein tyrosine phosphatase and human disease: in search of biochemical mechanisms. Arch Immunol Ther Exp (Warsz). 2002;50(2):95-104.

[535] Iwangoff, P, Armbruster,R, Enz,A, Ruge W. M, Glycolytic enzymes from human autoptic brain cortex: Normal aged and demented cases. Mechanisms of Ageing and Development, Volume 14, Issues 1–2, September–October 1980, Pages 203–209

[536]United States National Library of Medicine, Hazardous substances databank, Biomedical effect and Toxicity of Fluorides.

Loss of calmodulin activity has been linked to contractile dysfunction[537] which causes congestive heart failure characterized by a high incidence of sudden death from nonreentrant ventricular arrhythmias.[538]

More than 5,000 people suffer sudden cardiac death in Ireland each year, of which 60-80 are under 35 years of age. Sudden cardiac death (SCD) is defined as 'death due to natural causes within an hour of the onset of symptoms, in the absence of any other cause, and assumed or proven to have a cardiac cause'. Post mortem results of SCD in the young have shown a number of underlying conditions, such as cardiomyopathy, myocarditis and coronary heart disease. There are also many unexplained deaths (approximately 20-30%) in people under 35 years of age, where no structural abnormality is found at post mortem and these are classed as sudden arrhythmic death syndrome (SADS).[539]

Review of Irish post mortem data suggests that SCD in a population of 14-35 year olds occurs in three to four per 100,000, with males being seven times more likely to be affected. [540] While perhaps only coincidental it is nevertheless of importance that the indicent ratio for SCD for males is the same as that from Harvard College's School of Dental Medicine[541] which found that pre-adolescent boys who drink fluoridated water are at a seven-fold increased risk of osteosarcoma[542], an often fatal bone cancer.

A growing body of scientific evidence also shows that other major pathways, such as the calcium/calmodulin-dependent kinase and the mitogen-activated kinase cascades, are involved in activity-dependent regulation of gene expression and may also be implicated in mental and neurological health. Regulation of gene expression, whereby neurons and neuronal

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[537] Turla, M, B. Gnegy,. Margaret, E,. Epps, S,. Shlafer, M. Loss of calmodulin activity in cardiac sarcoplasmic reticulum after ischemia. Biochemical and Biophysical Research Communications Volume 130, Issue 2, 31 July 1985, Pages 617–620

[538] Pogwizd SM, Bers DM. Na/Ca exchange in heart failure: contractile dysfunction and arrhythmogenesis. Ann N Y Acad Sci. 2002 Nov;976:454-65.

[539] Task Force for Sudden Cardiac Death, 2004

[540] Morris VB, Keelan T, Leen E et al. Sudden cardiac death in the young: a 1 year post mortem analysis in the Republic of Ireland. Ir J Med Sci 2009; 178(3): 257-261

[541] Bassin EB, Wypij D, Davis RB, Mittleman MA. (2006). Age-specific Fluoride Exposure in Drinking Water and Osteosarcoma (U.S). Cancer Causes and Control 17: 421-8

[542] One hundred and eighty-three osteosarcoma cases were recorded on the island of Ireland between 1994 and 2006

networks adapt their short- and long-term responses to environmental stimuli, represents a major component of neurological disease.[543]

Activation of gene expression was shown to be involved in a large variety of processes in both the developing and mature nervous system, including proliferation of neuronal precursors, outgrowth of neuronal processes, learning and memory in invertebrates and vertebrates, induction of neurotrophic and neuroprotectant cellular programs and regulation of circadian rhythms.[544],[545],[546],[547],[548]

It is obvious that any scientifically documented substance that may increase the associated risk of neurological disease, learning or memory disabilities, sudden death syndrome (SDS) or other cardiovasculor disease should in no circumstance be injected into the public drinking water supplies.

This raises serious concerns for the possible health implications for high-risk individuals in particular for infants who are bottle-fed formula milk constituted with fluoridated water (exposing them to concentrations of fluoride at multiples above the recommended tolerable daily intake) and adults who consume large volumes of water (including active sport enthusiasts) as well as diabetics with impaired renal kidney function (who are already a high-risk sector for coronary disease).

These findings are of critical significance demanding, in the interests of public health and safety, an immediate response from the Government of Ireland requiring the immediate cessation of the water fluoridation policy. In light of the information provided in this report failure to act by the appropriate authorities may be deemed to be criminally and professionally negligent.

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[543] Tardito D, Perez J, Tiraboschi E, Musazzi l, Racagni g, Popoli, M. Signalling Pathways Regulating Gene Expression, Neuroplasticity, and Neurotrophic Mechanisms in the Action of Antidepressants: Critical Overview, *pharmacological Reviews* Vol. 58, No. 1

[544] Kandel ER (2001) The molecular biology of memory storage: a dialogue between genes and synapses. *Science (Wash DC)* 294:1030–1038.

[545] Mabuchi T, Kitagawa K, Kuwabara K, Takasawa K, Ohtsuki T, Xia Z, Storm D, Yanagihara T, Hori M, and Matsumoto M (2001) Phosphorylation of cAMP response element-binding protein in hippocampal neurons as a protective response after exposure to glutamate in vitro and ischemia in vivo. *J Neurosci* 21:9204– 9213

[546] Reppert SM and Weaver DR (2001) Molecular analysis of mammalian circadian rhythms. *Annu Rev Physiol* 63:647–676.

[547] Kida S, Josselyn SA, de Ortiz SP, Kogan JH, Chevere I, Masushige S, and Silva AJ (2002) CREB required for the stability of new and reactivated fear memories. *Nat Neurosci* 5:348–355

[548] Pittinger C, Huang YY, Paletzki RF, Bourtchouladze R, Scanlin H, Vronskaya S, and Kandel ER (2002) Reversible inhibition of CREB/ATF transcription factors in region CA1 of the dorsal hippocampus disrupts hippocampus-dependent spatial memory. *Neuron* 34:447–462.

7.13 Genetic Damage and Fluoride

That fluoride causes genetic damage *in vitro* is generally acknowledged. Sodium fluoride has been found to be mutagenic. Most of the *in vitro cytogenic* studies carried out with sodium fluoride demonstrate that it induces chromosome aberrations and sister chromatid exchanges, mainly chromatid gaps and breaks.[549][550],[551],[552],[553] While it is accepted that the fluoride concentrations used in the *in vitro* studies were many times higher than the level often present in drinking water, alarmingly however a significant increase in Sister Chromatid Exchange (SCE) rate has been reported in fluorosis patients [554],[555].

SCE analysis is a very sensitive measure of chromosome damage induced by some DNA damaging agent.[556] Results from Sajayan et al.[557] support the observation that there is a significant increase in the frequencies of chromosome aberrations and SCE in communities exposed to high levels of fluoride. The lymphocytes of these residents were also more susceptible, to a clastogen such as Mitomycin-C, than the other populations and displayed a significant increase in chromosome aberrations.

SCE was significantly increased in gastric cancer (GA) and chronic atrophic gastritis (CAG) patients. It is suggested that the increased SCE in patients reflects a genomic instability that may be operative in gastric carcinogenesis.[558]

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[549] U.S National Toxicology Program Technical Report Series No. 393. Sodium Fluoride (CAS No. 7681-94-4) in F344/N rats and B6C3F1 mice (drinking water studies).
[550] Gadhia PK, Joseph S. Sodium fluoride induced chromosome aberrations and sister chromatid exchange in cultured human lymphocytes. Fluoride 1997; 30(3):153-6.
[551] Tsutsui T, Suzuki N, Ohmori M. Sodium fluoride induced morphological and neoplastic transformation, chromosome aberrations, sister chromatid exchanges and unscheduled DNA synthesis in cultured Syrian hamster embryo cells. Cancer Res 1984;44,938-41.
[552] Thomson EJ, Kilanowski FM, Perry PE. The effect of fluoride on chromosome aberrations and sister chromatid exchange frequencies in cultured human lymphocytes. Mutat Res 1985;144:89-92.
[553] Sajayan Joseph, PK Gadhiaa Sister Chromatid Exchange Frequency And Chromosome Aberrations In Residents Of Fluoride Endemic Regions Of South Gujarat, Fluoride Vol. 33 No. 4 154-158 2000 Research Report
[554] Sheth FJ, Multani AS, Chinoy NJ. Sister chromatid exchanges: A study in fluorotic individuals of North Gujarat. Fluoride 1994;27(4):215-9.
[555] Evans HJ, O'Riordan ML. Human peripheral blood lymphocytes for the analysis of chromosome aberrations in mutagen tests. Mutat Res 1975; 31:135-48.
[556] Perry P. Evans H J. Cytological dctcction of mutagcncarcinogcn cxposurc bv sister chromatid exchangc. Nature 1975: 258: 121 -5.
[557] Sajayan Joseph, PK Gadhiaa Sister Chromatid Exchange Frequency And Chromosome Aberrations In Residents Of Fluoride Endemic Regions Of South Gujarat, Fluoride Vol. 33 No. 4 154-158 2000 Research Report
[558] Alteration of sister chromatid exchange frequencies in gastric cancer and chronic atrophic gastritis patients with and without H pylori infection by Ali Karaman, Doğan Nasir Binici, Mehmet Eşref Kabalar, Hakan Dursun, Ali Kurt, World Journal of

A person with gastric carcinoma has abnormal cells in the stomach that multiply out of control. These cells can form tumours and spread to other parts of the body. Gastric carcinoma is a serious healthcare concern and the second most frequent malignancy worldwide.[559] Nowadays it is accepted that environmental factors play a significant role in the development of the disease.[560]

The fact that fluoride has been demonstrated to significantly increase the SCE rate in humans and that gastric cancer patients have also been demonstrated to have increased SCE, presents a considerable risk factor for the percentage of the population that are known to be suffering from fluorosis in Ireland.

It can be assumed therefore that individuals with fluorosis may be considered at risk of developing gastric cancers or chronic atrophic gastritis (inflammation of the lining of the stomach). Given the association, the presence of fluoride or silicofluoride compounds in drinking water cannot be excluded as a possible likely cause of such disease.

As alarming is the fact that fluoride has been shown to cause genetic damage at a level of 1ppm, the level which is usually considered as being safe in fluoridation schemes.[561],[562],[563],[564],[565],[566] Bale et al.[567] reported that "*cytological analyses indicated that these treatments produced chromosome abnormalities, including bridges with or without fragments, univalents, fragments and micronuclei. The production of a significantly higher percentage of chromosomal aberrations by simultaneous treatment with a low concentration of sodium fluoride and dimethyl sulfoxide over that produced by treatment with low concentration of sodium fluoride alone indicates that dimethyl sulfoxide can enhance the uptake of the mutagen and thereby increase the mutagenic effect*".

Gastroenterology (2008) Volume: 14, Issue: 16, Publisher: The WJG Press and Baishideng, Pages: 2534-2539

[559] Roukos DH. Relevant prognostic factors in gastric Cancer. Ann Surg 2000; 232:719-20.

[560] Kabat G, Ng S, Wynder E. Tobacco, alcohol intake & diet in relation to adenocarcinoma of the esophagus & gastric cardia. Cancer Causes Control.1993;4: 123.

[561] S. S. Bale, G. E. Hart, Studies On The Cytogenetic And Genetic Effects Of Fluoride On Barley.: Ii. The Effects Of Treatments Of Seedling Coleoptiles With Sodium Fluoride, Canadian Journal of Genetics and Cytology, 1973, 15:(4) 703-712, 10.1139/g73-084

[562] Cancer Research 44:938-941 (1984) and Mutagenesis 1:(2) 157-167 (1986)

[563] W. L. Gabler, et al., " Effect of Fluoride on the Kinetics of Superoxide Generation by Fluoride," Journal of Dental Research, Vol. 64, p. 281 (1985).

[564] A. S. Kozlyuk, et al., " Immune Status of Children in Chemically Contaminated Environments," Zdravookhranenie, Issue 3, pp. 6-9 (1987).

[565] D. J. Newell, "Fluoridation of Water Supplies and Cancer - An Association," Applied Statistics, Vol. 26, No. 2, pp. 125-135 (1977).

[566] Robert N Hoover, Frank W Mckay, Joseph F Fraumeni Jr. Fluoridated Drinking water and the occurance of cancer, Journal of national cancer institute Vol 57 No 4 october 1976, 757-768

[567] S. S. Bale, G. E. Hart, Studies On The Cytogenetic And Genetic Effects Of Fluoride On Barley.: Ii. The Effects Of Treatments Of Seedling Coleoptiles With Sodium Fluoride, Canadian Journal of Genetics and Cytology, 1973, 15:(4) 703-712, 10.1139/g73-084

There is further data that supports the idea that fluorides can induce genetic alterations. Evidence indicating biochemical interactions of fluoride with the genetic mechanisms of cell division is presented in the U.S. NRC report[568] on fluoride in drinking water.

Early research indicating a link between fluoride in drinking water and Down syndrome found that there was a significant relationship between residents in a fluoridated community and an increased prevalence of Down syndrome in younger mothers.[569],[570],[571]

While this research remains disputed the connection between fluoridation and Down syndrome was supported by AW Burgstahler in 1966[572], 1975[573] and in 1997.[574] A review by Takahashi presenting evidence that fluoridation is associated with a higher incidence of Down syndrome was published in 1998.[575]

One of the most authoritative studies with relevant data comes from a study of births of children born in two areas of Atlanta, Georgia, as reported in 1976 by Erickson et al. [576],[577] Two different estimates of the number of children with Down syndrome and normal children were presented. One estimate of Down syndrome births was made by the examination of copies of birth certificates and the other was based on hospital records.

A re-examination of Erickson's data by Burgstahler showed an overall enhancement of Down's Down syndrome births to mothers from the fluoridated area. Later, in 1998, Takahashi did a fine grain analysis of data from a number of sources that included the corrected numbers from the 1966

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[568] National Research Council of the National Academies, FLUORIDE IN DRINKING WATER, A Scientific Review of EPA's Standards, Ch. 8. Effects on the Endocrine System, Pages 259-260

[569] Rapaport I. Contribution a l'etude du mongolism. role pathogenique du fluor. *Bulletin de 1'Academie Nationale Medecine* (Paris) *140* 525-531 1956.

[570] Rapaport I. Nouvelles Recherches sur le mongolisme a propos du role pathogenique du fluor. *Bulletin de 1'Academie Nationale Medecine* (Paris) *143* 367-370 1959.

[571] Rapaport I. Oligophrenie mongolienne et caries dentaire. *Revue de Stomatologie et de Chirurgie Maxillo-Faciale 64* 207-218 1963

[572] Burgstahler, A. W. (1966) Fluoridated water and Down's syndrome. Long abstract of a report of the 21st Conference of the International Society for Brain Research, Budapest.

[573] Burgstahler AW. Fluoride and Down's Syndrome (Mongolism). (Editorial review). Fluoride 1975;8:1-11, 120

[574] Burgstahler AW. Fluoridated water and Down's Syndrome. (Abstract). Fluoride, 1997;30:113.

[575] Takahashi K. Fluoride-linked Down Syndrome births and their estimated occurrence due to water fluoridation. Fluoride 1998;31:61-73.

[576] Erickson JD, Oakley GP, Flynt JW, Hay S. Water fluoridation and congenital malformations: No association. *Journal of the American Dental Association 93* 981-984 1976; *95* 476 1977.

[577] Erickson JD. Down syndrome, water fluoridation, and maternal age. *Teratology 21* 177-180 1980.

Erickson report.[578] In the Takahashi report a clear-cut relationship between fluoride exposure and the number of affected children was found in mothers 30 years of age and younger. While this study has been disputed a recent re-examination of the data by Professor L. Isaacson[579], Binghamton Public Research University, New York and Professor Juan C. Molino[580] found the same age-fluoride- Down syndrome birth effect using only data from hospital records.[581]

It is estimated that there are approximately 7,000 people in Ireland with Down syndrome, with one baby in every 546 births born with the congenital chromosomal anomaly.[582] Many of the associated health conditions with Down syndrome are also linked to potential fluoride toxicity including thyroid, neurological problems and gastro-intestinal problems as well as childhood cancers.

It is important to clarify that the research does not conclude that water fluoridation is the cause itself of Down syndrome. The previous research indicated that exposure to fluoride through water fluoridation appeared to increase the incidence of mothers, thirty years of age or younger, to having a child with Down syndrome.

It may be, though it is unproven, that the increased risk association may exist through the interaction of fluoride in Homocysteine Metabolism. As noted previously in this report, it has been found that Homocysteine metabolism is a risk factor for Down syndrome (DS). Fluoride is known to be an inhibitor of enzymatic activity and research has identified fluoride as an inhibitor of homocysteine hydrolase.[583] Inhibition of homocysteine hydrolase would result in cellular accumulation of homocysteine.[584]

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[578] Takahashi, K. (1998) Fluoride-linked Down syndrome births and their estimated occurrence due to water fluoridation. Fluoride, 31: 61-73.

[579] Robert L. Isaacson, Emeritus, Distinguished Professor of Psychology Ph.D., Binghamton University, New York. Member, National Research Council committee on possible toxic effects of Fluoride in drinking water, 2004-2005. Former Director, Center for Neurobehavioral Sciences, Past President, International Behavioral Neuroscience Society (IBNS), Fellow: American Psychological Society, Fellow: IBNS, Member, American Physiologic Society and Visiting Lecturer for Minority Institution, Member Editorial Boards: Brain Research . Past service on several NIH and NIMH Review Panels and Committees, Chairman and member of several committees for the Society for Neuroscience, Member, NRC Committee for the evaluation of possible hazards of fluoride in drinking water.

[580] Dr. Juan Carlos Molina is the Director of the Ferryra Research Institute at the University of Cordoba, Argentina, as well as holding his distinguished professor position there. He also is a visiting research professor at Binghamton University.

[581] My Views on the Fluoridation of Water Robert L. Isaacson Distinguished Professor of Psychology Binghamton University (See Appendices)

[582] Downs Syndrome Ireland Statistics

[583] Mehdi S, Jarvi ET, Koehl JR, McCarthy JR, Bey P. The mechanism of inhibition of S-adenosyl-L-homocysteine hydrolase by fluorine-containing adenosine analogs. J Enzyme Inhib. 1990;4(1):1-13.

[584] Liu S, Wnuk S F, Yuan C, Robins M J, Borchardt R T, Adenosine-5'-carboxaldehyde: a potent inhibitor of S-adenosyl-L-homocysteine hydrolase, *J. Med. Chem.*, 1993, *36*

It is known that Down syndrome is most common in infants of women thirty five years or older and that the incidence of Down Syndrome increases with increasing maternal age. It has been documented that women in Ireland are having children at later ages, therefore the overall incidence of Down Syndrome is likely to increase.[585] Generally Down syndrome occurs in 1 in 800 births and while Ireland may have a higher than average incidence of Down Syndrome, the significance of the relationship between fluoride exposure and homocysteine metabolism cannot be underestimated. Particularly when fluoride is a persistent toxic substance that poses a specific risk to human health and a toxin which demonstrates a linear accumulation in the human body over time.

It is medically plausible therefore, given that fluoride increases in the body in a linear fashion with age, that older women having higher exposure to fluoride with parallel increases in homocysteine levels, may subsequently have a greater risk of having children with Down syndrome.

It is interesting to note that other independent research has documented that there was a statistically notable increase in the incidence of Down syndrome worldwide for young mothers in the age group 20-24yrs and 25-29yrs between 1984-2002 compared to the period 1962-1983.[586] The general consensus however, from current research, appears to be that the increased incidence of genetic abnormalities in infants appears to be linked to environmental exposures to some as yet unknown contaminant.

In acknowledging this, one must also be aware that fluoride has in the past forty years become one of the most widely available conmtaminants that is present in artificially elevated concentrations in fluoridated drinking water, processed foods and drinks prepared with treated water as well as cooked foods prepared with fluoridated water. As a consequence the daily intake and exposure of the population to fluoride is now considerably higher than that of previous generations.

It is remarkable, therefore, that to this author's knowledge, no clinical studies have been undertaken to establish the likelihood that water fluoridation, fluoride or in particular silicafluoride interactive compounds may be a contributory factor to the increase in the risk of congenital chromosomal anomaly. This is particularly alarming given that a recent study by Machalinski et al.[587] reported that the four different human leukemic cell lines were susceptible to the effects of sodium hexafluorosilicicte. It is worth observing that there is a much higher incidence of kidney, bladder, colorectum, brain and leukaemia cancer in Ireland compared to the average worldwide

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(7), pp 883–887

[585] Hock, E.G. Lindsjo, A Down Syndrome in Live Births by Single Year Maternal Age.

[586] World Wide Incidence of Down Syndrome, Stephen G. Read, Learning Disability Research Unit, University of Huddersfield

[587] Machaliński B, Baskiewicz-Masiuk M, Sadowska B, Machalinska M, Marchlewicz M, Wiszniewska B, et al. The influence of sodium fluoride and sodium hexafluorosilicicte on human leukemic cell lines: preliminary report. Fluoride 2003;36;231-40

statistics based on figures compiled for 182 countries[588].

The importance of such studies cannot be overestimated as it is known that fluoride does cross the placenta from the mother's blood to the developing foetus.[589] According to the *Agency for Toxic Substances and Disease Registry* USA Public Health Service "*it is not known whether fluoride causes birth defects in people or animals. Fluoride does cross the placenta from the mother's blood to the developing fetus. No experiments have studied developmental effects of fluoride using standard testing methods. Some animal studies have found developmental effects of fluoride*".

It is obvious that to continue with any policy that effects the population at large in such an uncontrolled manner as fluoridation of the nation's drinking water supply, and without adequate testing to conclusively demonstrate that there are no health impacts, is completely unacceptable.

The significance of any association between fluoride, gastric carcinoma, peptic ulcers, homocysteine metabolism, genetic abnormalities or other critical diseases cannot be underestimated. These findings are of major significance demanding, in the interests of public health and safety, an immediate response from the State requiring the cessation of the water fluoridation policy without delay.

[588] Estimates of worldwide burden of cancer in 2008: GLOBOCAN 2008, International Journal of Cancer Volume 127, Issue 12, pages 2893–2917, 15 December 2010 and National Cancer register of Ireland.

[589] Toxicological Profile For Fluorides, Hydrogen Fluoride, And Fluorine, Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, April 1993

7.14 Pineal Gland and Fluoride

Fluoride readily accumulates in the human pineal gland[590],[591],[592], by old age the pineal gland has a higher fluoride content than either bone, teeth or enamel.[593]

The main pineal hormone is melatonin. Melatonin has strong antioxidant effects with a particular role in the protection of nuclear and mitochondrial DNA. Preliminary evidence suggests that melatonin may help strengthen the immune system. Melatonin also helps control the timing and release of female reproductive hormones. Animal studies suggest that melatonin may be effective for treating Alzheimer's disease and in the mechanisms of learning and memory. [594,595,596,597]

Pineal fluoride concentrations significantly correlate with pineal calcium. In fact, calcification of the developing enamel organs and the pineal gland occur concurrently. This could affect pineal metabolism in much the same way that high local concentrations of fluoride in the developing enamel organ affect ameloblast function (the structural development of enamel).[598] There is consequently a risk for bottle-fed babies who ingest milk formula made up from fluoridated water that fluoride will accumulate in the child's pineal gland. This is particularly significant given that large amounts of calcification have been demonstrated in the pineals of young children.[599],[600],[601],[602],[603]

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[590] Luke J. (1997). The Effect of Fluoride on the Physiology of the Pineal Gland. Ph.D. Thesis. University of Surrey, Guildford.

[591] Ongkana, Nutcharin; Zhao, Xiao-zhen; Tohno, Setsuko; High Accumulation of Calcium and Phosphorus in the Pineal Bodies with Aging Biological Trace Element Research, Volume 119, Number 2, November 2007 , pp. 120-127(8)

[592] Luke, Jennifer. "Fluoride Deposition in the Aged Human Pineal Gland". Caries Res 2991 (35): 125–28. Retrieved 2009-05-20.

[593] Michotte Y, Lowenthal A, Knaepen L, Collard M, Massart DL: A morphological and chemical study of calcification of the pineal gland. J Neurol 1977;215:209-219.

[594] Hardeland, RüDiger (2005). "Antioxidative Protection by Melatonin: Multiplicity of Mechanisms from Radical Detoxification to Radical Avoidance". *Endocrine* **27** (2): 119–30

[595] Reiter, Russel J.; Acuña-Castroviejo, Dario; Tan, DUN-Xian; Burkhardt, Susanne (2006). "Free Radical-Mediated Molecular Damage". *Annals of the New York Academy of Sciences* 939: 200–15

[596] Pappolla, MA; Sos, M; Omar, RA; Bick, RJ; Hickson-Bick, DL; Reiter, RJ; Efthimiopoulos, S; Robakis, NK (1997). "Melatonin prevents death of neuroblastoma cells exposed to the Alzheimer amyloid peptide". *The Journal of neuroscience : the official journal of the Society for Neuroscience* **17** (5): 1683–90

[597] Larson, John; Jessen, Ruth E.; Uz, Tolga; Arslan, Ahmet D.; Kurtuncu, Murat; Imbesi, Marta; Manev, Hari (2006). "Impaired hippocampal long-term potentiation in melatonin MT2 receptor-deficient mice". *Neuroscience Letters* 393 (1): 23–6

[598] Luke J. (2001). Fluoride deposition in the aged human pineal gland. School of Biological Sciences, University of Surrey, Guildford, UK, Department of Obstetrics and Gynaecology, The Royal London Hospital, Caries Research 35:125-128.

[599] Cooper ERA: The human pineal gland and pineal cysts. J Anat (Lond) 1932;67:28-46.

After finding that the pineal gland is a major target for fluoride accumulation in humans, Luke J (2001) conducted preliminary animal experiments to determine if the accumulated fluoride could impact the functioning of the gland - particularly the gland's regulation of melatonin. Luke found that animals treated with fluoride had lower levels of circulating melatonin, as reflected by reduced levels of melatonin metabolites in the animals' urine. This reduced level of circulating melatonin was accompanied by an earlier onset of puberty in the fluoride-treated female animals. Luke summarized her human and animal findings as follows: *"In conclusion, the human pineal gland contains the highest concentration of fluoride in the body. Fluoride is associated with depressed pineal melatonin synthesis by prepubertal gerbils and an accelerated onset of sexual maturation in the female gerbil. The results strengthen the hypothesis that the pineal has a role in the timing of the onset of puberty. Whether or not fluoride interferes with pineal function in humans requires further investigation."* [604]

This risk was further acknowledged by the U.S. National Research Council in their statement that "recent information on the role of the pineal organ in humans suggests that any agent that affects pineal function could affect human health in a variety of ways, including effects on sexual maturation, calcium metabolism, parathyroid function, postmenopausal osteoporosis, cancer, and psychiatric disease."[605]

The potential consequences of disturbances to functions of the pineal gland and resultant human health impacts from increased absorption of fluoride through dietary intake from water fluoridation cannot be underestimated. The first step in assessing a health risk by a substance to humans is the identification of its harmful effects on animals. A health risk to humans is assessed using results from human epidemiological studies in conjunction with results from animal studies. The Newburgh-Kingston Study (Schlesinger et al., 1956) identified that bone defects, anaemia and earlier female menstruation occur more often in children living in the fluoridated Newburgh than in non-fluoridated Kingston community.[606] Limited animal studies examining how fluoride affects the timing of the onset of sexual maturation suggest that fluoride inhibited pineal melatonin synthesis up until the time of sexual

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[600] Wurtman RJ: The pineal gland; in Endocrine Pathology. Baltimore, Williams & Wilkins, 1968, pp 117-132.

[601] Kerényi NA, Sarkar K: The postnatal transformation of the pineal gland. Acta Morphol Acad Sci Hung 1968; 16:223-236.

[602] Tapp E, Huxley M: The weight and degree of calcification of the pineal gland. J Pathol 197 1; 105:31-39

[603] Doskocil M: Development of concrements in the human pineal body. Folia Morphol (Praha) 1984;32:16-26.

[604] Luke J. (2001). Fluoride deposition in the aged human pineal gland. School of Biological Sciences, University of Surrey, Guildford, UK, Department of Obstetrics and Gynaecology, The Royal London Hospital, Caries Research 35:125-128.

[605] National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C. p221-22

[606] Newburgh-Kingston caries-fluorine study. XIII. Pediatric findings after ten years. J Am Dent Assoc. 1956 Mar;52(3):296-306. Schlesinger ER,Overton DE,Chase HC,Cantwell KT

maturation resulting in an accelerated onset of puberty in females. [607]The U.S *Agency for Toxic Substances and Disease Registry* similarly reported that fluoride was reported to affect negatively some endocrine organs, particularly the thyroid, in animal studies.[608] Camargo reported[609] that with Grandidierella lutosa and lignorum estuarine amphipods female fecundity was shown to be the most sensitive endpoint in a 90 day life-cycle test, with a maximum acceptable toxic concentration (MATC) of 4.15 mg F-/L. It is noticeable that below this value it was observed that fluoride was stimulating female fecundity (stimulating reproductive fertility).[610],[611] Consequently there is scientific evidence to show that fluoride acts as an endocrine disruptor in the aquatic environment as well as on human female reproduction.

Early sexual maturation has both physiological and psychological consequences in humans. For girls, it is also associated with an increased risk of certain cancers in later life.[612],[613] The implication therefore of fluoride exposure for long-term health or the environment cannot be overlooked. It is important to be aware that many of the man-made substances that are listed as known endocrine disruptors by the European Commission's 'Community Strategy for Endocrine Disruptors' (COM (1999)706), are fluoridated compounds. Fluoridated compounds form the basic building block of many insecticides and pesticides. Fluoridated elastomers are used in the plastics and silicon industries.

Fluoride is used because of its high chemical and biological reactivity. It is plausible that increased exposure to fluoride through water fluoridation may result in the creation of fluoride interactive xenobiotic compounds which are metabolised in the body and act as endocrine disruptors. A key therefore to limiting the effect of possible endocrine disruptors in the environment may be to limit the availability of fluoride or fluorsilicic compounds.

sis, secretion, transport, binding, action or elimination of natural hormones in the body responsible for maintaining homeostasis, reproduction, development and/or behaviour.[614],[615] Stated differently, such compounds

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[607] Luke J A, The Effect of Fluoride on the Physiology of the Pineal Gland, PhD dissertation School of Biological Sciences, University of Surrey, 1997

[608] ATSDR (Agency for Toxic Substances and Disease Registry) (2001) Toxicological profile for fluoride. US Department of Health and Human Services, Atlanta, Georgia.

[609] Camargo, J.A. Fluoride toxicity to aquatic organisms: a review. Chemosphere. 2003 Jan;50(3):251-64.

[610] Connell, A.D., Airey, D.D., 1982. The chronic effects of fluoride on the estuarine amphipods Grandidierella lutosa and G. lignorum. Water Res. 16, 1313–1317.

[611] McClurg, T.P., 1984. Effects of fluoride, cadmium and mercury on the estuarine prawn Penaeus indicus. Water SA 10, 40–45.

[612] Reproductive History and Breast Cancer Risk, Factsheet, U.S National Cancer Institute, National Institutes of Health, Department of Health and Human Services.

[613] Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, and Berenson GS, Relation of Age at Menarche to Race, Time Period, and Anthropometric Dimensions: The Bogalusa Heart Study, Pediatrics 2002; 110:e43

[614] Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine, U.S. Agency for Toxic Substances and Disease Registry, Dept Of Health & Human Services, 2003

[615] EPA. 1998c. National environmental methods index. EPA methods 340.1, 340.2, and

may cause toxicities that are mediated through the neuroendocrine axis. As a result, these chemicals may play a role in altering, for example, metabolic, sexual, immune and neurobehavioural function.[616]

Such compounds are also thought to be involved in inducing breast, testicular and prostate cancers, as well as endometriosis.[617],[618],[619]

There is some data to suggest that fluoride does adversely affect some endocrine glands. An increase in serum thyronine levels, in the absence of changes in triiodothyronine and thyroid stimulating hormone levels, was observed in individuals living in areas of India with high fluoride levels in the drinking water.[620]

It is noteworthy that as, far back as 1991, the U.S. Public Health Service recommended that further research be undertaken including the conducting of analytical epidemiological studies to determine the relationship, if any, among fluoride intake, fluoride bone levels, diet and body levels of nutrients such as calcium. It was also recommended that research on bone fractures be carried out as well as studies on the reproductive toxicity of fluoride using various dose levels including the minimally toxic maternal dose. Further studies were also recommended to investigate whether or not fluoride is genotoxic.[621]

It is rather disturbing, given the implications for human health, that similar recommendations were made in the York Review (2001), raised again in the NRC review published in 2006, and once more by the SCHER review in 2010. As yet, to my knowledge, no such research has commenced.

340.3. U.S. Environmental Protection Agency.

[616] Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine, U.S. Agency for Toxic Substances and Disease Registry, Dept Of Health & Human Services, 2003

[617] Berger GS. 1994. Epidemiology of endometriosis. In: Berger GS, ed. Endometriosis: Advanced management and surgical techniques. New York, NY: Springer-Verlag.

[618] Giwercman A, Carlsen E, Keiding N, et al. 1993. Evidence for increasing incidence of abnormalities of the human testis: A review. Environ Health Perspect Suppl 101(2):65-71.

[619] Hoel DG, Davis DL, Miller AB, et al. 1992. Trends in cancer mortality in 15 industrialized countries, 1969-1986. J Natl Cancer Inst 84(5):313-320.

[620] Michael M, Barot VV, Chinoy NJ. 1996. Investigations of soft tissue functions in fluorotic individuals of north Gujarat. Fluoride 29(2):63-71.

[621]Report of the Ad Hoc Subcommittee on Fluoride, Public Health Service Department Of Health And Human Services, February 1991.

7.15 Neurological Disease and Fluoride

As far back as 1993 the U.S. *Agency for Toxic Substances and Disease Registry* reported on the toxicological profile of fluorides and concluded "*(b)ecause fluoride interacts with calcium ions needed for effective neurotransmission, fluoride can affect the nervous system.*"[622]

Recent studies have shown accumulation of fluoride in the hippocampus of the brain causing degeneration of neurons, decreased aerobic metabolism, and altered free radical metabolism in the liver, kidney and heart.[623]

The most recent study by Valdez-Jimenez, et al.[624] published in the Journal Neurologia reported that *"the prolonged ingestion of fluoride may cause significant damage to health and particularly to the nervous system"*. The study examined how fluoride induces changes in the brain's physical structure and biochemistry which affects the neurological and mental development of individuals including cognitive processes, such as learning and memory. The study examined how fluoride can accumulate in the body, and how it has been shown that continuous exposure to fluoride causes damaging effects on body tissues, particularly the nervous system. The study found that fluoride can be toxic by ingesting at one part per million (ppm). It further observed that the effects at this concentration are not immediate and that it can take 20 years or more for its toxic effect to become evident. This study observed that chronic exposure to, and ingestion of, the synthetic fluoride chemicals added to water supplies can cause serious brain and neurological damage.

This in itself should be a major cause for concern for the Irish public, the Government of Ireland and its Health Service Executive, especially as it is estimated that over 725,000 people in the Republic of Ireland suffer from neurological conditions.[625] It is noteworthy that while neurological disorders constitute 6.3% of the global burden of disease[626], in Ireland the figure is 17.9% representing over twice the global average burden.[627]

Alarmingly, the HSE reported that there are over 43,000 newly diagnosed cases each year and it is estimated that the number of people in Ireland developing neurological conditions is set to increase dramatically to over

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[622] Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine, U.S. Agency for Toxic Substances and Disease Registry, Dept Of Health & Human Services, 1993, page 125.

[623] Cicek E, Ayden G, Akdogen M, Okutan H. Effects of chronic ingestion of sodium fluoride on mycardium in the second generation of rats. Human Exp. Toxicology, 24(2005)79

[624] Valdez-Jiménez L, Soria Fregozo C, Miranda Beltrán ML, Gutiérrez Coronado O, Pérez Vega MI. Neurologia 2011 Jun;26(5):297-300. Epub 2011 Jan 20.Effects of the fluoride on the central nervous system,

[625] Neurological care in Ireland, Medical Independent, March 2010

[626] Neurological Disorders Public Health Challenges, World Health Organization 2006

[627] Health Service Executive, 2007. Strategic Review of Neurology and Neurophysiology Services in Ireland. Ireland: Unpublished report

869,143 by 2021 as our population ages. Neurological disease has other consequences, as currently 62,000 people care for persons with neurological conditions at home, placing a significant burden on society as a whole.

Given the disturbing findings regarding the potential of fluoride to be a neurotoxin, it is not beyond consideration that Ireland (regarded as the most fluoridated populations in the world with over 70% of the population drinking fluoridated water) may therefore show an association with increased neurological disorders, as is currently evident in population burdens.

In examining the possible risks associated with the effect of fluoride on Alzheimer's or dementia the NRC stated *"it is apparent that fluorides have the ability to interfere with the functions of the brain….fluorides also increase the production of free radicals in the brain through several different biological pathways. These changes have a bearing on the possibility that fluorides act to increase the risk of developing Alzheimer's disease."*[628]

Alzheimer's or dementia affects almost 44,000 people in Ireland[629] and therefore any role of fluoride exposure no matter how small the risk in the development of Alzheimer's or dementia is potentially significant and must be minimised.

Fluoride is known to be capable of inhibiting a number of critical enzymes, including preglycolytic enzymes, phosphatases, and cholinesterase.[630] Glycolytic enzymes play a central role in dementia.[631] Cholinesterase plays a significant role in both Alzheimer's and cardiovascular disease. The brain and heart are rich in cholinesterases and their inhibition may adversely affect cardiac and neurological function. Cholinesterase inhibitors (ChEI) are known to raise blood pressure and slow the pulse rate through both central and peripheral mechanisms; they also reduce cardiac beat-by-beat fluctuations.[632]

According to Professor C Vyvyan Howard, fluoride is a developmental toxin, a neurotoxin which may affect the development of a child. Dr. Howard has stated "*that on a precautionary basis we should not continue with fluoridation of drinking water supplies*". Dr. Howard further stated that "*if governments don't have ways of making people who are susceptible to risks, such as those with marginal kidney function or bottle-fed babies safe from exposure to unnecessary toxins and ensure that sensitive sub-groups of the*

[628] National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C. p 187.
[629] The Alzheimer Society of Ireland
[630] United States National Library of Medicine, Hazardous substances databank, Biomedical effect and Toxicity of Fluorides.
[631] Iwangoff, P, Armbruster,R, Enz,A, Ruge W. M, Glycolytic enzymes from human autoptic brain cortex: Normal aged and demented cases. Mechanisms of Ageing and Development, Volume 14, Issues 1–2, September–October 1980, Pages 203–209
[632] Darren M. Malone, James Lindesay,Cholinesterase inhibitors and cardiovascular disease: a survey of old age psychiatrists' practice , *Age Ageing (2007) 36 (3): 331-333.*

population are protected then they have no right to use a mass medication like fluoridation of public water supplies." [633]

In support of this opinion, it has been reported in the Lancet (one of the world's leading general medical journals and speciality journals in Oncology, Neurology and Infectious Diseases) that *"(f)luoridated water may be having its most devastating effects on the most vulnerable, those in utero and infants less than one year old, whose brains are most sensitive to developmental neurotoxins such as fluoride."*[634] This is further supported by Dr. Albert W Burgstahler, Professor Emeritus of Chemistry, University of Kansas who stated that *"(a)lthough dental public health officials in countries promoting water fluoridation adamantly deny the existence of illness caused by fluoride in drinking water, undeniable medical ill effects from fluoride added to drinking water have been known and reported since the start of water fluoridation over 50 years ago".*[635]

A previous study by Mullenix, et al. reported in *Neurotoxicology and Teratology*, 1995, documented abnormal behavioural responses by animals exposed to fluoride at various stages of gestation, which resulted in the exposed animals exhibiting either permanent hyperactivity if exposed prenatally, or what layman refer to as "the rat version of couch potato" if exposed after birth[636]. It was further observed by Dr. Mullenix that *"still unexplored, however, is the possibility that fluoride exposure is linked with subtle brain dysfunction. This is the first study to demonstrate that central nervous system output is vulnerable to fluoride, that the effects on behaviour depend on the age at exposure and that fluoride accumulates in brain tissues. Of course behaviours per se do not extrapolate, but a generic behavioural pattern disruption as found in this rat study can be indicative of potential for motor dysfunction, IQ deficits and/or learning disabilities in humans. Substances that accumulate in brain tissue potentiate concerns about neurotoxic risk."*[637] Mullenix et al. recorded behavioural changes in rats after ingestion of fluoride and found that the severity of the effect on behaviour increased directly with plasma fluoride levels and fluoride concentration in specific brain regions.

In the introduction to their study, after referring to the increase in dental fluorosis in humans following decades of water fluoridation, the authors

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[633] Dr. Vyvyan Howard Professor of Bio-imaging, University of Ulster, is a medically-qualified toxico-pathologist and the current leader of the Nano Systems Research Group. He has held the Presidencies of the Royal Microscopical Society and the International Society for Stereology and was the General Editor of the Journal of Microscopy from 1985-91. From 2007 to 2009 he was the President of the International Society of Doctors for the Environment, an organisation representing some 30,000 medical doctors around the world which has WHO and UN recognition. He served on the DEFRA Advisory Committee on Pesticides from 2002-2008 as a toxicologist.

[634] Grandjean P, Landrigan PJ. Developmental neurotoxicity of industrial chemicals. Lancet 2006;368:2167-78.

[635] Fluoride Fatigue, Foreward by professor Albert W Burgstahler PhD

[637] Mullenix PJ, Denbesten PK, Schunior A, Kernan WJ. Neurotoxicity of sodium fluoride in rats. *Neurotoxicology and Teratology* 17 (2) 169-177 1995.

commented *"(o)ne concern that has not been fully investigated is the link between fluoride and effects on the central nervous system (CNS).... Many years of ubiquitous fluoride exposure have not resulted in obvious CNS problems such as seizures, lethargy, salivation, tremors, paralysis, or sensory deficits. Still unexplored, however, is the possibility that fluoride exposure is linked with subtle brain dysfunction."*

Of concern also are the findings of one of the most recent neurotoxicity studies undertaken by Rocha-Amador et al. (2007)[638] and noted in the SCHER review, which reported that the findings of this study established *"an inverse association between fluoride in drinking water and IQ after adjusting for relevant confounding variables".*[639] This language may be confusing for many and it is uncertain why SCHER did not use the unambiguous and clear text as written in the conclusion of the original study which stated that *"intake of fluoride in drinking water may contribute to the decreased intelligence in children".*[640]

This finding is supported by research by Tang et al[641] who observed that "*children who live in a fluorosis area have five times higher odds of developing low IQ than those who live in a nonfluorosis area or a slight fluorosis area"* and by Li XS[642] "*(t)he development of intelligence appeared to be adversely affected by fluoride in the areas with a medium or severe prevalence of fluorosis. A high fluoride intake was associated with a lower intelligence."* The authors of this study further concluded that: *"the risk is particularly acute for children, whose brains are particularly sensitive to environmental toxins. Furthermore, it would be advisable to re-examine the benefits of Fluoride given the documented health risks."*[643]

All of this research supports the observation that fluoride in drinking water has a potential neurotoxic effect in children. This is extremely concerning as circulating blood plasma fluoride passes the placenta and reaches the foetus.

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[638] Rocha-Amador D, Navarro ME, Carrizales L, Morales R, Calderón J, Facultad de Medicina, Universidad Autónoma de San Luis Potosí, San Luis Potosí, México. Decreased intelligence in children and exposure to fluoride and arsenic in drinking water; Cad. Saúde Pública, Rio de Janeiro, 23 Sup 4:S579-S587 (2007).

[639] Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water; Scientific Committee on Health and Environmental Risks, Director General for Health & Consumers (2010)

[640] Rocha-Amador D, Navarro ME, Carrizales L, Morales R, Calderón J, Facultad de Medicina, Universidad Autónoma de San Luis Potosí, San Luis Potosí, México. Decreased intelligence in children and exposure to fluoride and arsenic in drinking water; Cad. Saúde Pública, Rio de Janeiro, 23 Sup 4:S579-S587 (2007).

[641] Tang QQ, DuJ, Ma HH, Jiang SJ, Zhou XJ, Fluoride and children's intelligence: a meta-analysis, Biol Trace Elem Res. 2008 Winter: 126(1-3):115-20

[642] Li XS. (1995). Effect of Fluoride Exposure on Intelligence in Children. Fluoride 28:189-192

[643] Rocha-Amador D, Navarro ME, Carrizales L, Morales R, Calderón J, Facultad de Medicina, Universidad Autónoma de San Luis Potosí, San Luis Potosí, México. Decreased intelligence in children and exposure to fluoride and arsenic in drinking water; Cad. Saúde Pública, Rio de Janeiro, 23 Sup 4:S579-S587 (2007).

The level of fluoride in cord blood is about 75% of the level in maternal blood. The fluoride concentration in the placenta can be higher than in maternal blood. Research has demonstrated that exposure to 1.5mg fluoride during pregnancy was seen to markedly increase placental fluoride levels and to a lesser extent foetal blood levels.[644],[645] This research finding would further support the results of a series of studies undertaken in China by Wang et al.[646] examining the developmental effects of fluoride. Despite this latter research reported to have several shortcomings, it did reach the same conclusion as Rocha-Amador et al.[647] and others[648],[649] indicating that fluoride negatively impacts intelligence. Alarmingly the observed reductions between fluoride exposed and non-exposed children ranging from 8 to 11 points in IQ scale.

A further significant neurological fact is the recent research finding demonstrating that fluoride readily accumulates in the pineal gland.[650] Remarkably and worryingly it has now been found that after a lifetime exposure the pineal gland has a higher fluoride content than either bone, teeth or enamel.[651] The interactions of fluoride with homocysteine metabolism, as already examined in this report, and its significance as a contributory risk factor in neurological disease cannot also be overlooked. Homocysteine metabolism is associated with neurological diseases including neural tube defect,[652], [653], [654] schizophrenia,[655] bipolar disorder,[656]depression,[657]

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[644] Caldera R, Chavinie J, Fermanian J, Tortrat D, Laurent A (1988). Maternal-fetal transfer of fluoride in pregnant women. Biol Neonate 54: 263-269

[645] Shen YW and Taves DR (1974). Fluoride concentrations in the human placenta and maternal and cord blood. Am J Obstet Gynecol 119: 205-207

[646] Wang S-X Wang SX, Wang ZH, Cheng XT, Li J, Sang ZP, Zhang XD, Han LL, Qiao XY, Wu ZM, Wang ZQ (2007) Arsenic and Fluoride Exposure in Drinking Water: Children's IQ and Growth. Environ Health Perspect. 115: 643-7

[647] Rocha-Amador D, Navarro ME, Carrizales L, Morales R, Calderón J, Facultad de Medicina, Universidad Autónoma de San Luis Potosí, San Luis Potosí, México. Decreased intelligence in children and exposure to fluoride and arsenic in drinking water; Cad. Saúde Pública, Rio de Janeiro, 23 Sup 4:S579-S587 (2007).

[648] Tang QQ, DuJ, Ma HH, Jiang SJ, Zhou XJ, Fluoride and children's intelligence: a meta-analysis, Biol Trace Elem Res. 2008 Winter: 126(1-3):115-20

[649] Li XS. (1995). Effect of Fluoride Exposure on Intelligence in Children. Fluoride 28:189-192

[650] Luke J. (1997). The Effect of Fluoride on the Physiology of the Pineal Gland. Ph.D. Thesis. University of Surrey, Guildford.

[651] Michotte Y, Lowenthal A, Knaepen L, Collard M, Massart DL: A morphological and chemical study of calcification of the pineal gland. J Neurol 1977;215:209-219.

[652] Steegers-Theunissen RP, Boers GH, Trijbels fj. Finkelstein jd, Blom hj, Thomas CM, et al. Maternal hyperhomocysyeinemia: a risk factor for neural-tube defects? Metabolism 1994; 43: 1475-1480

[653] Ratan SK, Rattan KN, Pandey RM, Singhal S, Kharab S, Bala M, Singh V, Jhanwar A, Evaluation of the levels of folate, vitamin B12, homocysteine and fluoride in the parents and the affected neonates with neural tube defect and their matched controls. Pediatr Surg Int. 2008 Jul;24(7):803-8. Epub 2008 May 8.

[654] Brustolin S, Guigliana R, Felix T.T, Genetics of homocysteine metabolism & associated disorders. Brazialian Journal of Medical & Biological Research(2010) 43: 1-7

[655] Panagiotakos DB, Pitsavos C, Chrysohoou C, Tsetsekou E, Papageorgiou C, Christodoulou G, Stefanadis C. Increased plasma homocysteine concentrations in

Parkinson's disease,[658],[659] and epilepsy.[660] It has been demonstrated that fluoride is an inhibitor of homocysteine metabolism. Inhibition of a step in the initiation of polypeptide chains should result in the accumulation of one or more intermediates which are formed prior to the inhibited reaction.

According to Dr. Diaz-Arrastia[661] in *Neurological Review* practical knowledge concerning some details of homocysteine metabolism, the diagnosis of hyperhomocysteinaemia, and the use of polyVitamin therapy to lower homocysteine levels will be increasingly important in the treatment of patients with neurologic disease.

As noted elsewhere in this report, the most inexpensive and practical manner to inhibit homocysteine metabolism is to reduce fluoride exposure. This can only be achieved by ending the policy of fluoridating drinking water immediately.

It would appear that the emerging consensus from recent studies is not only the long gestation period for dementia[662],[663] and fluoride, but the implicit assumption that fluoride may have a profound influence on adverse health, cognitive aging and neurological disorders including dementia.[664],[665],[666]

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healthy people with hostile behavior: the ATTICA study. Med Sci Monit. 2004 Aug;10(8):CR457-62. Epub 2004 Jul 23.

[656] Levine J, Sela BA, Osher Y, Belmaker RH. High homocysteine serum levels in young male schizophrenia and bipolar patients and in an animal model. Prog Neuropsychopharmacol Biol Psychiatry. 2005 Sep;29(7):1181-91.

[657] Sheng C C, Chuan M C, Chun Y Y, Han Y Y, Lian L C, Fang Y C, Kuan L C, Cheng L Y, Plasma Homocysteine Levels and Major Depressive Disorders in Alzheimer Disease, American Journal of Geriatric Psychiatry: November 2010 - Volume 18 - Issue 11 - pp 1045-1048.

[658] Kuhn W, Roebroek R, Blom H, et al. Elevated plasma levels of homocysteine in Parkinson's disease. Eur Neurol. 1998;40:225-227.

[659] Muller T, Werne B, Fowler B, Kuhn W. Nigral endothelial dysfunction, homocysteine, and Parkinson's disease. Lancet. 1999;354:126-127.

[660] Schwarz S, Zhou G-Z. N-methyl-D-aspartate receptors and CNS symptoms of homocystinuria. Lancet. 1991;337:1226-1227.

[661] Ramon Diaz-Arrastia, MD, PhD, Homocysteine and Neurologic Disease, ARCH NEUROL/VOL 57, OCT 2000

[662] Alzheimer's Disease International. World Alzheimer report. Alzheimer's Disease International, 2009

[663] Launer LJ. The epidemiologic study of dementia: a life-long quest? Neurobiol Aging 2005;26:335-40.

[664] Rocha-Amador D, Navarro ME, Carrizales L, Morales R, Calderón J, Facultad de Medicina, Universidad Autónoma de San Luis Potosí, San Luis Potosí, México. Decreased intelligence in children and exposure to fluoride and arsenic in drinking water; Cad. Saúde Pública, Rio de Janeiro, 23 Sup 4:S579-S587 (2007

[665] Wang S-X Wang SX, Wang ZH, Cheng XT, Li J, Sang ZP, Zhang XD, Han LL, Qiao XY, Wu ZM, Wang ZQ (2007) Arsenic and Fluoride Exposure in Drinking Water: Children's IQ and Growth in Shanyin County, Shanxi Province, China. Environ Health Perspect. 115: 643-7

[666] USA National Research Council, Fluoride in Drinking Water: A Scientific Review of EPA's Standards, Committee on Fluoride in Drinking Water, (2006

A recent study by Picciotto et al.[667] identified another potentially significant risk that, while not in any way conclusive, does raise concerns regarding the environmental exposure of the population to toxins such as fluoride, especially given the information already provided in this report. Their 2009 study published in the Journal *Epidemiology* has suggested that rapid increases in autism rates in California cannot be explained by younger ages at diagnosis, differential migration, changes in diagnostic criteria and inclusion of milder cases but is instead most likely down to environmental exposures.[668]

According to 2007 statistics from the Centers for Disease Control and Prevention in the USA, 1 in every 150 births develops autism. Its growth rate, at 10 to 17% per year, is fastest among all developmental disabilities. A London School of Economics study estimated that the condition costs $90 billion each year, and the Autism Society of America estimated that this will rise to $200 and $400 billion in 10 years (this was calculated in February 2003). There are no accurate documented figures for Ireland, however, the HSE has acknowlewdged the challenges experienced by the HSE and funded non-statutory service providers in meeting the needs of huge increases in numbers of children presenting with autism and the need to balance the requirement to provide assessments and diagnosis with ongoing service intervention to children.

There is a consensus that environmental toxins may be partly responsible for the increase in autism. In Ireland, the numbers of autistic children have increased dramatically over the past 15 years. Autistic children have problems communicating and interacting socially; the symptoms usually are evident by the time the child is a toddler. It is documented that 70% of people with autism are male. It is also notable that the Harvard study identified that the impact of fluoride was observed to effect young boys more than young girls. It is not inconceivable therefore, in the absence of proper scientific assessment, to consider that fluoride may be a contributory factor. Finally, one cannot discuss neurological diseases without mentioning

[667] Irva Hertz-Picciotto, PhD: Dr. Hertz-Picciotto is the Director of the EARLI Network Sacramento Valley Field Site and is a Professor in the Department of Public Health Sciences at the University of California, Davis, School of Medicine. She has published widely on environmental exposures such as lead, arsenic, pesticides, PCBs, PBDEs, and air pollution and their effects on pregnancy and early childhood respiratory health and neurodevelopment, including autism and cognitive deficits. She is also an expert on theory and methods for epidemiologic research. Dr. Hertz-Picciotto directs the CHARGE – *Childhood Autism Risks from Genetics and the Environment* – Study, the MARBLES – *Markers of Autism Risk in Babies-Learning Early Signs* – Study and has conducted large international cohort studies on pregnancy outcomes, childhood morbidity, immune markers and neurodevelopment in polluted areas of Mexico, Chile, the Czech Republic and Slovakia. Dr. Hertz-Picciotto has served on scientific advisory panels at state, national and international level. She is currently Chief of the Division of Environmental and Occupational Health, Deputy Director of the University of California Davis Centre for Children's Environmental Health and Director of the Northern California Collaborative for the National Children's Study. She has taught epidemiology on four continents.

[668] *Irva Hertz-Picciotto*a,b *and Lora Delwiche*a, The Rise in Autism and the Role of Age at Diagnosis *Epidemiology,* Volume 20, Number 1, January 2009

epilepsy. Epilepsy is the most common serious brain disorder world wide. It is the second most commonly seen neurological condition in primary care, and the most commonly seen among neurologists.[669] The prevalence of epilepsy is estimated by calculating the number of people with epilepsy in the population at any one time, divided by the number of people in the same population at the same time. Prevalence is typically expressed as the number of cases per 1,000 people in the population.

Prevalence (per 1000) of active epilepsy in Europe[670]

Author, Country	**Prevalence**	**Age**
Zielinski, Poland	7.8	All ages
Granieri, Italy	6.2	All ages
Maremmani et al. Italy	5.1	All ages
Beghi et al. Italy	3.9	All ages
Giuliani et al, Italy	5.2	All ages
Joensen, Faroes, Denmark	7.6	All ages
Rocca et al, Italy	3.3	All ages
Loafsson and Hauser, Iceland	4.8	All ages
Linehan, Ireland	**8.3-9.0**	**All ages**
Keranen et al. Finland	6.3	Adults
Forsgren, Sweden	5.5	Adults
Óun et AL. Estonia	5.3	Adults over 55
De la Court et al. Netherlands	7.7	Children > 10
Luengo et al, Spain	4.1	Children 0-19yrs
Brorson, Sweden	3.5	Children 0-16yrs
Sidenvall et al. Sweden	4.2	Children 6-12yrs
Waaler et al. Sweden	5.1	Children 0-15yrs
Sillanpaa, Finland	3.2	Children 0-15yrs
Eriksonn and Koivikko, Finland	3.9	Children 0-15yrs
Endziniene, Lithuania	4.3	Children 0-15yrs
Beilmann et al, Estonia	3.6	Children 0-19yrs
Cavazzutti, Italy	4.5	Children 5-14yrs
Sangrador and Luaces, Spain	3.7	Children 6-14yrs
Tidman et al, England	4.3	Children 4-10yrs
Magalov, Azerbaijan, 5	5.9	All ages
Guekht, Russian Federation,	3.4	Over 14yrs

From the information reviewed to date it is not surprising to find that Ireland has among the highest incidence of epilepsy in the world with a prevalence of 8.3-9.0 per 1000 people. In some instances the prevalance of epilepsy is over **twice** that documented in many other European countries (see above).

Neurological conditions have significant implications for our society, for the individuals and families affected by these conditions, for health systems

[669] Linehan, C,. Walsh, P A,. The prevlance of Epilepsy in Ireland, Brainwave The Irish Epilepsy Association. 2009

[670] L Forsgren, E Beghi, A Oun, M Sillanpää. The epidemiology of epilepsy in Europe - a systematic review. European Journal of Neurology Volume 12. Issue: 4, Pages 245-253

providing care for them and for the wider economy. The Cost of Disorders of the Brain in Europe study[671] estimated that the cost of neurological disorders across 28 European countries (excluding dementia) is €84 billion, with dementia costing €55 billion. This represents a total of €139 billion. At present, up to 80% of all admissions to public hospitals in Ireland occur through A&E departments. Up to 20% of these are neurological many representing exacerbations of underlying chronic neurological conditions.[672]

Given the prevalence of neurological disease in Ireland, which is amongst the highest if not the highest in the world representing an astonishing 17% of the total population, the cost of this disease to both society and the Exchequer is truly staggering. It is expected that in parallel with some geographic locations in Ireland demonstrating higher incidences of certain cancers, in particular Southern Counties such as Cork and South Kerry, that a similar increased incidence of neurological disease may be prevalent within these geographic locations. The scientific evidence is clear as reported[673] by the WHO that the incidence of these diseases is directly associated with the quality of drinking water and, in particular, levels of calcium and magnesium. It is clear that water fluoridation reduces the bio-availability of calclium and magnesium thereby further exposing the population to unnecessary health risks that will result in further increased incidences of certain cancers and neurological diseases. It is also clear, as reported elsewhere in this report, that fluoride acts as a metabolic inhibitor of ATP, with resultant increased risk of developing tumours and cancers as well as its importance in endothelial dysfunction in developing coronary artery disease, diabetes mellitus and other critical neurological complications like hypertension.

The U.S. ATSDR has documented the effects of fluoride on human health which include *musculoskeletal*, reproductive (testicular), neurological and renal disorders.[674] Alarmingly, the ATSDR further observed "*that no data is available on the effects of intermediate-duration exposure to fluoride in humans while intermediate-duration exposure of animals to fluoride has resulted in effects on a number of organ systems, including bone, testes, kidney, neurobehavioral effects, and developmental effects.*"

According to the WHO Report, Neurological Disorders and Public Health Challenges (2006), "there is ample evidence that pinpoints neurological disorders as one of the greatest threats to public health." The scientific consensus appears to suggest now that ample evidence exists that clearly shows a convergence of evidence from neurobiology, cytotoxology and cancer epidemiology demonstrating the association between water fluoridation and ill health. Consequently water fluoridation itself, may prove to be one of the greatest threats to public health.

[671] Andlin-Sobocki P; Jönsson B;, Wittchen H U; Olesen J, 2005. 'Cost of Disorders of the Brain in Europe'. European Journal of Neurology. 12 (suppl. 1) 1-27.
[672] The Future for Neurological Conditions in Ireland: Neurological Alliance of Ireland. 2010.
[673] Calcium and Magnesium in Drinking-Water Public Health Significance, World Health Organization, 2009.
[674] Interactive profile for Fluoride, U.S. Department of Health and Human Services Public Health Service Agency for Toxic Substances and Disease Registry, May 2004

7.16 Osteoporosis and Fluoride

Fluoride has profound effects on the skeleton.[675] Empirical data suggest substantial variations in bone fluoride concentrations at any given water concentration.[676] While fluoride tends to increase the density of trabecular bone (cancellous bone), it tends to decrease the density of cortical bone. Reduced cortical bone density is a particularly important risk factor for hip fracture, as one of the primary sites of hip fracture (the femoral neck) gains up to 95% of its strength from the integrity of cortical bone.[677] Reduced cortical bone density is also important for most forms of "non-vertebral" fractures, particularly wrist fracture.[678]

Osteoporosis is a common multifactorial disorder of reduced bone mass manifesting clinically as fragility fracture. Fracture arises from minor trauma acting on a skeleton that has reduced bone strength.[679] Osteoporosis is characterized by a loss of bone mineral density (BMD), and often culminates in a fracture of the hip, wrist, and/or vertebrae. It is further acknowledged that the most susceptible group of the population to bone fractures is the elderly. Research has also demonstrated that the concentration of fluoride in the bones increased in an essentially linear fashion with an increase of fluoride in the drinking water.[680] The Department of Health in Ireland recently published figures stating that the number of people over the age of 65 is growing by 20,000 every year, with 25% of the people living in Ireland now over 65 years of age.[681] This means that the sensitive subgroup where total fluoride intake should be controlled, to prevent bone fractures, is expanding rapidly as life expectancy increases.

The diagnosis of osteoporosis is often made by using bone density measurements.[682] Bone mass declines and the risk of fractures increases as people age, especially as women pass through the menopause. Hip fractures, the most serious outcome of osteoporosis, are becoming more

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[675] Michael Kleerekoper, M. D. Center for Osteoporosis Research, Fluoride and the Skeleton, Critical Reviews in Clinical Laboratory Sciences, 33(2):139-161 (1996)

[676] U.S. National Research Council, Fluoride and Drinking Water: A scientific review of EPA's Standards. 2006.

[677] Gordon SL, Corbin SB. (1992). Summary of workshop on drinking water fluoride influence on hip fracture on bone health. (National Institutes of Health, 10 April, 1991). Osteoporosis International 2:109-17.

[678] Riggs BL. (1983). Treatment of osteoporosis with sodium fluoride: an appraisal. Bone and Mineral Research. 2: 366-393.

[679] Peacock M, Turner C H, Econs M J, Foroud T, Genetics of Osteoporosis, Endocrine Reviews June 1, 2002 vol. 23 no. 3 303-326

[680] Zipkin, I.; McClure, F. J.; Leone, N. C.; Lee, W. A. Fluoride deposition in human bones after prolonged ingestion of fluoride in drinking water, Public Health Reports (1958), 73, 732-40 CODEN: PHRPA6; ISSN: 0033-3549

[681] Department of Health December 2011

[682] Mattson JS, Cerutis DR, Parrish LC. Osteoporosis: a review and its dental implications. Department of Periodontics, Creighton University School of Dentistry, Omaha, Nebraska, USA, Compend Contin Educ Dent. 2002 Nov;23(11):1001-4, 1006, 1008 passim; quiz 1014.

frequent than before because the world's population is ageing and because the frequency of hip fractures is increasing by 1-3% per year in many areas of the world. Many deaths in elderly people are due to complications arising from bone fractures that occur as a result of osteoporosis.[683]

Remarkably, studies have found that with therapeutic oral administration of fluoride in amounts as low as 0.6 mg/kg body weight/day in postmenopausal women over several years increased the risk for non-vertebral bone fractures significantly.[684]

It is interesting to note that the two main forms of bone fracture associated with water fluoridation are hip and wrist fracture - as these two bone sites are primarily dependent on the quality of cortical bone. Given the increase in exposure of the population to fluoride and increasing frequency of osteoporosis, since the late 1960's in particular, one must consider the very real possibility that fluoride (even at levels felt to be safe and optimal for the prevention of dental caries) may, in fact, aggravate the risk of developing osteoporosis. This hypothesis was examined by Hillier et al.[685] who found that patients exposed to fluoridated water had increased amounts of fluoride in the cortical bone.

There is a growing weight of scientific research that demonstrates the associated risk of fluoride to osteoporosis. Epidemiological studies with water fluoridation suggest that communities with water fluoridation may have increased incidence of osteoporotic hip fractures.[686] This hypothesis was examined by Hillier et al.[687] who found that patients exposed to fluoridated water had increased amounts of fluoride in the cortical bone. It is know that the incidence of hip fracture is increasing more rapidly than can be accounted for by aging of the population. It has been observed in hip fractures studies[688] in the U.S., comparing two cities where water is fluoridated (Brigham City) and non--fluoridated (Cedar City) in Utah, that in the fluoridated (1 ppm) city, the hip fracture rate was twice as high as in the non-fluoridated city, in women around the age of 75. Men aged between 80-85 also had twice the hip fracture rate in fluoridated Brigham City.

[683] Cummings SR, Melton LJ, Epidemiology and outcomes of osteoporotic fractures. Lancet. 2002 May 18;359(9319):1761-7.
[684] Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Fluoride, The EFSA Journal (2005) 192, 1-65
[685] Hillier S, Cooper C, Kellingray S, Russell G, Hughes H, Coggon D, Fluoride in drinking water and risk of hip fracture in the UK: case-control study, Lancet 2000; 355: 265–69.
[686] Kleerekoper M, Center for Osteoporosis Research, Fluoride and the Skeleton, Critical Reviews in Clinical Laboratory Sciences, 33(2):139-161 (1996)
[687] Hillier S, Cooper C, Kellingray S, Russell G, Hughes H, Coggon D, Fluoride in drinking water and risk of hip fracture in the UK: a case-control study, Lancet 2000; 355: 265–69.
[688] Danielson C, Lyon JL, Egger M, Goodenough GK. Hip fractures and fluoridation in Utah.s elderly population. 1992;268(6):746-748.

It has been well documented that pharmacological doses of fluoride increase the risk of torsion-type fractures (such as hip fractures) despite the appearance of greater bone density. The results of a number of epidemiological studies[689],[690],[691],[692],[693],[694],[695],[696],[697],[698],[699] have indicated increased hip fractures in both naturally and artificially fluoridated areas and links between fracture rates and water fluoridation.

While it is generally regarded that fluoride's cumulative effect on bone can be devastating, it is well known that chronic ingestion of fluoride can cause osteofluorosis or skeletal fluorosis (crippling bone disease). This is further documented in the most recent U.S. EPA Fluoride Risk Assessment study.[700],[701]

The U.S. Department of Health and Human Services Public Health Service *Agency for Toxic Substances and Disease Registry* (ATSDR) has documented the effects of fluoride on human health which include *musculoskeletal,*

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[689] Sowers MR, Clark MK, Jannausch ML,Wallace RB, A Prospective Study of Bone Mineral Content and Fracture in Communities with Differential Fluoride Exposure, J. of Epidemiology, 1991, 133:7, 649-660

[690] Sowers MR, Wallace RB, Lemke JH, The relationship of bone mass and fracture history to fluoride and calcium intake: a study of three communities. Am J Clinical Nutrition, 1986 Dec, 44 (6): 889-898.

[691] Suarez-Almazor ME, Flowerdew G, Saunders DL, Soskolne CL, Russell A, The Fluoridation of Drinking Water and Hip Fracture Hospitalization Rates in Two Canadian Communities, Am J. Public Health, 1993 May, 83, 689-693.

[692] Riggs BL, et al., Effect of Fluoride Treatment on the Fracture Rate in Postmenopausal Women with Osteoporosis, New England J. of Medicine, 1990 March, 322:12, 802-9

[693] Lee.R.J. Fluoridation And Hip Fracture National Research Council Report: "Health Effects of Ingested Fluoride", Fluoride 1993 October, 26:4 274-277,

[694] Hillier S, Cooper C, Kellingray S, et al., Fluoride in drinking water and risk of hip fracture in the UK: a case-control study, Lancet 22 January 2000; 355: 265-269.

[695] Jacobsen SJ, O'Fallon WM, Melton III IJ. Hip fracture incidence before and after fluoridation of public water supply, Rochester, MN. American Journal of Public Health 1993; 83: 743-745.

[696] Toxicological Profile For Fluorides, Hydrogen Fluoride, and Fluorine, by the U.S. Agency for Toxic Substances and Disease Registry).

[697] Zipkin, I, The effects of the absorption of fluoride: IV The deposition of fluoride in human skeletal tissues as related to fluoride in drinking water, Arch. Indust. Health , 21: 329, 1960.

[698] Sogaard, C.H., Marked Decrease in Trabecular Bone Quality After Five Years of Sodium Fluoride Therapy - Assessed by Biomechanical Testing of Iliac Crest Bone Biopsies in Osteoporotic patients. Bone, 15(4):393-399, 1994.

[699] Riggs, B.L., Effect of Fluoride Treatment on the Fracture Rate in Postmenopausal Women Sogaard, C.H., Marked Decrease in Trabecular Bone Quality After Five Years of Sodium Fluoride Therapy - Assessed by Biomechanical Testing of Iliac Crest Bone Biopsies in Osteoporotic patients. Bone, 15(4):393-399, 1994.

[700] Fluoride Related Skeletal Effects: Evaluatison of Key Studies, health and Ecological Criteria Division, Office of Water, U.S. Environmental Protection Agency, Jan 2008.

[701] Fluoride: Exposure and Relative Source Contribution Analysis. Health and Ecological Criteria Division Office of Water, Environmental Protection Agency, December 2010.

reproductive (testicular), neurological and renal disorders.[702] Alarmingly, the ATSDR further observed "*that no data is available on the effects of intermediate-duration exposure to fluoride in humans while intermediate-duration exposure of animals to fluoride has resulted in effects on a number of organ systems, including bone, testes, kidney, neurobehavioral effects, and developmental effects.*" The ATSDR further noted that "*effects on the bone are commonly reported, including decreased bone growth, alterations in tooth enamel, delayed bone healing, and increased bone formation rate.*"

The U.S. *Agency for Toxic Substances and Disease Registry* has noted[703] that short-term high-dose fluoride studies have shown the same amount of fluoride accumulates in the bones of osteoporosis patients as would be found in some people who are chronically exposed to long-term "low" doses of fluoride (such as in fluoridated areas). People with renal insufficiency, for example, can incorporate four times more fluoride into bone than an average healthy individual and would therefore be more susceptible to the long-term effects of drinking "optimally" fluoridated water than the average individual.

In a county by county survey of hip fractures recorded in the U.S. in persons aged 65 or older, Jacobsen *et al.*[704] reported a significant relationship between osteoporotic hip fractures and the water fluoride content. The study involving a national analysis of data from the healthcare financing administration between 1984 and 1987 in the USA indicated a significant positive correlation between county-specific, age-adjusted incidence of hip fracture in white women aged 65 years or older, and the proportions of the counties' populations served with fluoridated water. This result is supported by another study in which a higher hip-fracture rate was recorded in a community with fluoridated water than in a nearby population without.[705] Research in France[706] showed that the risk of hip fracture was significantly increased in places where water fluoride was higher than 0·11 ppm.

The results of a study by Cooper *et* al.[707] reported a similar pattern in 39 counties in England. More recently, Danielson *et al.* [708] found an increased relative risk in hip fractures for a community whose water had been fluoridated to 1 ppm since 1966, compared with two control communities that had not been exposed to water fluoridation where the water fluoride level was 0.3 ppm. Similarly in a prospective study conducted in three rural

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[702] Interactive profile for Fluoride, U.S. Department of Health and Human Services Public Health Service Agency for Toxic Substances and Disease Registry, May 2004

[704] Jacobsen SJ, Goldberg J, Miles TP, Brody J, Stiers W, Rimm AA. Regional variation in the incidence of hip fracture. *JAMA* 1990; **264:** 500–02.
[705] Danielson C, Lyon JL, Egger M, Goodenough GK. Hip fractures and fluoridation in Utah's elderly population. *JAMA* 1992; **265:** 746–48.
[706] Jacqmin-Gadda H, Commenges D, Dartigues J-F. Fluorine concentration in drinking water and fractures in the elderly. *JAMA* 1995; **273:** 775–76.
[707] Cooper C, Wickham CA, Barker DJ, et al. Water fluoridation and hip fracture. *JAMA* 1991; 266: 513-4.
[708] Danielson C, Lyon JL, Egger M, et al. Hip fractures and fluoridation in Utah's elderly population. *JAMA* 1991; 268: 746-8.

communities in Iowa Sowers *et al.*[709] reported a faster rate of bone loss from the radius in women in the community with the highest fluoride content. Additional research has demonstrated that the concentration of fluoride in bones increased in an essentially linear fashion with an increase of fluoride in the drinking water.[710] To test whether water fluoride had a measurable effect on bone Hillier et al. [711] analyzed the fluoride content of the head section of the femoral bone. The investigation concluded that the fluoride content of cortical bone was increased in patients with higher exposures through drinking fluoridated water.

The relationship between trace elements including fluoride and bone quality was investigated by the Samuel Lunenfeld Research Institute, Canada.[712] The results demonstrated conclusively that increase in bone fluoride was associated with increased osteoid parameters and decreased bone microhardness. The results also suggested that the effects of fluoride and magnesium on bone quality may be exacerbated by their interaction with aluminium and that bone magnesium and aluminium contents were positively associated with bone pain and proximal myopathy (neuromuscular disease resulting in muscle weakness).

A recent study[713] examining the association between fluoride, magnesium, aluminium and bone quality in renal osteodystrophy observed that an increase in bone fluoride was associated with increased osteoid parameters and decreased bone micro hardness. Furthermore, bone magnesium and aluminium contents were positively associated with bone pain and proximal myopathy (weakness in the thigh and shoulder muscles). Most importantly, fluoride, magnesium and aluminium showed significant correlations with one another suggesting that the fluoride ion is binding with both elements and causing accumulation of magnesium fluoride and aluminium fluoride compounds in the bone.

The findings of this research suggest that bone fluoride may diminish bone micro hardness by interfering with mineralization and the effects of fluoride and magnesium on bone quality may be exacerbated by their interaction with aluminium. Aluminium compounds are used in purification of drinking water and can be found in treated water as aluminium fluorides.

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[709] Sowers MR, Clark MK, Jannausch ML. et al. A prospective study of bone mineral content and fracture in communities with differential fluoride exposure. *Am J Epidemiol* 1991; 133: 649-60.

[710] Zipkin, I.; McClure, F. J.; Leone, N. C.; Lee, W. A. Fluoride deposition in human bones after prolonged ingestion of fluoride in drinking water, Public Health Reports (1958), 73, 732-40 CODEN: PHRPA6; ISSN: 0033-3549

[711] Hillier S, Cooper C, Kellingray S, Russell G, Hughes H, Coggon D, Fluoride in drinking water and risk of hip fracture in the UK: a case-control study, Lancet 2000; 355: 265–69.

[712] Ng AH, Hercz G, Kandel R, Grynpas MD. Association between fluoride, magnesium, aluminum and bone quality in renal osteodystrophy. Bone. 2004 Jan;34(1):216-24.

[713] Ng AH, Hercz G, Kandel R, Grynpas MD. Association between fluoride, magnesium, aluminum and bone quality in renal osteodystrophy. Bone. 2004 Jan;34(1):216-24.

The link between fluoride exposure and weakening bone strength was accepted by the entire body of the U.S. Scientific Committee of the National Research Council in their review in 2006.[714]

It is interesting to note that a recent study examining the prevalence of eight chronic conditions in people aged over 50 in the Republic of Ireland (ROI) found that musculoskeletal pain was the most widely reported condition with a prevalence of 40%.[715] Remarkably, it is estimated that there are approximately 585,000 people in Ireland who suffer from chronic pain representing 36% of all households in Ireland. It is also known that up to 50% of Type ii diabetes sufferers may develop chronic pain associated with nerve damage.[716] It is now known that exposure to fluoride is a major risk factor associated with developing many chronic pain conditions.

This is a very significant fact given it has been scientifically observed and documented that the early stages of (sub-clinical) fluorosis is usually associated with stiffness, backache and joint pain which may suggest the diagnosis of rheumatism, rhematopid arthritis, ankylosing spondylitis and osteomalacia.[717],[718],[719],[720],[721] The U.S. ATSDR reported how in a study of 10 individuals with clinical manifestations of fluorosis[722], similar to those documented in the Irish study noted above, that a diet with adequate levels of calcium, vitamins C and E, and other antioxidants and access to drinking water with low levels of fluoride resulted in a decrease in urinary and blood fluoride levels and after a period of one year there was complete recovery of muscular weakness, polyuria, polydypsea, pain and rigidity in the joints as well as gastrointestinal complaints.[723]

Other musculoskeletal disorders/diseases include Ankylosing Spondylitis (AS) a long-term disease that causes inflammation of the joints between the spinal

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[714] National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C. p. 146.

[715] Multimorbidity and Disability in the Older Population of Ireland, Longitudinal Study of Ageing (TILDA),2011.

[716] Murphy, P. Neuropathic Pain, Chronic Pain, Oct 2008, Health Supplement Irish Indepenent Newspapers.

[717] Groth, E (1973).The Issues of Science and Public Policy. Air pollution Control in the San Francisco Bay Area, and Fluoridation of community water schemes. PH.D Dissertation. Department of Biological Sciences, Stranford University, May 1973.

[718] Teotia SPS, ET al. (1976) Symposium on the Non-Skeletal Phase of Chronic Fluorosis: The Joints. Fluoride. 9(1) 19-24.

[719] Cook. HA. 1971. Fluoride Studies in a Patient with Arthritis. The Lancet 1. 817

[720] Pak CY. (1989). Fluoride and osteoporosis. Proceedings of the Society for Experimental Biology and Medicine 191: 278-86.

[721] Boivin G, et al. (1989). Skeletal fluorosis: histomorphometric analysis of bone changes and bone fluoride content in 29 patients. Bone 10:89-99.

[722] Susheela AK, Bhatnagar M. 2002. Reversal of fluoride induced cell injury through elimination of fluoride and consumption of diet rich in essential nutrients and antioxidants. Mol Cell Biochem 234/235:335-340.

[723] Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine, U.S. Agency for Toxic Substances and Disease Registry, Dept Of Health & Human Services, 2003, P166

bones, and the joints between the spine and pelvis. AS is a painful, progressive rheumatic disease. It mainly affects the spine and sacroiliac joints but it can also affect other joints, tendons and ligaments. Rheumatoid arthritis (RA) is a long-term disease that leads to inflammation of the joints and surrounding tissues. It can also affect other organs. It is an auto-immune disease, which means the body's immune system mistakenly attacks healthy tissue.

Osteomalacia is a metabolic bone disease caused by defective bone mineralization leading to a softening of the bones. The softer bones seen in persons with osteomalacia have a normal amount of collagen, which gives the bones its structure, but lack the proper amount of calcium. Osteoporosis is a condition in which bones become porous and weak. As they lose strength, bones are more likely to fracture, particularly in the spine, hip, wrist, pelvis and upper arm. In children the condition is generally termed "rickets"; it can cause deformities such as bowed legs.

Rickets has been generally associated with calcium deficiency and low Vitamin D. Vitamin D helps the body to absorb calcium from food. The Health Service Executive and Food safety Authority in Ireland recently noted some concern regarding the re-emergence of rickets in Ireland. It is interesting to observe that the re-emergence of rickets in Ireland coincides with an alarming increase in dental fluorosis in Irish children.

Dental fluorosis is a biomarker of excess fluoride exposure and a sign of other adverse effects from excess fluoride ingestion. It is expected that children with dental fluorosis would be at higher risk of developing rickets as fluoride reduces the bio-availability of calcium in the body. It is also noteworthy that the European Food Safety Authority (EFSA) has observed that an infant's retention of fluoride in bone can be as high as 90% of the absorbed amount.[724] It is generally accepted that a lack of calcium is the major contributor factor to developing this disease. It may be that the recent increase in incidence in rickets in Ireland is due to a combination of high fluoride and low Vitamin D intake.

7.16.1 Vitamin D, Bone Health and Inhibition of Fluoride Toxicity

The Health Service Executive and the Food Safety Authority in Ireland have both published policy documents advising parents and healthcare providers to supplement the infant diet and foods with Vitamin D, for the purposes of maintaining infant bone health.[725],[726] It is remarkable that neither State body have at the same time highlighted the known risks of fluoride over-exposure for infants, in particular those consuming formula feed reconstituted with

[724] Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Fluoride. The EFSA Journal (2005) 192, 1-65

[725] Health Service Executive, Vitamin D Supplement for Infant's- Information For Health Professionals. 2010.

[726] Recommendations for a National Policy on Vitamin D Supplementation for Infant's, Food Safety Authority of Ireland, 2007.

fluoridated water, or for high-risk individuals with diabetes. It is astonishing that this association has not been reported given the high level of dental fluorosis documented in Ireland and more particularly given the scientifically documented impacts of fluoride on bone health.

While Vitamin D supplements are recommended for the treatment of 'rickets' it is alarming that as far back as 1940 definite radiographic evidence[727] was available in published studies demonstrating that fluorine markedly affects both the development of rickets and the healing produced by Vitamin D, yet this association has never been reported by the HSE or FSA. The latter research by Morgaretdge et al. found that overexposure to fluoride resulted in the development of "fluorine rickets" in animals. A significant observation of the study was that even at low concentrations the curative power of the Vitamin D had been definitely inhibited by fluorine and that the amount of inhibition was roughly proportional to the dose given.

In other words, where a child or adult may have low levels of Vitamin D in their diet, increasing their fluorine exposure, as occurs with water fluoridation, may result in a tipping point further limiting the benefits of this essential nutrient to the body and exposing the patient to other health complications. Based on this observation, it is entirely possible that it is the influence of overexposure to fluoride alone that is the principal causative agent in the recent re-emergence of rickets in Ireland.

This is supported by laboratory and animal studies by Morgaretdge et al. which demonstrated that the healing produced by the half unit of Vitamin D plus 3.0 mg. of fluorine was only 50% of that resulting from the same dose of Vitamin alone. Similarly, the 0.5 mg. dose of fluorine resulted in about a 10% inhibition in healing. The accepted treatment for patients who suffer from rickets or osteoporosis is Vitamin D and calcium supplements. Calcium supplements have been shown to prevent the toxic effects of fluoride in experimental animals[728],[729], because Vitamin D is known to facilitate gastro-intestinal absorption of calcium,[730] it is likely to have a counter the toxic effects of fluoride.[731] The latter study by Ekambaram et al. observed that orally administered calcium was likely to form insoluble CaF2 with fluoride available in the gastrointestinal tract. Thus, absorption of fluoride would be reduced

[727] Morgaretdge K,. Finn S B. The Institute of Optics and the Department of Biochemistry, University of Rochester, New York. Effect of Fluorine on the Activity of Vitamin D in Rachitic Rats. The Journal of Nutrtion.

[728] Ekambaram P, Paul V. Calcium preventing locomotor behavioral and dental toxicities of fluoride by decreasing serum fluoride levels in rats. Environ Toxicol Pharmacol 2001;9:141

[729] Ekambaram P, Paul V. Modulation of fluoride toxicity in rats by calcium carbonate and by withdrawal of fluoride exposure. Pharmacol Toxicol 2002; 90:53-8.

[730] Nicolaysen R, Eeg-Larsen N, Malm OJ. Physiology of calcium metabolism. Physiol Rev 1953;33:424-5.

[731] Ekambaram P, Paul V. Effect Of Vitamin D On Chronic Behavioral And Dental Toxicities Of Sodium Fluoride In Rats, Fluoride Vol. 36 No. 3 189-197 2003 Research Report

and may account for the protective effect of calcium on the toxic effects of fluoride.

Their laboratory and animal studies demonstrated how calcium supplements significantly reduced the serum fluoride level in animals with chronic fluoride exposure. In their study it was suggested that since Vitamin D is known to facilitate absorption of calcium, supplementation with this vitamin is likely to decrease availability of calcium in the gastrointestinal tract, resulting in uninhibited absorption of fluoride in the gastrointestinal tract. This view was supported by laboratory results showing that blood plasma fluoride levels remained unchained with the addition of Vitamin D and similar to those treated with sodium fluoride alone.

A further important observation in their study was that the deleterious effects of sodium fluoride on food intake, body weight gain, or exploratory motor activity (EMA) were significantly prevented when animals received Vitamin D as a supplement. This suggests that Vitamin D can inhibit the toxic biological and neurological effects of fluoride. It would appear therefore that Vitamin D may act as a psychological neurobiological inhibitor of the toxic effects of fluoride.

It is biologically plausible that the main beneficial function of Vitamin D is in the neuropsychiatric disease prevention; this observation is supported by recent research[732] published in the official journal of the International Society of Psychoneuroendocrinology.

7.16.2 Emerging and Future Risks

Research examined elsewhere in this report illustrates how such disorders would increase with exposure to fluoride. It is uncertain therefore why in such circumstances there is not at minimum a clear, simple and safe recommendation, as other public health authorities have provided, informing the public not to use fluoridated water in the preparation of infant formula feed. It also raises the public health urgency to ensure that adequate health and safety risk assessments are undertaken on water fluoridation products without further delay.

It is truly disturbing that while the lack of adequate scientific risk assessment has been documented repeatedly by international scientific committees for some time and in light of the public health risks, that any public or private agency would continue to inject chemicals of unknown toxicological consequences into a public water supply.

In Ireland, for adults osteoporosis is diagnosed in 1 in 3 women and 1 in 5 men over the age of 50.[733] The lifetime risk of osteoporosis fracture in women at

[732] Eyles DW, Feron F, Cui X, Kesby JP, Harms LH, Ko P, McGrath JJ, Burne TH. Developmental vitamin D deficiency causes abnormal brain development. Psychoneuroendocrinology. 2009 Dec;34 Suppl 1:S247-57.
[733] UCC, College of Science, Engineering and Food Science,

age 50 years is greater than the risk of breast cancer or cardiovascular disease.[734]

Currently, 300,000 people in Ireland over the age of 50 are estimated to have osteoporosis.

According to the Forum for Fluoridation Report (2002), in the year 1990 there were 1,509 hip fractures in the over 60 year olds; in 1999 this increased to 3,504 and by 2000 this figure had risen to 3,821. This represents an alarming increase and it has not been investigated whether exposure to fluoride may be a contributory factor. One assumes that fluoride exposure is a major factor given the findings of a recent study[735] published in the *British Journal of Radiology*, which found that individuals living in predominantly fluoridated communities in the U.S.A., at levels comparable to Ireland, had a substantially increased prevalence of osteoporosis for both sexes (55% in women, 68% in men) compared to a less fluoridated country (UK).

Dodds et al.[736] predicted that the incidence rate is expected to exceed 5,700 by 2026. Based on the current projector, it is likely that the expected incidence rate will far exceed this figure as the high risk sub-group of the population, those born following commencement of water fluoridation (1967) reach middle and old age.

The current cost of osteoporotic hip fractures to the Irish Government is estimated to be €402 million.[737] The HSE and Department of Health and Children estimated[738] in 2008 the inpatient cost of fall-related injury hospitalizations among older persons at approximately €59 million and inpatient hip fractures costs were estimated at €35 million. This figure does not include mortality linked to hip fractures. It is estimated that 20% of people aged 60+ who fracture a hip will die within 6-12 months due to the secondary complications of osteoporosis, which are blood clots, pneumonia or infection from being bed-bound.[739] This sadly equates to over 500 deaths per annum.

[734] WHO Study Group (1994) | Assessment of fracture risk and its application to screening for post menopausal osteoporosis | WHO Technical Report Series (843): pp 1-129

[735] Holt G, Khaw KT, Reid DM, Compston JE, Bhalla A, Woolf AD, Crabtree NJ, Dalzell N, Wardley-Smith B, Lunt M, Reeve J. Prevalence of osteoporotic bone mineral density at the hip in Britain differs substantially from the US over 50 years of age: implications for clinical densitometry. British Journal of Radiology 2002 Sep;75(897):736-42.

[736] Dodds M K,. Codd M B,. Looney A,. Mulhall K J. Incidence of hip fracture in the Republic of Ireland and future projections: a population-based study. Osteoporosis International Volume 20, Number 12, 2105-2110

[737] Irish Osteoporosis Society, 2010

[738] Report of the National Steering Group on the Prevention of Falls in Older People and the Prevention and Management of Osteoporosis throughout Life. June 2008

[739] Osteoporosis in the UK at... Breaking Point

It is now known that osteomalacia is especially prevalent in certain groups of the general population, such as in non-Caucasian immigrants living in Western Europe.[740] It is likely (but undocumented) that the prevalence of each of the above diseases may be significantly higher in Counties Cork and South Kerry compared to national averages due to the low bio-availability of calcium in drinking water sources as discussed previously in this report.
It is believed that fluoridation of drinking water supplies will increase the risk factor of developing these diseases as fluoride interacts with calcium thereby reducing its bio-availability for metabolism in the body.

It is possible that Ireland will see a marked increase in the prevalence of these diseases not only among the indigenous population but also as a consequence of the racial and ethnic demographics of Ireland changing significantly in the past twenty years. The number of non-Caucasian immigrants now living in Ireland is significant and as noted by Dunningan et al. non-Caucasians are especially prevalent to osteomalacia. The consequences for the native population who were born following water fluoridation may be even more dramatic. It is likely that as this group reach middle age the full implications of water fluoridation on bone health may become more evident.

As noted previously in this report and documented by Merrill et al[741], non-Caucasians also experience significantly higher levels of unstaged cancers of the stomach, rectum, colon, skin (melanoma), urinary bladder, thyroid, breast, corpus, cervix and ovaries. For both cancers and osteomalacia, calcium deficiency is a major significant factor. Any substance that is likely to affect the availability of calcium will increase the risk of ill health to any sensitive group of the population.

A remarkable study by Holt et al.[742], published in 2002, comparing hip bone density and hip bone mineral density (BMD) in Britain with a U.S. population, in order to assess geographic variation in the prevalence of osteoporosis, found that both male and female British subjects over 50 years old were found to have significantly higher mean BMD at the femoral neck and trochanter than their U.S. counterparts. The study also noted how the decline in BMD with age in British men appeared slower than in men in the U.S. British age-adjusted prevalences of osteopenia in women averaged 20% less than those of the U.S. whereas the prevalence of osteoporosis was substantially lower in British subjects of both sexes (55% in women, 68% in men).

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[740] Dunnigan MG, Paton JP, Haase S, McNicol GW, Gardner MD, Smith CM: Late rickets and osteomalacia in the Pakistani community in Glasgow. *Scott Med J* 1962, 7:159-167

[741] Merrill RM, Sloan A, Anderson AE, Ryker K. Unstaged cancer in the United States: a population-based study. BMC Cancer. 2011 Sep 21;11:402.

[742] Holt G, Khaw KT, Reid DM, Compston JE, Bhalla A, Woolf AD, Crabtree NJ, Dalzell N, Wardley-Smith B, Lunt M, Reeve J. Prevalence of osteoporotic bone mineral density at the hip in Britain differs substantially from the US over 50 years of age: implications for clinical densitometry. British Journal of Radiology 2002 Sep;75(897):736-42.

Given the information that is known about fluoride and its influence on bone mineralisation, the previous statistics have particular importance especially when one acknowledges the level of water fluoridation in the U.S. (approximately 65% of the population consume fluoridated water) compared to the UK (<10%).

It is likely, based on the policy of water fluoridation in Ireland, that the data for this country would be similar to that obtained from the U.S. population-based data demonstrating reduced bone mineral density at the hip, femoral neck and trochanter regions.

To the author's knowledge, however, no such studies have as yet been undertaken.

What is known however is that 6.5% of the population in Ireland is estimated to have oestoporosis[743] which is approximately twice the incidence rate recorded for the UK (3.2%).[744] It is clear that water fluoridation represents a significant risk factor for the development of this disease. The significance of any association between fluoride, osteoporosis rheumatoid arthritis, ankylosing spondylitis, osteomalacia, bone quality and fractures cannot be underestimated. These findings are of major significance, and in the interests of both public health and safety demand an immediate response from the State requiring the cessation of the water fluoridation policy without delay.

[743] Irish Osteoporosis Society, 2010
[744] Osteoporosis in the UK at... Breaking Point

7.17 Skeletal Fluorosis

Skeletal fluorosis is a disease involving changes in the structure of bones and calcification of ligaments, which results from the chronic intake of fluoride over a period of years or decades. The condition is irreversible.

Fluoride accumulation in hard tissues such as bone may be viewed as a defence mechanism against fluoride intoxication because of the removal of fluoride from body circulation.[745,746] Thus, retention by the skeleton may be one of the protective mechanisms employed by the animal against the toxicity of fluoride.[747]

Early clinical symptoms resemble arthritis, with patients experiencing pain and stiffness in bones and joints. Restriction of movement occurs in the spine, mainly in the cervical region, but also in the lower back, shoulder joint, hip and knee. Radiological symptoms may include osteoclerosis (thickening of the bones), periosteal bone formation and calcification of the interosseous membrane. The advanced stages of the disease, rarely seen in developed countries, include rigidity of the spine, deformities such as hyphosis and exostosis, wasting of the muscles and compression of nerves leading to paraplegia and quadriplegia in extreme cases.

Singh et al. reported that while "*(d)ental fluorosis is easily recognised, the skeletal abnormalities are not so obvious until the advanced stage of crippling fluorosis. However, radiological changes are discernible in the skeleton at a much earlier stage, and provide the only means of diagnosing the early and relatively asymptomatic stage of fluorosis. These cases may include young adults whose only complaints are vague pains noted most frequently in the small joints of hands and feet, the knee and those of the spine. Such cases frequent in the endemic area and may be misdiagnosed as rheumatoid or osteoarthritis. Such symptoms may be present prior to the development of definite radiological signs*." [748]

In temperate regions such as Ireland, it is likely that the early stages of skeletal fluorosis have been misdiagnosed as rheumatoid or osteoarthritis. Most of Irelands drinking water supplies were only fluoridated by the early 1970's and since fluoride accumulates in the bones over peoples lifetimes, it may be reasonable to expect cases of skeletal fluorosis to appear as people who were born in the 1960's and 70's and were exposed even higher levels of fluoridated water from infancy approach middle age.

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[745] Sigler, W.F., Neuhold, J.M., 1972. Fluoride intoxication in fish: A review. J. Wildlife Diseases 8, 252–254.
[746] Kessabi, M., 1984. M_etabolisme et biochimie toxicologique du fluor: Une revue. Rev. M_ed. V_et. 135, 497–510.
[747] Muhler J C: Effect of vitamin C on skeletal fluoride storage in the guinea pig. J Am Dent Assoc 56:335-40, 1958.
[748] Singh A, Jolly SS, Bansal BC, Mathur CC. Endemic Fluorosis. Medicine 42, 229-246 (1963)

Skeletal fluorosis is most likely to become prevalent among high risk groups in artificially fluoridated areas with low calcium and magnesium levels in the drinking water. Those at most risk will be individuals that as infants were formula-fed using fluoridated water, individuals who consume large volumes of high fluoride beverages such as certain teas, individuals with kidney malfunction and/or individuals with high water intake such as active sports people(especially those of below average body mass).

There are no properly conducted scientific studies to investigate the prevalence of skeletal fluorosis within this high-risk group. Though uncertain and unproven, the musculoskeletal pain observed in the latter Irish study[749] may be associated with fluoride accumulation in skeletal tissues, which acts as a depository for fluoride in the body.

It has been recommended by the WHO that where the risk for skeletal and dental fluorosis is high as a consequence of excess fluoride intake from drinking water, fluoride levels in drinking water should be reduced to safe levels, or a lower fluoride source used, especially for young children.[750] Unfortunately consumers in Ireland do not have access to non- fluoridated public water supplies presenting particular challenging social, legal and ethical issues pertaining to human rights.

According to the U.S. National Research Council fluoride is a biologically active ion with demonstrable effects on bone cells, both osteoblasts and osteoclasts. As noted in their review of 2006 "*perhaps the single clearest effect of fluoride on the skeleton is its stimulation of osteoblast proliferation*".[751]

Because fluoride stimulates osteoblast proliferation, there is a theoretical risk that it might induce a malignant change in the expanding cell population.
To fully comprehend this statement, one must examine the research findings of the Harvard Fluoride & Osteosarcoma Control Study.[752] Published in in 2006, it provides alarming evidence of the link between fluoride and cancer in young boys. The NRC report which was published before the Harvard report noted how the findings of the Harvard study could ultimately decide future public policy on water fluoridation. It would appear that, while the findings of the Harvard research have been largely accepted, the policy on water fluoridation remains to be changed.

[749] Multimorbidity and Disability in the Older Population of Ireland, Longitudinal Study of Ageing (TILDA),2011.
[750] Nutrients in Drinking Water, Water, Sanitation and Health Protection and the Human Environment World Health Organization, Geneva, 2005.
[751] U.S. National Research Council, Fluoride and Drinking Water: A scientific review of EPA's Standards. 2006.
[752] Fluoride & Osteosarcoma - Harvard Case-Control Study (2006)

7.18 Bone Cancer and Fluoride

Bone tumours make up about 3–5% of childhood cancers and less than 1% of cancers in adults. Of these, osteosarcoma is the most commonly diagnosed primary malignant bone tumour.[753] Around 10 cases of osteosarcoma are diagnosed in the Republic of Ireland each year.[754] Cancer deaths due to bone and joint malignant neoplasms represent 8.9% of all childhood and adolescent cancer deaths.[755]

In investigating the carcinogenesis of fluoride it is important to consider also that along with arsenic these two elements are the two most serious inorganic contaminants of drinking water.[756] While arsenic is now a well-established human carcinogen, before 2001 inorganic arsenic was found negative in all well-designed animal carcinogenicity studies. As with fluoride, only extremely harsh conditions induced cancerous growths.[757]

Fluoride was found to be an equivocal carcinogen by the U.S. National Cancer Institute Toxicology Program.[758] The NTP found that the incidence of osteosarcoma or bone cancers was dose dependent and could develop in male animals at concentrations above 0.8mg fluoride/kg body weight/day in drinking water.

A number of international epidemiological studies have found increased osteosarcoma rates in young men in fluoridated areas. Osteosarcoma is a rare bone cancer which mostly originates in the growing end of bones. The most recent Harvard study, noted previously, provides the most substantial evidence to date of the link between fluoridation of drinking water and bone osteosarcoma. The health risks are further compounded by the findings of the Harvard study which observed that fluoride levels in drinking water was associated with an increased risk of osteosarcoma for males, supported by additional research by Harvard College's School of Dental Medicine research[759] which demonstrated that pre-adolescent boys who drink

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[753] Mirabello, L,. Troisi R, J,. and Savage ,S A,.International osteosarcoma incidence patterns in children and adolescents, middle ages and elderly persons, Int. J. Cancer: 125, 229–234 (2009)

[754] Bone Cancer Research Trust

[755] The Epidemiology of Osteosarcoma, Pediatric And Adolescent Osteosarcoma, Cancer Treatment and Research, 2010, Volume 152, Part 1, 3-13, DOI: 10.1007/978-1-4419-0284-9_1

[756] Chouhan S, Flora S J S,. Arsenic and Fluoride: Two major groundwater pollutant.Indian Journal of Expermental Biology. Vol 48, July 2010. pp666-678.

[757] Chronic Arsenic Toxicity, U.S. Armed Forces Institute of Pathology, Washington D.C.

[758] NTP (National Toxicology Program) (1990). Technical Report on the toxicology and carcinogenesis studies of sodium fluoride in F344/N rats and B6C3F1 mice (Drinking water studies), Technical Report Series No 393

[759] Bassin EB, Wypij D, Davis RB, Mittleman MA. (2006). Age-specific Fluoride Exposure in Drinking Water and Osteosarcoma (U.S). Cancer Causes and Control 17: 421-8

fluoridated water are at a seven-fold increased risk of osteosarcoma[760], an often fatal cancer. Interestingly, blood fluoride levels were also observed to be significantly higher in patients with osteosarcoma than in control groups, according to research published in Biological Trace Element Research (April 2009). These results showed higher level of fluoride with osteosarcoma patients compared to others suggesting a role of fluoride in the disease.[761] Further research has also demonstrated that the concentration of fluoride in the bones increased in an essentially linear fashion with an increase of fluoride in the drinking water.[762]

The findings of the Harvard study are of particular significance as an earlier study[763], examining the rates of bone cancer in fluoridated and non-fluoridated areas, authored by Perry D. Cohn, Ph.D., M.P.H., for the New Jersey Department of Environmental Protection and the New Jersey Department of Health, documented that males under the age of 50 had 3 to 7 times as many bone cancers in the fluoridated areas. Males between 10-19 years old fared the worst.

A two-year study[764] conducted by the U.S.National Toxicology Program (NTP), using rats and mice provided positive evidence linking sodium fluoride in drinking water to osteosarcoma. The positive results of that study (in which malignancies in tissues other than bone were also observed), concurs with a host of data from tests showing fluoride's ability to cause mutations and data showing increases in osteosarcoma in young men in New Jersey[765], Washington and Iowa[766] based on their drinking fluoridated water.

Hoover et al.[767]found 47 and 79% increases in the incidences of bone and joint cancer and osteosarcoma, respectively, among males and females living in areas with fluoridated water. In contrast, 34 and 4% declines in bone and joint cancer and osteosarcoma, respectively, were found in the non-fluoridated areas.

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[760] One hundred and eighty-three osteosarcoma cases were recorded on the island of Ireland between 1994 and 2006

[761] Sandhu R, Lal H, Kundu ZS, Kharb S. Serum fluoride and sialic Acid levels in osteosarcoma. Biol Trace Elem Res. 2011 Dec;144(1-3):1-5. Epub 2009 Apr 24.

[762] Zipkin, I.; McClure, F. J.; Leone, N. C.; Lee, W. A. Fluoride deposition in human bones after prolonged ingestion of fluoride in drinking water, Public Health Reports (1958), 73, 732-40 CODEN: PHRPA6; ISSN: 0033-3549

763 Cohn PD. An Epidemiologic Report on Drinking Water and Fluoridation NewJersey Department of Health, November 1992.

764 Evaluation of the National Toxicology Program (NTP) Cancer Bioassay on Sodium Fluoride, 1991 Jun (Amherst), commissioned by the East Bay Municipal Utility District, Calabrese E, PhD

[765] A brief report on the association of drinking water fluoridation and the incidence of osteosarcoma among young males. Cohn, P.D. New Jersey Department of Health 1992.

[766] Surveillance, Epidemiology and End Results program. National Cancer Institute Review of fluoride benefits & risks. Department of Health & Human Services. F1-F7 1991.

[767] Hoover RN, Devesa SS, Cantor KP, et al. 1991b. Review of fluoride benefits and risks. Appendix F. Time trends for bone and joint cancers and osteosarcomas in the surveillance, epidemiology and end results (SEER) program. National Cancer Institute. Public Health Service. Bethesda, MD: Department of Health and Human Services.

In the Cohn study[768] of the New Jersey cancer registry data, significant increases in the osteosarcoma incidence risk ratios were found among males under the age of 20 years living in areas with fluoridated water.

It is uncertain why the NHS York Review (2000) excluded all of these studies showing an association between fluoridation and osteosarcoma in young males[769]. The York Review also excluded the Takahasi report rebutting the deficient epidemiological studies claiming to refute earlier research connecting higher rates of Down Syndrome births to elevated levels of fluoride in drinking water in the North Central United States. Importantly, however, the SCHER review undertaken in 2010 subsequently agreed that some epidemiological studies seem to indicate a possible link between fluoride in drinking water and osteosarcoma disparities.

Regardless of the risk or uncertainty of the dangers of fluoride on public health through reproductive or genetic abnormalities, it is clear that there exists insufficient research to exclude this risk with absolutely certainty. For such reasons the precautionary approach must be adhered to by the Government of Ireland, who have a legal and moral duty to protect its citizens.

A recent study[770] undertaken in Ireland by the National Cancer Registry, Cork could not prove beyond reasonable doubt that osteosarcoma incidence in the island of Ireland is related to public water fluoridation. An earlier study demonstrated the osteosarcoma rate in the Republic of Ireland (ROI), which has had community water fluoridation since the 1960's, had an incidence rate of 0.27/100,000, about 30% elevated compared to Northern Ireland (non-fluoridated) with an incidence rate of 0.21/100,000. [771] The incidence rate for Ireland is higher than for the entire UK. As with the rest of Europe, Northern Ireland does not fluoridate public drinking water supplies. Osteosarcoma is also documented in canine animals. It has also been documented[772] that osteosarcoma is the by far the most common bone tumour of canines. It has been noted[773] that this may be of significance where animals are drinking fluoridated water or as a result of high fluoride content of the animal feed. Osteosarcoma of the limbs is called appendicular osteosarcoma and accounts for 75-85% of the cases of bone cancer in canines. It is further documented that more than 60% of Irish wolfhounds die from osteosarcoma[774].

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[768] Cohn PD. 1992. An epidemiologic report on drinking water and fluoridation. Environmental Health Service. New Jersey Department of Health.

[769] Yiamouyiannis, J.A. Fluoridation and cancer: The biology and epidemiology of bone and oral cancer related to fluoridation. Fluoride 1993;26:83-96

[770] Drinking water fluoridation and osteosarcoma incidence on the island of Ireland. Comhar H, Deady S, Montgomery E, Gavin A. National Cancer Registry. 2009.

[771] Comparison of Rep.of Ireland and N.Ireland figures for Osteosarcoma 1994-2000. Rep.of Ireland figures: National Cancer Registry-2001Data

[772] Animal Health Trust, oncology research Group, Dr Mike Starkey, 2006

[773] Toxicology and carcinogenesis studies of Sodium Fluoride in F344/N Rats and B6C3F Mice (Drinking Water Studies) NTP Report Number 393

[774] 2006, Kennel Club/Small Animal Veterinary Association Purebred Dog Health Survey

In a published technical memorandum[775] by Wm L. Marcus, Ph.D., Senior Science Advisor, Criteria & Standards Division, ODW, U.S. Environmental Protection Agency, the significance of the research[776] by Yiamouyiannis and Burk is documented. Dr. Marcus observed that Yiamouyiannis and Burk's published epidemiology studies have since been revised twice, by Burk (former head of the Cytochemistry section at the National Institute of Health).

In these extensively peer-reviewed papers; the authors found that about 10,000 deaths a year are attributable to fluoride water treatment. Dr. Marcus observed that where the U.S. Public Health Service (U.S.PHS) method of analysis was applied to the database, it confirmed that 10,000 excess cancer deaths yearly were linked to fluoridation of water supplies. Dr. Marcus reported how this evidence has been tested most recently in the Pennsylvania Courts and found to be scientifically sound after careful scrutiny. There were three different short-term in vitro tests performed on fluoride and all these tests proved fluoride to be mutagenic. In this case, the mutagenicity of fluoride supports the conclusion that fluoride is a probable human carcinogen.

The significance of any association between fluoride, osteofluorosis, bone pain, proximal myopathy, musculoskeletal pain or other critical diseases cannot be underestimated. These findings are of major significance demanding, in the interests of public health and safety, an immediate response from the State requiring the cessation of the water fluoridation policy without delay.

[775] Wm L. Marcus, Ph.D., Senior Science Advisor, Criteria & Standards Division, ODW (WH-550D), Office of Water, Environmental Protection Agency. Fluoride Conference to Review the NTP Draft Fluoride Report, May 1 1990. Appendix 4

[776] Graham, J.R., Burk, O., and Morin, P. 1987. A current reStatement and continuing reappraisal concerning demographic variables in American time-trend studies an water fluoridation and human cancer. Proc Pennsylvania Academy of Sci. 61:138-146

7.19 Hypersensitivity to Fluoride

The minimum acute dose leading to gastrointestinal effects has been found to be 0.4 to 5 mg/kg body weight.[777],[778],[779] The acute toxicity dose is lower for the more soluble salts of fluorine, which may be present in fluoridated drinking water. The gastrointestinal effects arise from the action of hydrofluoric acid which is produced from fluoride salts in the stomach.[780]

Clinical reports of hypersensitivity to 1ppm fluoride in drinking water or 1mg per day in fluoride tablets have been published by Walbott, Feltman, Shea, Grimbergen and Petraburg.[781],[782],[783],[784],[785],[786],[787] Reactions included some of the following symptoms: gastrointestinal upsets, skin rashes, mouth sores, migraine like headaches, arthritic-like pains, dryness of the throat, excessive water consumption, frequent need to urinate, chronic fatigue, depression, nervousness and respiratory difficulties. These symptoms were observed to subside after discontinuation of the exposure to fluoride. To confirm the association double blind tests were undertaken.

Kaminsky L S et al. reported[788] that some individuals may experience hypersensitivity to fluoride-containing agents. This is supported by research published in the *Journal of Dental Medicine* which demonstrated that 1% of the population is allergic to fluoride.[789]

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[777] Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Fluoride, The EFSA Journal (2005) 192, 1-65

[778] Eichler HG, Lenz K, Fuhrmann M, Hruby K (1982). Accidental ingestion of NaF tablets by children--report of a poison control center and one case. Int J Clin Pharmacol Ther Toxicol 20: 334-338

[779] Whitford G (1996). The metabolism and toxicity of fluoride. 2nd revised edition. Monographs in Oral Science, Vol. 16, pp 156

[780] Spak CJ, Sjöstedt S, Eleborg L, Veress B, Perbeck L, Ekstrand J (1990). Studies of human gastric mucosa after application of 0.42% fluoride gel. J Dent Res 69: 426-429

[781] Waldbott GL, Burgstahler AW, McKinney HL. Fluoridation the great dilemma. Coronado Press, Lawrence, Kansas 1978

[782] Waldbott GL, Chronic Fluoride intoxication from drinking water, Int Arch, Allergy Immunol 7, 70-74 (1955)

[783] Feltman R, Kosel G. Prenatal and postnatal ingestion of fluoride: fourteen years of investigation; final report. *Journal of Dental Medicine* 16 190-198 1961

[784] Shea JJ, Gillespie SM, Waldbott GL, Allergy to Fluoride, *Annals of Allergy*, 1967 July, 25, 388- 391

[785] Grimbergen GW. A double blind test for determination of intolerance to fluoridated water (preliminary report). Fluoride 7 146-152 1974

[786] Petraborg HT. Chronic fluoride intoxication from drinking water (preliminary report). *Fluoride* 7 47-52 1974

[787] Petraborg HT. Hydrofluorosis in the fluoridated Milwaukee area. *Fluoride* 10 165-169 1977

[788] Kaminsky LS, Mahoney MC, Leach J, Melius J, Miller MJ. Fluoride: benefits and risks of exposure Critical Reviews in Oral Biology & Medicine, 1990;1(4):261-81

[789] Feltmnn, R. and Kosel, G., Prenatal and Postnatal Ingestion of Fluorides -- Fourteen Years of Investigation -- Final Report, Journal of Dental Medicine, 16:190, October 1961.

This latter observation means that in Ireland, with a population of approximately 4.6 million, 46.000 people will evidence some sensitivity in one way or another to fluoridated drinking water.

Petraborg[790,791] described a wide spectrum of symptoms in 27 persons exposed to fluoridated water. He considered that since none of the persons were aware that their drinking water was fluoridated or were familiar with the manifestations of fluoride toxicity, that the accounts of their illnesses were equivalent in validity to those associated with double-blind procedures. He noted that several patients were not convinced that something in their drinking water was causing their illness and resumed drinking fluoridated water. Relapses of their illnesses followed. The symptoms included extreme chronic fatigue, polydipsia, general pruritis, headaches and gastrointestinal symptoms.

Intolerance to fluoride may also be seen to occur, for example, through the formation of corrosive undissociated hydrofluoric acid when fluoride ions are in contact with acidic gastric secretions. This potential mechanism for fluoride damaging the gastroduodenal mucosa has been supported by Susheela et al.[792] along with other potential mechanisms such as enzyme system inhibition. By studying patients intensively, including by endoscopy and biopsy for histopathological and scanning electron microscope examination, Sushella et al. found that the gastroduodenal mucosa could be severely damaged by the toxic effects of fluoride resulting in dyspeptic symptoms.

Professor Sushella[793] concluded that:

- Ingested fluoride damages gastroduodenal mucosa.
- Gastrointestinal discomfort can be an early warning sign of fluorosis.
- Fluoride toxicity should be considered a possible reason for non-ulcer dyspepsia, especially in fluorosis endemic areas.
- Gastrointestinal discomfort during sodium fluoride therapy calls for extreme caution and close monitoring.
- Gastrointestinal discomfort in the form of dyspeptic symptoms should be an important diagnostic feature when identifying fluorosis patients and should not be dismissed as non-specific.

The New York State Department of Health acknowledge that some individuals may react idiosyncratically to fluoride.[794] Warnings regarding hypersensitivy to fluoride are also provided on certain topical dental products and

[790] Petraborg HT. Chronic fluoride intoxication from drinking water (preliminary report). *Fluoride* 7 47-52 1974

[791] Petraborg HT. Hydrofluorosis in the fluoridated Milwaukee area. *Fluoride* 10 165-169 1977

[792] Susheela AK, Das TK, Gupta IP et al. Fluoride ingestion and its correlation with gastrointestinal discomfort. Fluoride 25 5-22 1992. [1]Department of Anatomy, [2]Department of Gastroenterology, [3]Department of Otolaryngology, India Insitute of Medical Sciences

[793] Prof. A.K. Susheela, a histocytochemist and director of Fluorosis Research and Rural Development Foundation in India has over 70 studies on the adverse health effects of fluoride.

[794] New York State Department of Health, Fluoride: Report of benefits and risks of exposure Final Report. Sept 1989

medications containing fluoride. This was supported by studies by Epstein[795] demonstrating fluoridated toothpaste as a cause of acne-like eruptions in sensitive individuals.

This evidence is further supported by studies undertaken by Grimbergen[796] involving double-blind testing with 60 patients which demonstrated that certain individuals were intolerant to fluoride and that exposure to this could reproduce gastrointestinal symptoms, stomatitis, joint pains, polydipsia, headaches and visual disturbances.

The U.S. *Agency for Toxic Substances and Disease Registry* (ATSDR) reported a study by Susheela et al.[797] documenting 10 individuals with clinical manifestations of fluorosis. It was found that a diet with adequate levels of calcium, vitamins C and E, and other antioxidants and access to drinking water with low levels of fluoride resulted in a decrease in urinary and blood fluoride levels and a decrease in clinical signs. One year after intervention, there was complete recovery of gastrointestinal complaints, muscular weakness, polyuria, polydypsea, and pain and rigidity in the joints.[798]

Perhaps therefore it is worth ending with the words of Dr. H.Trendley Dean, the first director of the U.S. National Institute of Dental Research and subsequent leading exponent of water fluoridation, who found that "*(t)he same amount of fluorine that causes a mild toxic reaction in one individual may cause a severe reaction in another. In other words we are dealing with a low-grade chronic poisoning of the formative dental organ in which case some individuals may show a more severe reaction than others having a comparable fluorine intake*.[799]"

[795] Epstein, E. 1976, Fluoride toothpaste as a cause of acne-like eruptions, Arch, Dermotal.12.1033

[796] Grimbergen GW. A double blind test for determination of intolerance to fluoridated water (preliminary report). Fluoride 7 146-152 1974

[797] Susheela AK, Bhatnagar M. 2002. Reversal of fluoride induced cell injury through elimination of fluoride and consumption of diet rich in essential nutrients and antioxidants. Mol Cell Biochem 234/235:335-340.

[798] Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine, U.S. Agency for Toxic Substances and Disease Registry, Dept Of Health & Human Services, 2003, page 166

[799] Journal of The American Dental Association, August 1943

7.20 Dental Fluorosis

It is known that the systemic exposure of the population to fluoride from fluoridation of drinking water may impair normal development of enamel in the pre-eruptive tooth and cause fluorosis.[800] Dental fluorosis is a toxic effect that is consistent with prevailing risk assessment definitions of adverse health effects. Excessive intake of fluoride during enamel development can lead to enamel fluorosis, a condition of the dental hard tissues in which the enamel covering of the teeth fails to crystallize properly, leading to defects that range from barely discernable markings to brown stains and surface pitting.[801]

Exposure to fluoride during maturation causes a dose-related disruption of enamel mineralization resulting in widening gaps in its crystalline structure, excessive retention of enamel proteins, and increased porosity.[802] Maturation of various tissue systems including tooth formation has been used in the literature to describe the developmental stages of growing children. Primary baby teeth start to form between the sixth and eight weeks and permanent teeth begin to form in the twentieth week.[803] Clinically, mild forms of enamel fluorosis are evidenced by white horizontal striations on the tooth surface or opaque patches, usually located on the incisal edges of anterior teeth or cusp tips of posterior teeth (see photo below).

Mild Dental Fluorosis

In moderate to severe forms of fluorosis, porosity increases and lesions extend toward the inner enamel. After the tooth erupts, its porous areas may flake off, leaving enamel defects where debris and bacteria can be trapped. The opaque areas can become stained yellow to brown, with more severe structural damage possible, primarily in the form of pitting of the tooth surface. It is known that mild fluorosis can be induced by as low as 1 ppm fluoride in drinking water.[804]

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[800] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

[801] U.S. National Research Council, Fluoride in Drinking Water: A Scientific Review of EPA's Standards, Page 103-105

[802] U.S. National Research Council, Fluoride in Drinking Water: A Scientific Review of EPA's Standards, Page 103-105

[803] Ten Cate, A. R. (1998). *Oral histology: development, structure, and function*. St. Louis: Mosby. pp. 81–102

[804] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of

It is generally acknowledged that the high risk period for developing dental fluorosis appears to be in the first 3 years of life[805],[806] however, recent research by Levy et al.[807] clearly shows that the middle of the first year of life is most important in fluorosis etiology (a study of the causes of dental disease).

Because the severity of fluorosis is related to the duration, timing and dose of fluoride intake, cumulative exposure during the entire maturation stage, not merely during critical periods of certain types of tooth development, is probably the most important exposure measure to consider when assessing the risk of fluorosis.[808]

Several epidemiological studies clearly demonstrate the relationship between dental fluorosis in humans and the level of fluoride in water supplies. These studies and others have shown that, in a population, there is a direct relationship among the degree of enamel fluorosis, plasma and bone fluoride levels, and the concentration of fluoride in drinking water. [809]

DenBesten et al. concluded that dental fluorosis is a reflection of fluoride exposure only during the time of enamel formation. In addition, the degree of fluorosis is dependent not only on the total fluoride dose, but also on the timing and duration of fluoride exposure.

It has been accepted and reported that the prevalence of dental fluorosis is increasing in Ireland[810],[811] and it is acknowledged that water fluoridation can only be the principle cause of this increased incidence[812] as infants under six months do not consume any other food except breast milk or infant formula reconstituted with fluoridated water. Therefore the critical exposure pathway is through fluoridated water and, in particular, boiled fluoridated water used for infant formula feed.

Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

[805] Evans, R.W., and J.W. Stamm. 1991. An epidemiologic estimate of the critical period during which human maxillary central incisors are most susceptible to fluorosis. J. Public Health Dent. 51(4):251-259.

[806] Ishii, T., G. Suckling.1991. The severity of dental fluorosis in children exposed to water with a high fluoride content for various periods of time. J. Dent. Res.70(6):952-956.

[807] Levy, S.M., S.L. Hillis, J.J. Warren, B.A. Broffitt, A.K. Mahbubul Islam, J.S. Wefel, and M.J. Kanellis. 2002a. Primary tooth fluorosis and fluoride intake during the first year of life. Community Dent. Oral Epidemiol. 30(4):286-295.

[808] DenBesten, P.K. 1999. Biological mechanisms of dental fluorosis relevant to the use of fluoride supplements. Community Dent. Oral Epidemiol. 27(1):41-47.

[809] DenBesten, P.K. 1994. Dental fluorosis: Its use as a biomarker. Adv. Dent. Res. 8(1): 105-110.

[810] Forum on Fluoridation 2002

[811] Browne D, Whelton H, O' Mullane D, Oral Health Services Research Centre, University Dental School, Cork. Fluoride metabolism and fluorosis, Journal of dentisty, Volume 33 Issue 3, March 2005, Pages 177-186

[812] Levy, S.M., S.L. Hillis, J.J. Warren, B.A. Broffitt, A.K. Mahbubul Islam, J.S. Wefel, and M.J. Kanellis. 2002a. Primary tooth fluorosis and fluoride intake during the first year of life. Community Dent. Oral Epidemiol. 30(4):286-295.

It is remarkable that the Forum on Fluoridation committee established in Ireland would make a statement that "there is no evidence that the ingestion of infant formula reconstituted with fluoridated water is a risk factor for any condition other than dental fluorosis of the anterior permanent teeth."

Dental fluorosis is a biomarker of excess fluoride exposure in early childhood and therefore a possible sign of other adverse effects from excess fluoride ingestion including neurological damage, endocrine damage, skeletal disorders, and other possible harm to biological processes and organs.

In examining the impact of fluorine toxicity on infants in Ireland, one must clearly examine it on an individual response level to fluoride over-exposure, including factors such as body weight, activity level, nutritional factors, skeletal and bone health, cognitive development, risk of neurological disease, sudden death syndrome and other medical complications. No such study has ever been undertaken. It is scientifically irrational to expect a highly toxic enzymatic substance like fluoride to show a direct biomarker sign of excess intake with damage limited to only teeth (as considered by the Forum on Fluoridation) and not include its impact on bone development or its interplay with other biological, biochemical or behavioural health disorders.

This statement is particularly remarkable because the uptake of fluoride by bones is much higher in infants and young children than in adults[813] and the exposure of infants to fluoridated drinking water is a contributor to skeletal fluorosis in older people.[814] When fluoride is consumed in excess health problems may result, which affect the young and old[815]. At higher fluoride concentrations metabolic processes are affected in humans, and over-exposed individuals may suffer from skeletal or dental fluorosis, non-skeletal manifestations, or combinations of these maladies.[816] It has been agreed by both opponents and supporters of fluoridation that there has been a dramatic increase in the number of cases of fluorosis in Ireland in recent years. It is known that approximately 40% of individuals in Ireland suffer from dental fluorosis[817],[818], representing one in three children in the Republic of Ireland. It has been further demonstrated how by the age of 15 years approximately 1% of children in Ireland will have developed moderate dental

[813] Zipkin L et al. Unnary fluoride levels associated with the use of fluoridated waters. Public Health Report 17, 767-772 (1956).
[814] Mark Diesendorf, Ph.D. The Health Hazards of Fluoridation: A Re-examination, Human Sciences Program, Australian National University, Canberra. International Clinical Nutrition Review, April 1990 Volume 10, No 2
[815] The World Health Report 1996
[816] Susheela, A. K., Kumar, A., Bhatnagar M.and Bahadur, M.. Prevalence of endemic fluorosis with gastro intestinal manifestations in people living in some north Indian villages. Fluoride, 26, 97-104 (1993)
[817] Verkerk, Robert H.J. The paradox of overlapping micronutrient risks and benefits obligates risk/benefit analysis, Journal of Toxicology, Feb 2010.
[818] Lewis DW, Banting DW. Water fluoridation, current effectiveness and dental fluorosis. Community Dentistry Oral Epidemiology 1994;22:153-8.

fluorosis with another 1% developing severe dental fluorosis.[819],[820]

These figures are truly alarming as fluorosis is a condition which demonstrates chronic fluoride poisoning[821] in the form of staining on the teeth which indicates unhealthy toxicity in the body. It is remarkable that the study by Harding et al. identified that the prevalence of dental fluorosis in communities with no fluroidated water was as low as 1.5% compared to 37% in fluoridated communities. It was also documented that no children were observed with moderate or severe dental fluorosis in non-fluoridated communities.

Severe enamel fluorosis is treated to prevent further enamel loss and to address the cosmetic appearance of teeth. Treatments include bleaching, microabrasion and the application of veneers or crowns. Bleaching and microabrasion are typically used with the mild to moderate forms of enamel fluorosis. Bleaching is the least invasive procedure, but does not eliminate the dark stains associated with severe enamel fluorosis. Partial veneers, composite veneers and crowns provide the best aesthetic results for very severe enamel fluorosis, but are the most invasive treatments. It has been estimated that treatment costs for severe dental fluorosis are between €500 and €2,000 per tooth. Frequently, 10 to 20 teeth are treated and the average life span of a veneer is 15 years. Lifetime costs can be expected to be up to €75,000.[822]

Since both moderate and severe dental fluorosis were only observed to occur in communities consuming fluoridated water, it is reasonable to suggest that in light of these findings, and this information being provided to the HSE and state agencies in this report, that any further incidences of this disease that may occur due to a continuation of the policy of water fluoridation, in subsequent years may result in compensation cases against the State by innocent parties.

In quantifying the risk of fluoridation of drinking water it is interesting to note that research[823] undertaken by Dr. Robert H.J. Verkerk published in the *Journal of Toxicology*, examined the risk benefit analysis of fluoridation, for the purpose of limiting maximum dosage of minerals and supplements across the European Union. The study observed that conventional risk assessment on fluoride, as undertaken by European and U.S. authorities, if applied by public authorities in a manner which is consistent with that used for other nutrients,

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[819] North South Survey of Children's Oral Health in Ireland 2002. (Authors H.Whelton, E.Crowley, D.O'Mullane, M.Harding, H Guiney, M.Cronin, E.Flannery, V.Kelleher.) Final Report December 2006,

[820] Dental Fluorosis In Primary Teeth Of 5-Year-Olds In Ireland D.M. O'mullane1, M. Harding1, H.P. Whelton1, M.S. Cronin1, And J.J. Warren2, 1 University College Cork, Ireland, 2 University Of Iowa, USA

[821] Poisoning occurs when any substance interferes with normal body functions after it is swallowed, inhaled, injected, or absorbed. Fluoride interferes with bone and teeth development, icellurar metabolism, the central nervous system and cardiovascular system.

[822] Dr. Bill Osmunson DDS, MPH practicing family, cosmetic, and neuromusuclar dentist with a Master's Degree in Public Health. Washington Oregon. USA.

[823] Verkerk, Robert H.J. The paradox of overlapping micronutrient risks and benefits obligates risk/benefit analysis, Journal of Toxicology, Feb 2010.

would make public drinking water fluoridation programmes unfeasible in light of the dental fluorosis risk to children alone.

It is interesting to note therefore the previous findings of the York Review estimated the prevalence of fluorosis at a water fluoride level of 1.0ppm to be 48% and for fluorosis of aesthetic concern it was predicted to be 12.5% of the total population. The York Review further estimated that the number of people who would have to be exposed to water fluoride levels of 1.0ppm for one additional person to develop fluorosis of any level is six.

It is plausible therefore, given the current and historical level of fluoridation of public water supplies in Ireland, that 775,000 people are likely to have dental fluorosis in the country. Of these, based on York estimates, approximately one hundred and thirty thousand individuals will have fluorosis of aesthetic concern in Ireland.

It would appear, however, that these estimates may be considered low given the recent findings demonstrated that one one in three children in Ireland have dental fluorosis. As water fluoridation commenced in 1967 every adult born subsequently would be considered at high risk of having developed dental fluorosis. The majority of these were bottle-fed as infants increasing their exposure to risk. Approximately 2,500,000 adults are under the age of fifty in Ireland. Based on current estimates 830,000 are likely to suffer dental fluorosis with 8,300 individuals suffering severe dental fluorosis.

What it further highlights is that with 1,056,947 children under 18 years of age in Ireland and up to 40% of youth (>400,000 individuals) demonstrating chronic over-exposure to fluoride with definite fluorosis, the public water supply can no longer be justified, based on the precautionary principle, as a mechanism for exposing the population to fluoride. This is especially so when it is known to potentially harm so many of those using it.

> The precautionary principle or precautionary approach states that if an action or policy has a suspected risk of causing harm to the public or to the environment, in the absence of scientific consensus that the action or policy is harmful, the burden of proof that it is not harmful falls on those taking the action. This principle allows policy makers to make discretionary decisions in situations where there is the possibility of harm from taking a particular course or making a certain decision when extensive scientific knowledge on the matter is lacking. The principle implies that there is a social responsibility to protect the public from exposure to harm, when scientific investigation has found a plausible risk. These protections can be relaxed only if further scientific findings emerge that provide sound evidence that no harm will result. In the law of the European Union, the application of the precautionary principle has been made a statutory requirement.[824] The Government of Ireland in continuing with the policy of water fluoridation is therefore in violation of the Treaty of the European Union.

[824] Recuerda, Miguel A. (2006). "Risk and Reason in the European Union Law". *European Food and Feed Law Review* 5

8.0 INFANTS AND FLUORIDE

The proper approach to risk assessment in toxicology and the environment is to identify the high risk groups in the community and to set safety standards for daily doses with sufficient margin to protect them with a high degree of certainty. Based on clinical reports it may be concluded that if a young child, under 6, ingests a fluoride dose in excess of 15 mg fluoride/kg death is likely to occur. Therefore, the probably toxic dose, which can be defined as the threshold dose that could cause serious or life-threatening systemic signs and symptoms and will need immediate emergency treatment is considered to be 5 mg fluoride/kg.[825],[826]

The adequate intake of fluoride for infants aged from 0-6 months, as defined by the Food and Nutrition Board (FNB) Institute of Medicine of the National Academies, is 0.01mg/l.[827] It is an absolute certainty that all bottle-fed infants under 6 months of age bottle-fed with formula reconstituted from fluoridated water would exceed by multiples of 6-10 this recommended level. It is also evident, as noted by the U.S. *Agency for Toxic Substances and Disease Registry* (ATSDR), that damage may not be evident until a later stage of development. The agency reported in their toxicological profile of fluorides that children also have a longer remaining lifetime in which to express damage from over-exposure to such chemicals; this potential is particularly relevant to cancer.[828]

It is generally accepted that infants under 12 months are the highest risk group for developing dental fluorosis due to consumption of formula milk feed reconstituted with fluoridated water in the first year of life. Dental fluorosis is the most obvious visible toxic effect of fluoride. It is further known that the condition overwhelmingly applies to infants who are fed formula milk prepared with fluoridated water. Fluoridated water is the only public source of drinking water in the Republic of Ireland. This view is supported by UK Medical Research Council who found that individuals most likely to have supra-optimal fluoride intakes are formula-fed infants in fluoridated areas.[829]

Bottle-fed babies therefore can be described as a targeted risk. Yet astonishingly no warnings or recommendations are provided by the Health

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[825] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

[826] Whitford G, 1996 Fluoride toxicology and health effects Chapter 10. IN: Fluoride in Dentistry (second edition) Eds., O. Fejerskov, J. Ekstrand & B. Burt, Munksgaard, Copenhagen, 167 – 186

[827] FNB (Food and Nutrition Board) (1997). Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D and Fluoride. Institute of Medicine National Academy Press, Washington DC.

[828] Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine, U.S. Agency for Toxic Substances and Disease Registry, Dept Of Health & Human Services, 2003

[829] Medical Research Council working group report: Water fluoridation and health, September 2002

Service Executive (HSE) or the Food Safety Authority (FSA) to parents or healthcare professionals warning them of any health risks. Ireland unlike all other European countries (who don't practise water fluoridation) has one of the lowest levels of breastfeeding internationally and the lowest in Europe.[830] As a consequence the population of Ireland as a whole, over their lifetime, is one of the most over-exposed to the chemical fluoride toxin in the world.

While it is acknowledged universally that all infants should be exclusively breastfed from birth to about 6 months of age, and at least for the first 4 months of life, and preferably beyond the first year of life, the prevalence of breastfeeding in Ireland establishes that only 35% of infants are breastfed for the first three months compared to 75% for Sweden and 60% for Germany and Denmark respectively.[831] Approximately 25% of infants are breastfed in Ireland beyond four months meaning that 75% of infants in this country are therefore known to exceed the recommended daily intake of fluoride by consuming formula feed made up with fluoridated water.

As reported by SCHER, infants solely fed with a baby formula diluted with water containing 0.8 mg F/L ingest, at a minimum, **0.137 mg** F/kg/day compared with **0.001 mg F/kg/day** for an infant, who is solely breastfed.[832] Consequently, infant bottle-fed formula milk made from fluoridated water consumes at a minimum 137 times the total fluoride intake of that of a breastfed baby. When body weight is taken into account, non-nursing infants receiving formula made with water fluoridated at or near the level of 1 mg fluoride (F)/litre (L) or 1 part per million (ppm), who are less than one year old, have been estimated to have a fluoride intake on average of about three times that of adults[833].

This level of exposure is extraordinarily high and poses immediate and far reaching consequences for the nation's health yet public health authorities in Ireland have failed to warn healthcare practitioners and parents of any risk. It is noteworthy that Ireland has one of the highest incidences of dental fluorosis, neurological disease, including epilepsy and cardiovascular disease, in the world, as well as very high incidences of certain cancers and musculoskeletal pain. All of these diseases have been associated with the biological toxicity of fluoride on the human body as outlined in this report. Regardless of the long-term potential implications, precautionary approaches must recognize the unique vulnerability of the infant's developing human brain and control the intake of toxins such as fluoride that are associated with developmental neurotoxicity.

[830] Euro Growth Study, Conducted at 22 European Centres from 1992-1996
[831] WHO Regional Office for Europe.
[832] Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water. Scientific Committee on Health and Environmental Risks, Director General for Health & Consumers (2010).
[833] U.S. National Research Council of the National Academies. Fluoride in drinking water: a scientific review of EPA's standards. Washington, DC: The National Academies Press; 2006

The level of fluoride in formula milk reconstituted with fluoridated water could be deemed critical regarding the potential dental fluorosis that may result from high concentrations of dietary fluoride.[834],[835] There is a wide variation in the reported concentrations of fluoride in infant formula made with fluoridated drinking water. Fluoride concentrations of various commercially available infant milk formulations ranged from 0.7 ppm to 7.45 ppm.[836], [837],[838] It is generally accepted that infant formulas reconstituted with higher fluoride water can provide 100 to 200 times more fluoride than breast milk, or cows milk.[839],[840]

It has been found that a daily dose exceeding 0.05 mg/kg/day can result in dental fluorosis.[841] In fact an EU Scientific Committee found that a daily intake as low as 0.020 mg F-/kg body weight may result in mild forms of fluorosis in the permanent dentition.[842],[843],[844],[845]

An EU scientific committee examining the safety of fluoride compounds in children found as far back as 2003 that fluoride in infant formula was a problem as a consequence of water fluoridation. It is extremely worrying and inexcusable that this information was not acted on immediately by either the Food Safety Authority or Health Service Executive in Ireland. The failure to act on this information and inform parents is completely unacceptable.

[834] Fejerskow O, Manji F, Baelum V. The nature and mechanism of dental fluorosis in man. *J Dent Res.* 1990;69:699–700.
[835] Ekstrand J, Fomon SJ, Ziegler EZ, Nelson SE. Fluoride pharmacokinetics in infancy. *Pediatr Res.* 1994;35:157–163
[836] Rahul P, Hedge AM, Munshi AK. Estimation of the fluoride in human breast milk, cow's milk and infant formulate. *J Clin Pediatr Dent.* 2003;27:257–260
[837] McKnight-Hanes MC, Leverett DH, Adair SM, Shields CP. Fluoride content of infant formulas: soy-based formulas as a potential factor in dental fluorosis. *Pediatr Dent* 1988;10(3):189-94.
[838] Silva M, Reynolds EC. Fluoride content of infant formulae in Australia. *Aust Dent J* 1996;41(1):37-42.
[839] Levy SM, Guha-Chowdhury N. (1999). Total fluoride intake and implications for dietary fluoride supplementation. *Journal of Public Health Dentistry* 59: 211-23.
[840] Fomon SJ, Ekstrand J. Fluoride intake by infant's. *J Public Health Dent* 1999; 59 (4):229-34.
[841] Whitford GM. The physiological and toxicological characteristics of fluoride. *J Dent Res* 1990;69 Spec No:539-49; discussion 556-7.
[842] McDonagh MS, Whiting PF, Wilson PM, Sutton AJ, Chestnutt I, Cooper J, Misso K, Bradley M, Treasure E, Kleijnen J, 2000. Systemic review of water fluoridation. Brit Med J Oct 7, 321 (7265):844-5
[843] Levy SM, Hillis SL, Warren JJ; Broffitt BA, Mahbubul Islam AKM, Wefel JS, Kanellis MJ, 2002. Primary tooth fluorosis and fluoride intake during the first year of life. Community Dent Oral Epidemiol 30:286-95.
[844] Fejerskov O, Baelum V, Richards A 1996 (B) Dose-Response and dental fluorosis Chapter 9 . IN: Fluoride in Dentistry (second edition) Eds., O. Fejerskov, J. Ekstrand & B. Burt, Munksgaard, Copenhagen, 153 – 166
[845] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP) intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

In stark comparison, the U.S. Centres for Disease Control and Prevention (CDC) and the American Dental Association (ADA) in 2006 advised that using fluoridated water to mix formula could cause infants to develop fluorosis.

The ADA report stated that *"infants could receive a greater than optimal amount of fluoride through liquid concentrate or powdered baby formula that has been mixed with water containing fluoride during a time that their developing teeth may be susceptible to enamel fluorosis.*" The ADA warned that, in order to prevent tooth damage, fluoridated water should not be mixed into formula or foods intended for babies aged 1 and younger.

If the liquid (boiled water[846]) is fluoridated water, this corresponds to a daily intake of 0.12-0.18mg fluoride per kg. For a 6 month old infant of with a body mass of 8kg, this yields a daily dose range of 0.96-1.44mg, which is 4-6 times the daily doses currently recommended by American paediatricians for fluoride supplements in unfluoridated areas (0.25mg for children aged 0-2 years).[847], [848]

Through the second 6 months after birth, babies average fluid intake, per kg of body mass, decreases to about 120ml per kg of bady mass, but this is compensated for to some extent by fluoride in supplementary solids. So, formula-fed infants in fluoridated areas are likely to have high fluoride intakes over the whole of the first year of life. The *Journal of Public Health Dentistry* reported that more than 50 % of infants are formula fed by one month of age and these infants are likely to be exposed to high levels of fluoride for nine or ten months[849].

The long-term medical consequences of this level of fluoride intake on children have never been studied. A review of some of the possible health implications have been provided elsewhere in this report.

It is a certain fact that fluoride is now introduced at a much earlier stage of human development than in generations previous to the commencement of water fluoridation in the late 1960's and early 70's in Ireland. This has consequently altered the normal fluoride-pharmacokinetics in infants.[850] It has been further reported[851] in the Lancet that "(f)*luoridated water may be having its most devastating effects on the most vulnerable, those in utero and*

[846] In comparison earlier estimates yielded much lower values of fluoride intake by infant's by not taking into consideration the increase in concentration in fluoride as a consequence of boiling water. Fluoride concentations increase when water is boiled.
[847] Committee on Nutrition Fluoride supplementation. Paediatrics 77, 758-761 (1986)
[848] Erdal S and Buchanan SN. A quantitative look at fluorosis, fluoride exposure, and intake in children using a health risk assessment approach. *Environ Health Perspect* 2005;113(1):111-7.
[849] Forom S j Ekstrand, J (1999) Fluoride intake by infant's, Journal of public health dentristy 59(4)-229-34.
[850] Luke J. (1997). *The Effect of Fluoride on the Physiology of the Pineal Gland*. Ph.D. Thesis. University of Surrey, Guildford. p. 176.
[851] Grandjean P, Landrigan PJ. Developmental neurotoxicity of industrial chemicals. Lancet 2006;368:2167-78.

infants less than one year old, whose brains are most sensitive to developmental neurotoxins such as fluoride."

The most recent peer-reviewed Fluoride Risk Assessment and Relative Source Contribution study[852] undertaken by the U.S. EPA in response to the 2006 National Research Council (NRC) report: *Fluoride in Drinking Water* concluded that the main mechanism for fluoride intake in children is through ingestion of drinking water and diet. This report provides a technical examination of the human dose-response data on dental fluorosis, skeletal fluorosis and skeletal fractures. The study noted that fluoride exposure, particularly in children during the period of enamel development, can lead to dental fluorosis, a condition in which the enamel covering of the teeth fails to crystallize properly.

The U.S. EPA reported that possible problems resulting include enamel defects ranging from barely discernable markings to brown stains and surface pitting. The U.S. EPA further noted how prolonged high intake of fluoride, at any age, can result in skeletal fluorosis, an irreversible condition which may increase bone brittleness, and a potential increase in the risk of bone fracture.

In the U.S. EPA report, the daily fluoride intake for infants was calculated for different stages of infant development from first semester to twelve months of age. The fluoride dose for infants using boiled fluoridated water in formula feed was calculated at between 0.5 mg/kg/day and 0.12 mg/kg/day. The level of 0.5mg/kg/day would be regarded as a worst case scenario. This concentration would be in the order of **TEN TIMES the Minimum Risk Level** (MRL) for hazardous substances as estimated by the Agency for Toxic Substances and Disease Registry (ATSDR).

The ATSDR set a chronic maximum recommended limit (MRL) for fluoride *of* 0.06 mg/kg/day, which is based on chronic human data examining the most sensitive known endpoint of fluoride toxicity.[853] In regard to renal effects, an MRL value of 0.06 mg/kg/day was adopted as the Target Organ Toxicity Dose $(TTD)_{RENAL}$ for fluoride. In regard to reproductive effects, an MRL value of 0.06 mg/kg/day was adopted as the TTD_{REPRO} for fluoride. In regard to neurological effects, an MRL value of 0.06 mg/kg/day was adopted as the TTD_{NEURO} for fluoride.

Effects	MRL value	Target Organ Toxicity Dose for fluoride
Renal	0.06 mg/kg/day	TTD_{RENAL}
Reproductive	0.06 mg/kg/day	TTD_{REPRO}
Neurological	0.06 mg/kg/day	TTD_{NEURO}

852 Fluoride: Dose-Response Analysis For Non-cancer Effects Health and Ecological Criteria Division Office of Water, Environmental Protection Agency, January, 2011.
853 Interaction Profile For: Cyanide, Fluoride, Nitrate, And Uranium, U.S. Department of Health and Human Services Public Health Service Agency for Toxic Substances and Disease Registry. May 2004

It is clear, therefore, that the current levels of exposure to fluoride in infants in Ireland presents an immediate risk, not just for dental fluorosis, but for much wider health risks from this level of over-exposure in the population. The lower range of exposure presents a doubling of the MRL and is representative of infants at twelve months of age. The lower dose levels in themselves are also of concern as they likewise exceed the recommended levels and would therefore present a clear risk of dental fluorosis developing in children.

The level of exposure to fluoride through drinking water is particularly alarming given the findings of a new study by researchers Valdez-Jimenez, et al.[854] published in *Neurologia* which revealed *that "the prolonged ingestion of fluoride may cause significant damage to health and particularly to the nervous system.*" The study examined how fluoride induces changes in the brain's physical structure and biochemistry which affects the neurological and mental development of individuals including cognitive processes, such as learning and memory.[855] This has significant ramifications given the research findings by Archana Singh-Manoux et al. [856]just published in the *British Medical Journal*, which concluded that cognitive decline can start to deteriorate from the age of 45, and not 60 years, as had previously been believed.

Further clinicopathological studies show good correlation between neuropathology and the severity of cognitive decline[857],[858],[859]and neurofibrillary tangles and amyloid plaques, the hallmarks of pathology, which are known to be present in the brains of young adults.[860],[861] The Valdez study observed how fluoride can accumulate in the body, and it has been reported that continuous exposure to it causes damaging effects on body tissues, particularly the nervous system. The study also found that fluoride can be toxic by ingesting one part per million (ppm), and the effects are not immediate as they can take 20 years or more to become evident. It further observed that chronic exposure to, and ingestion of, the synthetic fluoride chemicals added to water supplies can cause serious brain and neurological

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[854] Neurología is the official scientific Journal of the Spanish Neurology Society (Sociedad Española de Neurología, SEN)

[855] Neurologia 2011 Jun;26(5):297-300. Epub 2011 Jan 20.Effects of the fluoride on the central nervous system, by Valdez-Jiménez L, Soria Fregozo C, Miranda Beltrán ML, Gutiérrez Coronado O, Pérez Vega MI

[856] Archana Singh-Manoux,, Mika Kivimaki, M Maria Glymour, Alexis Elbaz, Claudine Berr, Klaus P Ebmeier, Jane E Ferrie, Aline Dugravot, Timing of onset of cognitive decline: results from Whitehall II prospective cohort study, BMJ 2012;344: d7622

[857] Nelson PT, Braak H, Markesbery WR. Neuropathology and cognitive impairment in Alzheimer disease: a complex but coherent relationship. *J Neuropathol Exp Neurol*2009;68:1-14

[858] Fomon SJ, Ekstrand J, Ziegler EE. (2000). Fluoride intake and prevalence of dental fluorosis: trends in fluoride intake with special attention to infant's. *Journal of Public Health Dentistry* 60(3):131-9.

[859] Johnson DK, Storandt M, Morris JC, Galvin JE. Longitudinal study of the transition from healthy aging to Alzheimer disease. *Arch Neurol*2009;66:1254

[860] Braak H, Braak E. Frequency of stages of Alzheimer-related lesions in different age categories. *Neurobiol Aging*1997;18:351-

[861] Duyckaerts C, Hauw JJ. Prevalence, incidence and duration of Braak's stages in the general population: can we know? *Neurobiol Aging*1997;18:362-

damage. It is extremely alarming therefore that, as reported by SCHER[862], all infants of a certain age solely fed formula milk prepared with fluoridated water exceed the daily tolerable upper intake level for fluoride.[863]

This undoubtedly contributes to more than just dental fluorosis as the European Food Safety Authority (EFSA)have observed that in infants, retention of fluoride in bone can be as high as 90% of the absorbed amount[864]. The EFSA did, however, observe that excessive intake of fluoride during enamel maturation before tooth eruption from birth can lead to reduced mineral content of enamel and to dental fluorosis of deciduous but predominantly of permanent teeth.

The scientific panel for the EFSA also noted that the upper level (UL) for fluoride is 0.1 mg fluoride/kg/day in children aged between 1-8 years. For infants under 3 months of age, this requires an equivalent fluoride content in drinking water of < 0.5mg/l which cannot be achieved with fluoridated water. The EFSA observed that breast-fed infants have very low fluoride intakes from human milk (2-10 μg/L) and are not at risk of developing enamel fluorosis and that the use of fluoridated drinking water in preparing infant formula would considerably increase the fluoride intake for infants.

In comparison the EU Scientific Committee examining the safety of fluorine compounds in oral hygiene products for children found[865] that both water fluoridation studies and fluoride supplement studies indicate that a daily intake of 0.020 mg F-/kg body weight may result in mild forms of fluorosis in the permanent dentition.

The exposure of bottle-fed infants from formula reconstituted with fluoridated water to multiples of the recommended daily tolerable intake of fluoride is of particular concern in this regard. The concern is not just for dental fluorosis but because fluoride will accumulate in the pineal gland as well as bone and fluoride in blood plasma has profound impacts on inhibiting metabolic pathways with unforeseen negative consequences.

The obvious risks from dietary fluoride exposure are consequently of particular significance especially where it may be shown to effect synthesis and secretion of melatonin or other function of the pineal gland in humans or other important metabolic interactions.

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862 Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, Scientific Committee on Health and Environmental Risks, Director General for Health & Consumers (2010)

863 Strunecka, A. (1999) Aluminum plus Fluoride: a new deadly duo. Dement.1:2-3.

864 Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Fluoride. The EFSA Journal (2005) 192, 1-65

865 Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

This was acknowledged by the U.S. National Research Council in their review when they observed that "recent information on the role of the pineal organ in humans suggests that any agent that affects pineal function could affect human health in a variety of ways, including effects on sexual maturation, calcium metabolism, parathyroid function, postmenopausal osteoporosis, cancer, and psychiatric disease".[866]

> The UK Expert Group on Vitamins and Minerals concluded that with respect to fluoride, the most vulnerable groups are babies and young children.[867] They further observed that the most commonly observed clinical sign of chronic over-exposure to fluoride is fluorosis of the teeth, which they noted has been observed in almost 50% of children receiving the suggested 'optimal' level of fluoride (1 ppm) in their drinking water. The Scientific Committee observed that the fluoride content of the infant formulae if reconstituted with fluoridated water would exceed (by 2-3 times) the recommended 'optimal' upper daily fluoride limit of 0.07 mg/kg body mass.[868],[869],[870]

Prolonged consumption (beyond 12 months of age) of infant formula reconstituted with optimally fluoridated water could therefore result in large amounts of fluoride being ingested during enamel development of the anterior permanent teeth and could be a high risk factor for fluorosis of these teeth. The committee noted that no studies were available to examine the potential neuro-developmental impacts of such high levels of fluoride on infants.

Despite this knowledge, remarkably the Food Safety Authority in Ireland have remained silent on the risks associated with using fluoridated water in preparing infant feed. It is clear that the health and developmental impacts of water fluoridation on infants are incompletely understood and the short- and long-term consequences cannot be accurately predicted. This is especially so given that no adequate risk assessments have ever been undertaken to ensure the health, safety and protection of the public from water fluoridation.

While the primary objective of water fluoridation originally was to reduce social inequalities in dental health, it has been demonstrated by the NHS Centre for Reviews and Dissemination in 2000 (York Review) that there is little evidence to support this claim. Alarmingly, what may in fact be

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[866] U.S. National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C. p221-22

[867] Review Of Fluoride, Expert Group On Vitamins And Minerals, UK Expert Group on Vitamins and Minerals, EVM/01/03/P, May 2001.

[868] American Academy of Pediatrics, 1986.

[869] Whitford GM (1994) Effects of plasma fluoride and dietary calcium concentrations on GI absorption and secretion of fluoride in the rat. Calcif Tissue Int 54: 421-425.

[870] UK Expert Group on vitamins and minerals, review of fluoride, May 2001, evm/01/03/p, page 51

resulting is the opposite of this objective as the WHO and UNICEF have observed that it is the most educated women who breastfeed their infants significantly longer than women with lower levels of education.[871] Consequently, it is the infants from lower income families that may be disproportionately at risk from over-exposure to fluoride in the early years of life with lifelong consequences.

The WHO and UNICEF report on feeding and nutrition of infants noted that while there appears to be general consensus that an optimal fluoride intake should be secured through water fluoridation, fluoride supplements or the use of fluoridated toothpaste, this is based on one or either of the above intake pathways. For example, in countries such as those in Scandinavia, where public dental awareness is very high and alternative vehicles for fluoride (e.g. fluoridated toothpaste) are widely available *and* widely used, public authorities do not fluoridate drinking water. [872] In contrast, in Ireland fluoride intake is from **BOTH** water fluoridation and the use of fluoridated toothpaste, which was introduced into Ireland in the late 1960's. Through pursuing both public health policies, the Government of Ireland is placing even a wider sector of society at risk from over-exposure to fluoride.

It is noteworthy therefore that the *American Academy of Pediatrics* recommends NO fluoride supplements before the age of 6 months and not more than one cup of fluoridated water (0.25mg) from 6 months to 3 years of age. [873] In Denmark, the Public Health Authority recommended that "*a major effort should be used to avoid the use of fluoridated water for dilution of formula powders. In addition when economically feasible young infants fed formulas prepared from concentrated liquids should have these formulas made up with non fluordinated water.*" [874]

A significant correlation has also been demonstrated between the fluoride concentrations of breast milk and blood plasma levels of mothers. Breast milk is the major dietary intake of infants in the early stage of life. The dietary intake of fluoride in breastfeeding mothers will affect the levels of fluoride in their breast milk. The lower the exposure of mothers to fluoride the lower the concentration in breast milk. [875]

A recent extremely precise study undertaken in New Zealand examining the total fluoride intake of children aged between 3-4 years in both fluoridated and non-fluoridated communities provides some astonishing results. Guha et

[871] World Health Organization Europe and Unicef, Feeding and Nutrition of Infant's and Young Children, Guidelines for the WHO European Region, EU Series NO.87
[872] Nutrients in Drinking Water, Water, Sanitation and Health Protection and the Human Environment World Health Organization, Geneva, 2005.
[873] Pediatrics May 1998 Vol. 95, Number 5 RE9511
[874] Ekstrand, J 1996, Fluoride intake, Fluoride in Dentistry second edition Denmark pages 40-52.
[875] Yamur Şener, DDS, PhD,a Gül Tosun, DDS, PhD,a Firdevs Kahveciolu, DDS, PhD,b Alparslan Gökalp, DDS, PhD,c and Hasan Koç, MD, Fluoride Levels of Human Plasma and Breast Milk, Eur J Dent. 2007 January; 1(1): 21–24. PMCID: PMC2612944

al.[876] examined comprehensively the diets and total exposure of children to fluoride in both communities and found that the fluoride intake of children in non-fluoridated communities from foods, drinks and toothpaste if combined with supplementary fluoride (i.e. drinking fluoridated water) would increase the risk of dental fluorosis in children and exceed the dose level of 0.05 mg/kg/day which had been recommended as an Adequate Intake (AI) by the *American Institute of Medicine* (IOM, 1997). The reseachers recommend therefore that the level of supplementary fluoride be reduced.

This view was supported by Burt B.A (1999) in the *Journal of Public health Dentistry* when he concluded that, there are three reasons why (fluoride supplements) are inappropriate today among infants and young children in the United States. Evidence for the efficacy of water fluoride is weak, water fluoridation is a known risk factor for dental fluorosis, and fluoride has little pre-eruptive effect in caries prevention. It is notable that similar findings were observed in the NHS York Review in 2000 and in the SCHER review of 2010. Yet despite the clear scientific evidence demonstrating harm no intervention has been taken by the authorities in Ireland.

As far back as 1997, the *National Research Council* in Canada noted that fluoride is a persistent bio-accumulator, and is entering into human food-and-beverage chains in increasing amounts. [877] The *Canadian Journal of Human Lactation* warned that when infants are formula-fed, parents should be advised to reconstitute or dilute infant formula with deionised water (reverse osmosis, distilled, or low-fluoride bottled water) in order to reduce the amount of systemically ingested fluoride.[878]

Fomon et al.[879] in the *Journal of Public Health Dentistry* concluded that fluoride intakes of infants and children have shown a rather steady increase since 1930, are likely to continue to increase, and will be associated with further increase in the prevalence of enamel fluorosis unless intervention measures are instituted and recommended the use of water with relatively low-fluoride content (e.g. 0 to 0.3 ppm) as a dilutent for infant formulas and recommend that no fluoride supplements be given to infants.

Levy et al.[880] recommended in the same peer-reviewed journal that breastfeeding of infants should be encouraged, both for the many documented, general health benefits *and the relative protection against ingestion of excessive fluoride from high quantities of intake of fluoridated*

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[876] Guha-Chowdhury N, Drummond BK, Smillie AC.Total fluoride intake in children aged 3 to 4 years--a longitudinal study. J Dent Res. 1996 Jul;75(7):1451-7.
[877] Guha-Chowdhury N, Drummond BK, Smillie AC.Total fluoride intake in children aged 3 to 4 years--a longitudinal study. J Dent Res. 1996 Jul;75(7):1451-7.
[878] Brothwell D, Limeback H. (2003). Breastfeeding is protective against dental fluorosis in a nonfluoridated rural area of Ontario, Canada. *Journal of Human Lactation* 19: 386-90.
[879] Fomon SJ, Ekstrand J, Ziegler EE. (2000). Fluoride intake and prevalence of dental fluorosis: trends in fluoride intake with special attention to infant's. *Journal of Public Health Dentistry* 60(3):131-9.
[880] Levy SL, et al. (1995). Sources of fluoride intake in children. *Journal of Public Health Dentistry* 55: 39-52.

water used to reconstitute concentrated infant formula early in infancy and that the use of powder concentrate would be recommended only for those with low-fluoride water.

It is unfortunate therefore despite the inherent public health benefit that no data on chemical surveillance for fluoride concentrations on bottled waters marketed, produced or sold in Ireland are available from the Food Safety Authority (FSA). This is a matter that should be addressed by the Minister for Agriculture and Food and the FSA of Ireland to ensure, in particular, the adequate supply of safe drinking water for the preparation of infant foods where no other supply of low-fluoride water is available. Despite the obvious known and potential health risks for infants fed on formula milk reconstituted with fluoridated water, it is astonishing that the FSA have also reported that the effects of formula feeding on infant health have not been investigated in Ireland.[881]

This is even more alarming when one considers the interactivity of fluoride with aluminium. Aluminium is a potent neurotoxic agent in humans.[882] Aluminium fluoride (AlF4-) has been found to inhibit phospholipase in animal studies in a concentration-dependent response.[883] Inhibition was not caused by either fluoride or aluminium alone, however, inhibition of phospholipase D (PLD) was observed to be potently inhibited by aluminium fluoride.[884] The phospholipase family play critical roles in the initiation and modulation of inflammation and oxidative stress. Neurological disorders, such as ischaemia, spinal cord injury, Alzheimer's disease, multiple sclerosis, prion diseases and epilepsy are characterized by inflammatory reactions, oxidative stress, altered phospholipid metabolism, accumulation of lipid peroxides and increased phospholipase A_2 activity.[885]

It has been reported that aluminium by itself may not exert toxic affects on the nervous system. It may only become a toxin after joining together with a fluoride to become an aluminium fluoride.[886] Alarmingly, it has been reported that in drinking water treated with aluminium compounds the addition of fluoride at a level of 1 mg/L was found to increase the amount of soluble aluminium by a factor of 10.[887] It is known that in aluminium-treated drinking

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[881] Food Safety Authority of Ireland, Recommendations for a national infant feeding policy 1999.

[882] Aluminum Compounds Review of Toxicological Literature Abridged Final Report, USA National Institute of Environmental Health Sciences, October 2000

[883] Marc Paulais and . James Turne, Activation of the Na'-K'-2Cl- Cotransporter in Rat Parotid Acinar Cells by Aluminum Fluoride and Phosphatase Inhibitors, The Journal of Biological Chemistry, Vol. 267, No. 30, Issue of October 25, pp. 21558-21563,1992

[884] Li L, Fleming N., Aluminum fluoride inhibits phospholipase D activation by a GTP-binding protein-independent mechanism. FEBS Lett. 1999 Sep 24;458(3):419-23.

[885] Akhlaq A. Farooqui, Wei-Yi Ong, Lloyd A. Horrocks, Inhibitors of Brain Phospholipase A2 Activity: Their Neuropharmacological Effects and Therapeutic Importance for the Treatment of Neurologic Disorders *Pharmacological Reviews* September 2006 vol. 58 no. 3 591-620

[886] Professor Robert L. Isaacson, My Views on the Fluoridation of Water Fluoridation, Department of Psychology and Center for Developmental and Behavioral Neuroscience, Binghamton University, NY

[887] Integrated Laboratory Systems, Aluminum Compounds Review of Toxicological

water, fluorides can exist as fluoroaluminates.[888] The increased bio-availability of aluminium as a consequence of its interaction with fluoride in fluoridated drinking water is clearly therefore a major healthcare concern.

The targeted risk for bottle-fed infants using formula reconstituted with fluoridated water is particularly alarming. It is known that bone and liver are the tissues most frequently affected by increased absorption of aluminium. It is also reported that intake of aluminium is a factor in certain brain, neurological and renal diseases. Elevated plasma aluminium levels have been reported in healthy infants and in patients with chronic renal failure, total body aluminium can be markedly increased.[889]

The presence of aluminium-fluoride in infant formula contaminated from fluoridated compounds in drinking water is consequently of enormous concern. To our knowledge no research has been undertaken. It is remarkable that, despite concerns and warnings issued by the Committee on Nutrition in the peer-reviewed journal *Pediatrics*, no health impact studies or risk reduction recommendations have been provided by either the Food Safety Authority or the Health Service Executive in Ireland.

The Food Safety Authority has observed that the nutritional needs of each infant are unique, this is certainly true as each infant presents unique factors such as body weight, activity level, skeletal and bone health, cognitive development or other medical complications; as such the 'one size fits all' approach of water fluoridation poses many medical risks and concerns.

It is obvious therefore that the Government of Ireland, by supporting a policy of water fluoridation, cannot protect the rights of the individual to equality before the law. It is clear also that the pursuit of such a policy by Government would be in violation of the European Convention For the Protection of Human Rights and Fundamental Freedoms.

While the primary objective of water fluoridation was to reduce social inequalities in dental health, it has been demonstrated by the NHS Centre for Reviews and Dissemination in 2000 (York Review) and the SCHER Review (2010) that there is little evidence to support this claim.

Literature Abridged Final Report, U.S National Institute of Environmental Health Sciences, October 2000

[888] Integrated Laboratory Systems, Aluminum Compounds Review of Toxicological Literature Abridged Final Report, U.S National Institute of Environmental Health Sciences, October 2000

[889] Aluminum Toxicity in Infants and Children, Committee on Nutrition PEDIATRICS, Vol. 97 No. 3 March 1996

Alarmingly, what may in fact be resulting is the opposite of this objective as the WHO and UNICEF have observed that it is the most educated women who breastfeed their infants significantly longer than women with lower levels of education.[890]

Consequently, it is the infants from lower income families that may be disproportionately at risk from over-exposure to fluoride in the early years of life with lifelong consequences.

As previously mentioned in this section, the principle objective of water fluoridation was to promote social equality in the area of dental health, however, there is little evidence to support this claim. Rather, it appears that, if anything the fluoridation of water is creating social inequalities as it is more educated women who breastfeed their infants while women with lower levels of education are inclined to bottle-feed with formula reconstituted with fluoridated water thereby exposing their infants to fluoride from an early age.

The potential risk and future consequences arising from such exposure in the early years of life with lifelong consequences.

[890] World Health Organization Europe and Unicef, Feeding and Nutrition of Infant's and Young Children, Guidelines for the WHO European Region, EU Series NO.87

9.0 DENTAL HEALTH AND WATER FLUORIDATION

This review will not address in any detail the perceived benefits of water fluoridation as they have already been examined by scientific reviews elsewhere where it was concluded that the evidence in support of water fluoridation is both weak and inconclusive. It is also acknowledged that systemic fluoride exposure from fluoridation of drinking water may impair normal development of enamel in the pre-eruptive tooth and cause fluorosis.[891]

This report has attempted to review the conclusions of the most critical and important scientific investigations and research. This chapter will also attempt to present the many questionable findings of the perceived benefits of water fluoridation as this is important in any cost benefit analysis of public programmes.

There is now however, an increasing body of evidence to support the fact that the cariostatic activity of fluoride (the prevention of dental decay)is mainly due to its effects on erupted teeth. It has been found that the continual presence of fluoride in the saliva and in the fluid phase of dental plaque is critical to its mechanism of action. There is strong evidence that through its interaction with the surface of enamel, fluoride in saliva and dental plaque inhibits the demineralization, and promotes remineralization at the surface of the tooth. Hence, the predominant cariostatic effect is topical directly on the tooth surface. The recent decline in dental decay is due to the widespread use and daily exposure to fluoride from toothpaste[892] and not water fluoridation.

The NHS YORK review[893] (2000) concluded remarkably, especially after over 50 years of water fluoridation costing billions in taxpayers money, that it was unable to identify one high-quality study to show that the practice is effective or safe.

The SCCNFP study[894] (2003) found that systemic fluoride which results from fluoridation of drinking water may impair normal development of enamel in the pre-eruptive tooth and cause fluorosis.

Likewise the NRC[895] (2006) and SCHER[896] (2010) reviews have not demonstrated conclusively that fluoridation is in any way beneficial. Both scientific assessments

[891] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

[892] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

[893] NHS Centre for review and Dissemination, A systematic Review of water fluoridation, The University of York, Report 18

[894] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

concluded that water fluoridation was likely to have a beneficial effect but that the range could be anywhere from a substantial benefit to a slight disbenefit to children's teeth. That is it could be just as likely to be harmful as beneficial.

The SCHER review concluded that the scientific evidence for the protective effect of topical fluoride (toothpaste) application is strong, while the respective data for systemic application via drinking water (or water fluoridation) is less convincing. This statement is remarkable especially in the current economic crisis where countries are forced to decide to cut funding for critical health, infrastructure and educational programmes. Faced with limited resources, countries must decide to reduce general expenditure by limiting resources devoted to education or healthcare for children or the wider population.

In the long run it is apparent that continuing to invest much-needed public monies into such a questionable programme is unthinkable. Not to mention the potential enormous costs of undertaking the necessary risk assessments that are required urgently on the fluoride products to protect public health.

It is abundantly clear, that following comprehensive and robust scientific examination by international peer-reviewed scientific committees, the perceived minor benefits of water fluoridation do not justify continued public expenditure. The following section presents further information that would justify this informed view.

9.1 Fluoride's Lack of Benefit

Current scientific literature is generally finding little or no effectiveness from fluoridation.[897],[898],[899],[900],[901],[902],[903],[904],[905],[906],[907],[908],[909],[910],[911],[912],[913],[914],[915],[916]

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[895] U.S National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C.

[896] Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, Scientific Committee on Health and Environmental Risks, Director General for Health & Consumers 2010

[897] "Fluorosis prevalence increased significantly with higher water fluoride levels; however, caries prevalence did not decline significantly." Hong L, Levy S, Warren J, Broffit B. (2006). Dental caries and fluorosis in relation to water fluoride levels. *ADEA/AADR/CADR Conference*, Orlando Florida, March 8-11, 2006.

[898] "No fluoride, socioeconomic status or beverage variables were significantly associated with lesion progression." Warren JJ, Levy SM, Broffitt B, Kanellis MJ. (2006). Longitudinal study of non-cavitated carious lesion progression in the primary dentition. *Journal of Public Health Dentistry* 66(2):83-7.

[899] "In the present study, fluoridated water did not seem to have a positive effect on dental health, as it might have been expected in a community with the respective caries prevalence." Meyer-Lueckel H, et al. (2006). Caries and fluorosis in 6-and 9-year-old children residing in three communities in Iran. *Community Dentistry and Oral Epidemiology* 34:63-70

[900] "The WHO data do not support fluoridation as being a reason for the decline in dental decay in 12 year olds that has been occurring in recent decades." Neurath C. (2005). Tooth decay trends for 12 year olds in nonfluoridated and fluoridated countries. *Fluoride* 38:324-325

[901] "Our analysis shows no convincing effect of fluoride-intake on caries development." Komarek A, et al. (2005). A Bayesian analysis of multivariate doubly-interval-censored dental data. *Biostatistics* 6:145-55.

[902] "Levels in fluoridated and non-fluoridated areas were similar." Harding MA, et al. (2003). Dental erosion in 5-year-old Irish school children and associated factors: a pilot study. *Community Dental Health* 20(3):165-70.

[903] "There was no statistically significant difference between DMFT in municipalities of the same size, regardless of the presence or absence of fluoride in the water supply..." Sales-Peres SH, Bastos JR. (2002). [An epidemiological profile of dental caries in 12-year-old children residing in cities with and without fluoridated water supply in the central western area of the State of Sao Paulo, Brazil]. *Cadernos de Saude Publica* 18: 1281-8

[904] "Water fluoridation status of the children's area of residence did not have a significant effect on Early Childhood Caries (ECC) at the 0.1 level of significance in the unadjusted logistic regression analysis, nor was it found to be a confounder of the effect of race/ethnicity on ECC prevalence in the multivariable model." Shiboski CH, et al. (2003). The association of early childhood caries and race/ethnicity among California preschool children. *Journal of Public Health Dentistry* 63(1):38- 46

[905] "[E]ven a longitudinal approach did not reveal a lower caries occurrence in the fluoridated than in the low-fluoride reference community." Seppa L. et al. (2002). Caries occurrence in a fluoridated and a nonfluoridated town in Finland: a retrospective study using longitudinal data from public dental records. *Caries Research* 36: 308-314

[906] "The magnitude of [fluoridation's] effect is not large in absolute terms, is often not statistically significant and may not be of clinical significance." Locker, D. (1999). Benefits and Risks of Water Fluoridation. An Update of the 1996 Federal- Provincial Sub-committee Report. Prepared for *Ontario Ministry of Health and Long Term Care*

[907] "[R]esults of recent large-scale studies in at least three countries show that, when similar communities are compared and the traditional DMFT index of dental caries is used, there is no detectable difference in caries prevalence. This has been demonstrated for schoolchildren in the major cities of New Zealand, Australia, the US and elsewhere." Diesendorf,M. et al. (1997). New Evidence on Fluoridation. *Australian and New Zealand Journal of Public Health*. 21: 187-190

[908] "Higher fluoride proportions appeared to be associated with lower dfs + DFS, with an estimated difference between fluoridated and non-fluoridated groups of 0.65 decayed or filled surfaces per child, but this association was not statistically significant. The effects of fluoridation on the other outcomes were small and not statistically significant." Domoto P, et al. (1996). The estimation of caries prevalence in small areas. *Journal of Dental Research* 75:1947-56

[909] "Children attending centers showed no significant differences (in baby bottle tooth decay) based on fluoride status for the total sample or other variables." Barnes GP, et al. (1992). Ethnicity, location, age, and fluoridation factors in baby bottle tooth decay and caries prevalence of head start children. *Public Health Reports* 107: 167-73

[910] "The fluoride incorporated developmentally – that is, systemically into the normal tooth mineral – is insufficient to have a measurable effect on acid solubility." Featherstone JDB, M.Sc., Ph.D. , Cover Story; J American Dental Association, Vol. 131, July 2000, p. 890.

[911] "It is no longer acceptable to use fluoride supplements on large populations, even if the caries rate is higher than average." Limeback H. "A re-examination of the pre-eruptive and post-eruptive mechanism of the anticaries effects of fluoride: is there any anti-caries benefit from swallowing fluoride?" Community Dentistry and Oral Epidemiology 27: 62-71, 1999.

[912] Confounding factors such as delay in tooth eruption are not included in studies. See Komarek A, et al. Biostatistics. 2005 Jan;6

[913] McDonagh, M., P. et al 2000a. A Systematic Review of Public Water Fluoridation. NHS Centre for Reviews and Dissemination, U. of NY

[914] Leroy R, et al. (2003). The effect of fluorides and caries in primary teeth on

The Canadian Pediatric Society recommended that no fluoride should be given to infants before teeth have erupted.[917] This statement supports the recent findings published in the *Journal of American Dental Association* where it was reported that consumption of infant formula made up from fluoridated water was associated with a higher prevalence of enamel fluorosis in the permanent dentition[918]. This finding was supported by further published research[919] by David G. Pendrys, D.D.S., Ph.D.

A further study by Lewis et al.[920] on water fluoridation its current effectiveness and dental fluorosis concluded that the portion of dental fluorosis due to water fluoridation is now (40%) and more importantly that the effectiveness of water fluoridation alone cannot now be determined.

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permanent tooth emergence. Community Dentistry and Oral Epidemiology31(6):463-70

915 "Fluoride's caries-preventive properties initially were attributed to changes in enamel during tooth development because of the association between fluoride and cosmetic changes in enamel and a belief that fluoride incorporated into enamel during tooth development would result in a more acid-resistant mineral. However, laboratory and epidemiologic research suggests that fluoride prevents dental caries predominately after eruption of the tooth into the mouth, and its actions primarily are topical for both adults and children." Centers for Disease Control; MMWR Weekly Report. 1999;48:933-940.

916 "In 1970, during a meeting in Switzerland on fluoride research, I was astounded to hear the statement from a European cardiologist of great reputation that the mechanism of action of fluoride against dental caries was entirely topical! At that time I believed, along with the majority of American caries researchers, that fluoride worked because it became incorporated into enamel – especially developing enamel – to increase its resistance to acid demineralization. We thought that where this could not be accomplished preeruptively by water fluoridation, we ought to try to achieve the same goal posteruptively by short-term regimens of very highconcentration fluoride solutions and gels. I thought that my European colleague was very poorly informed. Now, twelve years later, I continue to be impressed by the wisdom of his assertion. Probably it was not completely correct; absolute statements about biological processes rarely are. However, each year since then the evidence has continued to accumulate to support the hypothesis that the anti-caries mechanism of fluoride is mainly a topical one. As a direct consequence any method which places particular emphasis on incorporation of bound fluoride into dental enamel during formation may be of limited value. Therefore, there is limited scientific data to support the assertion that systemic fluoride treatment should be initiated from shortly after birth." Fejerskov O. et al. "Rational use of fluorides in caries prevention". Acta Odontol. Scand. 1981, 39:241-249.

917 The use of fluoride in infants and children, Nutrition Committee, Canadian Paediatric Society (CPS), Paediatr Child Health 2002;7(8):569-72, Reference No. N02-01

918 Infant Formula and Enamel Fluorosis A Systematic Review, Philippe P. Hujoel, MSD, PhD, Lívia G. Zina, DDS, MSc, Suzely A.S. Moimaz, DDS, MSc, PhD and Joana Cunha-Cruz, DDS, PhD, J Am Dent Assoc, Vol 140, No 7, 841-854.

919 Pendrys, David G. D.D.S., Ph.D. Risk Of Enamel Fluorosis In Nonfluoridated And Optimally Fluoridated Populations: Considerations For The Dental Professional , J Am Dent Assoc, Vol 131, No 6, 746-755

920 Lewis DW, Banting DW. Water fluoridation, current effectiveness and dental fluorosis. Community Dentistry Oral Epidemiology 1994;22:153-8.

The observation of this study would be supported by the findings of an Irish study[921] which demonstrated that the percentage of tooth surfaces with fluorosis was higher in communities with water fluoridation compared to non-fluoridated areas. The study observed that 30% of the children were diagnosed with definite fluorosis in areas with water fluoridation while only 1.2% of children were diagnosed with fluorosis in the non-fluoridated areas.

The SCHER review accepted that systemic exposure to fluoride in drinking water is associated with an increased risk of dental and bone fluorosis in a dose-response manner without a detectable threshold. SCHER observed that limited evidence from epidemiological studies available points towards adverse health effects following systemic fluoride consumption, e.g., carcinogenicity, developmental neurotoxicity and reproductive toxicity, but using a weight of evidence approach these observations cannot be substantiated due to a lack of proper scientific assessment (further studies required to prove beyond reasonable doubt).

All scientific reviews and assessments of water fluoridation to date have called for an evaluation of new scientific developments on its hazard profile, further details of which are provided in Chapter 9 of this report. Given the urgency that should be pursued in undertaking this important work, there continues to be unacceptable delays in commencing these studies.

Notwithstanding this, there exists a considerable and growing body of data that challenges the public health benefits of fluoridation as noted in various published peer-reviewed journals across a range of fields including science, medical, public health dentistry, toxicology, neurotoxicology, environment, epidemiology, biological chemistry, pathology, molecular biology, neurology and radiology; the practice of water fluoridation remains in a few countries.

In the 21st century, Ireland remains the lone country within Europe with its legislative policy of mandatory water fluoridation. On April 9, 2003, the only city in the country of Switzerland to fluoridate its water supply (Basel-population 170,000) decided, by parliamentary vote, to end its water fluoridation programme after 41 years. Basel's Health & Social Commision (GSK) conducted a re-examination of fluoridation's merits. The GSK presented their advice to end water fluoridation in a report[922] delivered to Basil's city parliament. The GSK report expressed serious doubts about the effectiveness of water fluoridation and believed that other cities in Europe were equally successful at reducing tooth decay without water fluoridation. The parliament voted 72-23 against water fluoridation after reviewing GSK's recommendations.

A similar review of fluoridation was undertaken in Natick (pop. 35,000)

[921] Dental Fluorosis In Primary Teeth Of 5-Year-Olds In Ireland D.M. O'mullane1, M. Harding1, H.P. Whelton1, M.S. Cronin1, And J.J. Warren2, 1 University College Cork, Ireland, 2 University Of Iowa, USA

[922] Commission on Health and Social Issues (GSK) GSK - "Bericht der Gesundheits-und Sozialkomission des Grossen Rates zum Anzug Rene Brigger betreffend Fluoridierung des Basler Trinkwassers" [9229/P975485]. 2003 Report which led to the cessation of water fluoridation in Basel.

Massachusetts, USA.[923] The Natick Fluoridation Study Committee reached the firm conclusion that the risks of over-exposure to fluoride far outweigh any current benefit of water fluoridation. The summary findings of the Natwick committee are provided in Appendix 6. As it currently stands, the following western European countries have rejected water fluoridation: Austria, Belgium, Denmark, Finland, France, Germany, Iceland, Italy, Luxembourg, Netherlands, Norway, Sweden and Switzerland.

The only western European country whith a policy for mandatory water fluoridation is Ireland where over 73% of the population ingest artifically fluoridated water. The result is that the population of Ireland remains one of the most fluoridated in the world, and one of only two countries in the world (Ireland & Singapore) with a nationally-mandated legislative fluoridation policy.

This continues in Ireland despite fluoride being added to commercial toothpaste in the early 1970's where it has since become the most common and internationally accepted method for controlling dental caries. The policy of water fluoridation in Ireland is therefore superfluous. as it has there being a general consensus that the major reasons for the general decline of tooth decay worldwide, both in non-fluoridated and fluoridated areas, is the widespread use of fluoridated toothpaste, improved nutrition and overall improved general and dental health (antibiotics, preservatives, hygiene etc).

It is of interest to note that despite a policy of fluoridation lasting four decades, the improvement in dental hygiene is no better in Ireland than in any of the other European countries where drinking water is not fluoridated. It is obvious that there exists within the public health profession and scientific community significant doubts regarding the very limited to marginal benefit of adding chemical compounds such as fluoride products to drinking water for oral health benefits. In the most recent EU review of the risks and benefits of fluoridation[924] SCHER stated the following:

- That topical application of fluoride is most effective in preventing tooth decay and that no obvious advantage appears in favour of water fluoridation compared with topical prevention. Hence, there is no need to ingest fluoride to derive its purported benefit for teeth.
- That the continued systemic exposure of fluoride from whatever source is questionable once the permanent teeth have erupted. Therefore there is no medical benefit to the wider population.
- That there is a risk of dental fluorosis in children in EU countries with systemic fluoride exposure.
- That fluoride can weaken bone and increase the risk of bone fractures under certain conditions[925].

[923] Norman R. Mancuso, Ph.D. Benedict J.Gallo, Ph.D. J ason Kupperschmidt, B. Alfred J. Murray, M. S. T. Harlee S. Strauss, Ph.D. Natick Fluoridation Study Committee Report 9/27/97

[924] Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, Scientific Committee on Health and Environmental Risks (SCHER) Director General for Health & Consumers (2010).

[925] McDonagh M, Whiting P, Bradeley M, Cooper J, Sutton A, Chestnut I, Misso K, Wilson

- That enamel fluorosis seen in areas with fluoridated water has been attributed to inappropriate high fluoride intake.
- That numerous studies have demonstrated that exposure to fluoride levels during tooth development can result in dental fluorosis.
- That excess systemically absorbed fluoride may impair normal development of enamel in the pre-eruptive tooth.
- That water fluoridation was intended to have a beneficial effect on caries prevention but could also induce fluorosis with a very narrow margin of exposure.
- That the systemic exposure to fluoride in drinking water is associated with an increased risk of dental and bone fluorosis.
- That the benefits of fluoridation to adult and elderly populations in terms of reductions in coronal and root decay are limited.

The Chairman, Professor Trevor A. Sheldon, of the Advisory Group for the systematic review on the effects of water fluoridation has observed that while there is some evidence to demonstrate that water fluoridation is effective in reducing caries, the overall quality of the research that these findings are based on is questionable.

Professor Sheldon further and most importantly stated[926] "*that the review did not show fluoridation to be safe,*" but "*found water fluoridation to be significantly associated with high levels of dental fluorosis which was not characterised as "just a cosmetic issue*". The review also found that " *there was little evidence to show that water fluoridation has reduced social inequalities in dental health*".

In a published article in the *Journal of the American Dental Association*[927], it was stated that "(f)*luoride incorporated during tooth development is insufficient to play a significant role in caries protection*" and that "*(i)n fact, fluoridation of municipal water supplies resulted in greater tooth decay overall in some studies and has not been demonstrated to be effective in prevention of decay even in the most convincing studies*".

Research data from Auckland, New Zealand demonstrated that there was virtually no difference in tooth decay rates between the fluoridated and non-fluoridated communities.[928] The data demonstrated directly conflicting observations that the percentage of 12 and 13 year old children who were free of tooth decay - that is, had perfect teeth - was greater in the non-fluoridated part of the district.

Other large-scale surveys from Missouri and Arizona in the USA have since revealed the same picture, that is that there is no real benefit to teeth from

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P, Treasure E, Kleijnen J (2000) A systematic report on public water fluoridation. NHS Center for Reviews and Dissemination. York, UK, University of York: 259.

[926] Letter from Professor Trevor A. Sheldon, Chairman of the Advisory Group for the systematic review of fluoridation, Appendix 1.

[927] Ref: Featherstone JDB. The science and practice of caries prevention. J Am Dent Assoc 2000;131:887-899.

[928] Colquhoun J. New evidence on fluoridation. Social Science and Medicine 19 1239-1246 1984.

fluoride in drinking water[929]. Further research based on extensive and multiple studies has presented considerable evidence to question the efficacy of fluoridation.

There appear to be at least four different publications from the U.S., Canada and New Zealand that have reported similar or lower tooth decay rates in non-fluoridated areas as compared to fluoridated areas.[930],[931],[932],[933]

Professor Steelink in Tucson, Arizon in the USA obtained information on the dental status of all schoolchildren – 26,000 in total – as well as information on the fluoride content of Tucson water.[934] Professor Steelink found: *"When we plotted the incidence of tooth decay versus fluoride content in a child's neighborhood drinking water, a positive correlation was revealed."* In other words, the more fluoride a child drank, the more cavities appeared in the teeth.

Hardy Limeback, B.Sc., Ph.D., D.D.S., head of the Department of Preventive Dentistry for the University of Toronto and President of the Canadian Association for Dental Research has stated that "*children under three should never use fluoridated toothpaste or drink fluoridated water. And baby formula must never be made up using Toronto tap water. He remarked that Vancouver, never fluoridated, has a lower cavity rate than Toronto, which has been fluoridated for 36 years*". [935,936]

In 1998, the results of a fifty-year fluoridation experiment involving Kingston, New York (non-fluoridated) and Newburg, New York (fluoridated) were published.[937]

In summary, it observes that there was no overall significant difference in rates of dental decay in children in the two cities, but children in the fluoridated city showed significantly higher rates of dental fluorosis than children in the non-fluoridated city.

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[929] Hildebolt CF, Elvin-Lewis M, Molnar S et al. Caries prevalences among geochemical regions of Missouri. American Journal of Physical Anthropology 78 79-92 1989.

[930] Colquhoun, J. 1987. Comm. Health Studies. 11:85.

[931] Gray, a. 1987. J. Canadian Dental Assoc. 53:763.

[932] Hildebolt, C.F. et al. 1989. Amer J, Physiol. Anthropol. 78:79-92.

[933] Diesendorf, M. 1986. Nature. 321:125.

[934] Jones T, Steelink C, Sierka J. Analysis of the causes of tooth decay in children in Tucson, Arizona. Paper presented at Annual Meeting of the American Association for the Advancement of Science, San Francisco, USA, February 1994. Abstract in Fluoride 27 (4) 238 1994.

[935] Forbes B. Prominent researcher apologizes for pushing fluoride. Mesa, Ariz., Dec 5, 1999. Available at: http://www.apfn.net/messageboard/101704/discussion.cgi.5.html.

[936] Limeback H. A re-examination of the pre-eruptive and post-eruptive mechanism of the anti-caries effects of fluoride: is there any anti-caries effect from swallowing fluoride 1999;27(1):62-71.

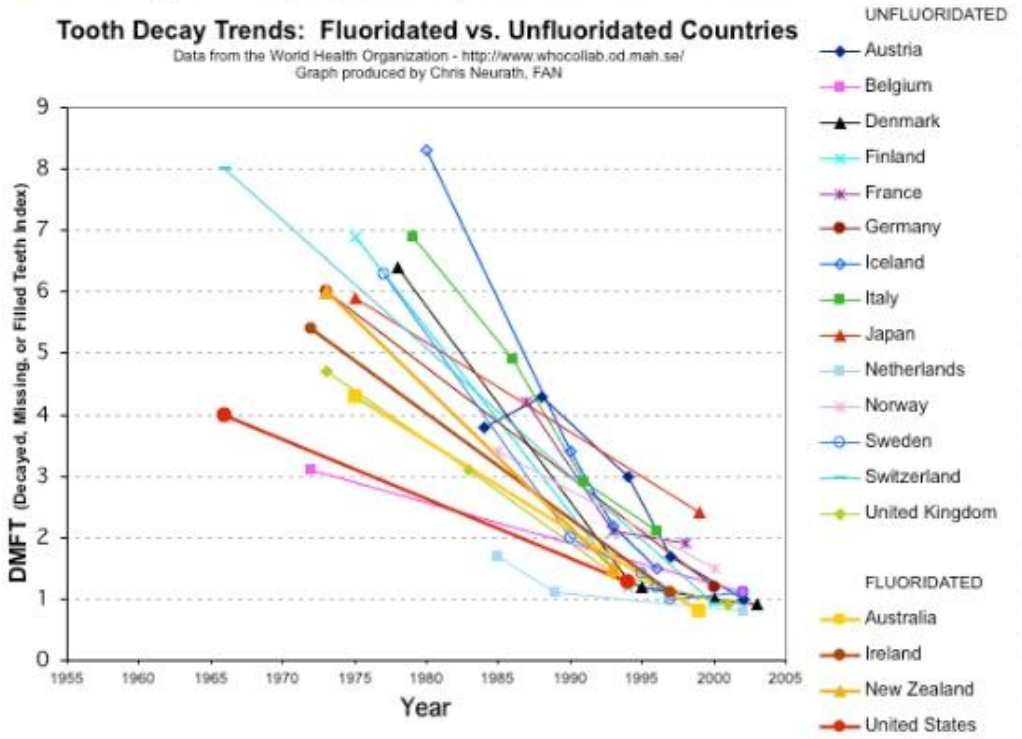
[937] Recommendations for fluoride use in children. Kumar, J.V. and Green, E.L. New York State Dent. J. (1998) 40-47.

Other key findings on tooth dental hygiene trends in fluoridated and non-fluoridated countries have reported the following stated observations (references provided for each source):

"In most European countries, where community water fluoridation has never been adopted, a substantial decline in caries prevalence has been reported in the last decades, with reductions in lifetime caries experience exceeding 75%".[938]

"Graphs of tooth decay trends for 12 year olds in 24 countries, prepared using the most recent World Health Organization data, show that the decline in dental decay in recent decades has been comparable in 16 nonfluoridated countries and 8 fluoridated countries which met the inclusion criteria of having (i) a mean annual per capita income in the year 2000 of US$10,000 or more, (ii) a population in the year 2000 of greater than 3 million, and (iii) suitable WHO caries data available."[939]

World Health Organization Data (2004) -
Tooth Decay Trends (12 year olds) in Fluoridated vs. Unfluoridated Countries:



"The WHO data (above) does not support fluoridation as being a reason for the decline in dental decay in 12 year olds that has been occurring in recent decades.[940]

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[938] Pizzo G, et al. (2007). Community water fluoridation and caries prevention: a critical review. *Clinical Oral Investigations* 11(3):189-93.
[939] Neurath C. (2005). Tooth decay trends for 12 year olds in nonfluoridated and fluoridated countries. *Fluoride* 38:324-325.
[940] Neurath C. (2005). Tooth decay trends for 12 year olds in nonfluoridated and fluoridated countries. Fluoride 38:324-325.

"Although the prevalence of caries[941] *varies between countries, levels everywhere have fallen greatly in the past three decades, and national rates of caries are now universally low. This trend has occurred regardless of the concentration of fluoride in water or the use of fluoridated salt, and it probably reflects use of fluoridated toothpastes and other factors, including perhaps aspects of nutrition".*[942]

"the decline in dental decay began in the early 1970s in most industrialized countries, notably those European countries without water fluoridation. The explanation is the introduction, widespread use and daily exposure to fluoride from toothpaste. Fluoride toothpaste now account for over 90% of toothpaste sales in most economically developed countries. The reduction in caries is mirrored by the significant increase in the use of fluoride toothpaste by the public despite no paralleled reduction in sugar intake".[943],[944]

"It is remarkable... that the dramatic decline in dental caries which we have witnessed in many different parts of the world has occurred without the dental profession being fully able to explain the relative role of fluoride in this intriguing process. It is a common belief that the wide distribution of fluoride from toothpastes may be a major explanation, but serious attempts to assess the role of fluoridated toothpastes have been able to attribute, at best, about 40-50% of the caries reduction to these fluoride products. This is not surprising, if one takes into account the fact that dental caries is not the result of fluoride deficiency." [945]

"The current reported decline in caries tooth decay in the US and other Western industrialized countries has been observed in both fluoridated and non-fluoridated communities, with percentage reductions in each community apparently about the same".[946]

"In lifetime residents of fluoridated areas 47% had evidence of erosion; in 21% erosion had progressed to the dentine or pulp. The corresponding figures in non-fluoridated areas were 43% and 21% respectively... Levels in fluoridated and non-fluoridated areas were similar ". [947]

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[941] Definition of caries: Tooth decay. The destruction of tooth enamel and dentin. Segen's Medical Dictionary, 2011.
[942] Cheng KK, et al. (2007). Adding fluoride to water supplies. *British Medical Journal* 335(7622):699-702.
[943] Rolla G, Ögaard B,de Almeida Cruz R, 1991. Clinical effect and mechanism of cariostatic action of fluoride-containing toothpastes: A review. Int Dent J 41: 171 – 174.
[944] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003
[945] Aoba T, Fejerskov O. (2002). Dental fluorosis: chemistry and biology. *Critical Review of Oral Biology and Medicine* 13: 155-70.
[946] Heifetz SB, et al. (1988). Prevalence of dental caries and dental fluorosis in areas with optimal and above-optimal water-fluoride concentrations: a 5-year follow-up survey. *Journal of the American Dental Association* 116: 490-5.
[947] Harding MA, et al. (2003). Dental erosion in 5-year-old Irish school children and associated factors: a pilot study. *Community Dental Health* 20(3):165-70.

"[R]esults of recent large-scale studies in at least three countries show that, when similar communities are compared and the traditional DMFT index of dental caries is used, there is no detectable difference in caries prevalence. This has been demonstrated for schoolchildren in the major cities of New Zealand, Australia, the US and elsewhere." [948]

"Higher fluoride proportions appeared to be associated with lower dfs + DFS, with an estimated difference between fluoridated and non-fluoridated groups of 0.65 decayed or filled surfaces per child, but this association was not statistically significant. The effects of fluoridation on the other (beneficial dental health) outcomes were small and not statistically significant".[949]

"Lifetime exposure to fluoridation is associated with average reductions of 2.0 dmfs and between 0.12 and 0.30 DMFS per child compared with non-exposed children." (Note: DMFS = Decayed, Missing & Filled Surfaces. There are 128 tooth surfaces in a child's mouth. This study found a difference of 0.12 to 0.30 decayed surfaces, out of 128, between children in fluoridated & non-fluoridated communities)". [950]

"Data from Head Start surveys show the prevalence of baby bottle tooth decay is about three times the national average among poor urban children, even in communities with a fluoridated water supply." [951]

"We found that caries prevalences do vary between the geochemical regions of the state. In the total sample, however, there were no significant differences between those children drinking optimally fluoridated water and those drinking sub optimally fluoridated water".[952]

"Parents who put their children in bed with propped milk bottles contributed to the formation of dental caries in their infant's because almost no saliva flows during sleep" (AAPD, 2009; Hallett & O'Rourke, 2002; Lin & Tsai, 1999; Twetman et al., 2000.)[953],[954],[955],[956]

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[948] Diesendorf, M. et al. (1997). New Evidence on Fluoridation. *Australian and New Zealand Journal of Public Health*. 21: 187-190.
[949] Domoto P, et al. (1996). The estimation of caries prevalence in small areas. Journal of Dental Research 75:1947-56.
[950] Spencer AJ, et al. (1996). Water fluoridation in Australia. Community Dental Health 13(Suppl 2):27-37.
[951] Von Burg MM et al. (1995). Baby Bottle Tooth Decay: A Concern for All Mothers. Journal of Pediatric Nursing 21: 515-519.
[952] Hildebolt CF, et al. (1989). Caries prevalences among geochemical regions of Missouri. *American Journal of Physical Anthropology* 78:79-92.
[953] American Academy of Pediatric Dentistry (AAPD). (2009). Clinical guidelines on infant oral healthcare.
[954] Hallett, K., & O'Rourke, P. (2002). Early childhood caries and infant feeding practice. Community Dental Health, 19, 237- 242.
[955] Lin, Y., & Tsai, C. (1999). Caries prevalence and bottle feeding practices in 2-yearold children with cleft lip, cleft palate, or both in Taiwan. Cleft Palate Craniofacial, 36(6), 522-526.
[956] Twetman, S., Garcia-Godoy, F., & Goepferd, S. (2000). Infant oral health. Dental Clinics of North America, 44(3), 487- 505.

"The magnitude of [fluoridation's beneficial] effect is not large in absolute terms, is often not statistically significant and may not be of clinical significance." [957]

The United States Public Health Service have stated[958] that "(s)*egments of the population are unusually susceptible to the toxic effects of fluoride*". They include "*postmenopausal women and elderly men, pregnant woman and their foetuses, people with deficiencies of calcium, magnesium and/or Vitamin C, and people with cardiovascular and kidney problems.*"

In regard to the risks of fluoridation it is worth reporting the quote attributable to Dr. Simon Beisler, Chief of Urology, Roosevelt Hospital and Past President of the American Urological Association who stated that "*it is now clear that fluoride is a potentially harmful substance when present in the drinking water in any amount*".

Finally Dr. Arvid Carlsson[959], Pharmacologist and Nobel Laureate in Medicine (2000) stated in a recorded interview in 2005 that the practice of fluoridation "*is against all principles of modern pharmacology. It's really obsolete. No doubt about that…those nations that are using it should feel ashamed of themselves. It's against science*". [960]

Dr. Carlsson also observed that while "*fluorine has a protecting action against caries, but this is a local effect... If you drink it, you are running the risk of all kinds of toxic actions. And, of course, there are such actions. This is something you shouldn't expose citizens to. I would advise against fluoridation.. Side-effects cannot be excluded .. In Sweden, the emphasis nowadays is to keep the environment as clean as possible with regard to pharmacologically active and, thus, potentially toxic substances.*"

It is worth noting, perhaps, the results of two recent studies on dental healthcare on a community after discontinuation of water fluoridation. The city of Kuopio in

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[957] Locker, D. (1999). Benefits and Risks of Water Fluoridation. An Update of the 1996 Federal-Provincial Sub-committee Report. Prepared for Ontario Ministry of Health and Long Term Care.

[958] United States Public Health Service Report (ATSDR TP-91/17, pg. 112, Sec.2.7, April 1993)

[959] Arvid Carlsson, M.D., Ph.D. is a co-recipient of the 2000 Nobel Prize for Medicine, Dr. Carlsson is Professor Emeritus at the University of Gothenburg, and is a member of the Swedish Academy of Sciences and a foreign affiliate of the U.S. National Academy of Sciences. Dr. Carlsson has authored several hundred articles, which have helped to form the basis of modern neuropsychopharmacology. In 1975, he was elected as a Foreign Corresponding Fellow of The American College of Neuropsychopharmacology. In addition to the Nobel Prize, he has been the recipient of The Japan Prize in Psychology and Psychiatry, The Research Prize of the Lundbeck Foundation (Denmark) and the Lieber Prize (USA) for research in schizophrenia. He was also the recipient of the Legion of Honour (France). Dr. Carlsson?s memberships include Member of the Academia Europaea, Member of the Royal Swedish Academy of Sciences, Honorary Fellow of the World Federation of Societies of Biological Psychiatry, Honorary Foreign Associate of the Institute of Medicine, National Academy of Sciences, U.S.A. and Honorary Member of the German Society of Biological Psychiatry. Dr. Carlsson received his M.D. and Ph.D. in Pharmacology from the University of Lund, Sweden.

[960] http://www.fluoridealert.org/videos/carlsson1a.mov

central Finland had fluoridated piped water for 33 years, beginning in 1959. Due to strong opposition by various civic groups, water fluoridation was stopped at the end of 1992. Despite discontinuation of water fluoridation, the first study[961] by the Institute of Dentistry concluded that no increase of caries frequency in primary teeth was observed in Kuopio within a three-year period following discontinuation of water fluoridation.

The aim of the second study[962] was to further observe the occurrence and distribution of caries in Kuopio and Jyväskylä, which was used as the reference town for Kuopio. The total numbers of subjects examined were 688, 1,484 and 1,530 in 1992, 1995 and 1998, respectively. Calibrated dentists registered caries clinically and radiographically. The study found no indication of increasing caries could be found in the previously fluoridated town during 1992–1998. The report concluded that the fact that no increase in caries was found in Kuopio despite discontinuation of water fluoridation and a decrease in preventative procedures suggests that not all of these measures (water fluoridation) were necessary for each child.

Finally a recent study[963] in the USA examining bottled water consumption (with low-fluoride content) and dental health observed that while bottled water users had significantly lower fluoride intakes, especially fluoride from water, there were no significant differences found in either permanent tooth caries or primary second molar caries.

The recent 1986–1987 U.S. National Institute of Dental Research examination[964] by J Yiamouyiannis of the results of over 39,000 schoolchildren, ranging from 5 to 17 years of age in 84 communities with fluoridation, partial fluoridation, or no fluoridation revealed only minor, insignificant differences in decayed, missing, and filled permanent teeth. Yiamouyiannis showed that there were no significant differences in decay rates of permanent teeth or the percentages of decay-free children in fluoridated, partially fluoridated or non-fluoridated areas. The "official" report by JA Brunelle and JP Carlos of the NIDR showed a "benefit" of 17.7% (DMFS). This represents a difference of less than one tooth surface.

In communities where fluoridation has been discontinued, as in Finland, former Eastern block countries, Canada, Cuba, Holland, and the U.S., tooth decay rates have not increased but continued to decrease.[965],[966] Improved nutrition

[961] Caries in the primary dentition, after discontinuation of water fluoridation, among children receiving comprehensive dental care. Seppä L, Kärkkäinen S, Hausen H.Institute of Dentistry, University of Oulu, Finland.
[962] Caries Trends 1992–1998 in Two Low–Fluoride Finnish Towns Formerly with and without Fluoridation, L. Seppä, S. Kärkkäinen, H. Hausen, Institute of Dentistry, University of Oulu, Finland
[963] Broffitt B, Levy SM, Warren JJ, Cavanaugh JE. An investigation of bottled water use and caries in the mixed dentition. Department of Preventive and Community Dentistry, College of Dentistry, University of Iowa, J Public Health Dent. 2007 Summer;67(3):151-8.
[964] Yiamouyiannis J. Water fluoridation and tooth decay: results from the 1986-1987 national survey of U.S. schoolchildren. Fluoride 1990;23:55-67
[965] Ziegelbecker R. Fluoridation in Europe [letter]. Fluoride 1998;31:171-4.
[966] World Health Organization Data (2004) Tooth Decay Trends (12yr olds) in fluoridated and Un fluoridated Countries.

and better dental care appear more likely to account for decreasing caries rates than water fluoridation.

The WHO and UNICEF report on feeding and nutrition of infants noted that while there appears to be general consensus that an optimal fluoride intake should be secured through either water fluoridation, fluoride supplements or the use of fluoridated toothpaste, this recommendation is based on one or either of the above intake pathways. That is if you such fluoridated toothpaste you do not need fluoridated water. It is accepted that in countries where public dental awareness is high and alternative vehicles for fluoride such as fluoridated toothpaste are widely available *and* widely used, public authorities do not need to fluoridate drinking water.[967]

This contrasts with Ireland where fluoride intake is from **BOTH** water fluoridation and the use of fluoridated toothpaste as well as by other dietary means. Fluoridated toothpaste was introduced into Ireland in the late 1960's and is now present in over 95% of available toothpastes sold in Ireland.

It is absolutely certain that the pursuit of a policy of fluoridating drinking water puts consumers in Ireland at a higher risk of over-exposure to fluoride resulting in an alarming increase in dental fluorosis amongst the population. The implications for general health of chronic exposure to fluoride have not been examined in Ireland thought it is likely to result in significant health burdens. It is likely that fluoridating water may act as a tipping point for wider health complications amongst the overall population.

[967] Nutrients in Drinking Water, Water, Sanitation and Health Protection and the Human Environment World Health Organization, Geneva, 2005.

10.0 LEGAL FRAMEWORK

The concept of rule of law forms a cornerstone of the European Union as do the development of human rights and democratization activities. This chapter reflects the diversity of various laws and international treaties in regard to the legal and ethical basis for water fluoridation. It not only describes the formal legal frameworks, but also attempts to explain where the violations in law may occur.

Law is distinguished from policy by the fact that laws are standard rules and regulations that are compulsory. Policies are objectives that a government sets for itself. The policy of water fluoridation is enacted in national legislation under the Health (Fluoridation of Water Supplies) Act, 1960, which was established as a public health measure in the field of preventative dentistry that legislates for fluoride or fluoride products to be added to waters to prevent disease in human beings. Only one other country in the world (Singapore) has a legislative act for the implementation of water fluoridation of public water supplies.

The provisions of the 1960 Act were brought into force in 1965 by a series of Statutory Instruments containing Ministerial Regulations. This 1960 Act is the primary legislation governing the fluoridation of public drinking water supplies in Ireland. Water fluoridation was introduced in Dublin in 1964 and in Cork in 1965. Over the next ten years the majority of the remainder of domestic water supplies were fluoridated. The provisions of the 1960 Act are still in force. In contrast to this Act, almost every other piece of active water or environmental legislation with legal status was introduced following Ireland's entry into the European Economic Community in 1973. There is no EU legislative provision allowing for the fluoridation of drinking water supplies. Ireland remains the only country within the EU with legislative provisions for the mandatory fluoridation of drinking water.

The most recent scientific review undertaken for the European Commission Director General for Health and Consumers by the European Commission's Scientific Committee on Health and Environmental Risks (SCHER) of the hazards and health effects of fluoridating agents and fluoride could not prove without sufficient doubt that fluoridation of water supplies was beneficial and noted that while the scientific evidence for the protective effect of topical fluoride (toothpaste) application is strong, the respective data for systemic application via drinking water (or water fluoridation) is less convincing. The EU review supported previous observations by the WHO, that for countries where topical fluoridated toothpaste was widely available (as is the case in Ireland) and in geographic locations where the risk of dental fluorosis was high, water fluoridation was unnecessary and posed additional health risks.

Fluoridated toothpaste first came on the market in the Republic of Ireland in 1971 and fluoride is now present in over 95% of toothpastes sold in Ireland. The typical fluoride concentrations ranges from 1000-1500ppm. In recent years there has been a significant increase in the incidence of dental fluorosis amonsgt the population of Ireland indicating over-exposure of the population to fluoride. It is accepted that fluoridated drinking water is a major contributor of fluoride, particularly for infants. The recent SCHER review concluded that infants under one year of age, bottle-fed with formula milk reconstituted from fluoridated water ingest multiples of the daily

maximum recommended tolerable intake for fluoride, with consequences for infants in later life. In this report the policy of water fluoridation and its impact on the environment and public health have been extensively reviewed. The implications of pursuing such a policy have been examined in light of more recent advances in environmental, food and health law. The potential impact of water fluoridation on compliance with EU and national regulatory legislation is examined as well its implications for compliance with international law and treaties. It is found that the policy of water fluoridation contravenes a total of thirty two European environmental laws and international treaties as well as six EU Environmental Action plans.

The Government of Ireland represents the only state in Europe that continues to accept the mandatory fluoridation of public water supplies in flagrant breach of European laws, regulations, treaties and policies including:

1. The Charter For Fundamental Rights Of The European Union
2. European Council Water Framework Directive
3. European Council Dangerous Substances Directive (80/68/EEC).
4. European Council Groundwater Directive (2006/118/EC)
5. European Council Drinking Water Directive (98/83/EEC)
6. European Council Urban Wastewater Treatment Directive(91/271/EEC)
7. European Council Fresh Water Fish Directive (2006/44/EEC)
8. European Council Habitats Directive (92/43/EEC)
9. European Council Impact Assessment Directive (85/337/EEC)
10. European Council Marine Strategy Framework Directive 2008/56/EC
11. European Council Common Fisheries Policy (2371/2002).
12. European Council Habitats Directive (92/43/EEC)
13. European Council Dangerous Substances Directive (2006/11/EC)
14. European Council Environmental Liability Directive (2004/35/CE)
15. Urban Waste Water Treatment Regulations, S.I. No. 254/2001
16. Water Quality (Dangerous Substances) Regulations (S.I. No. 12 /2001)
17. Local Government (Water Pollution) Regulations (SI No.271/1992)
18. Fisheries (Consolidation) Act, 1959
19. European Council Food Hygiene Legislation (178/2002)
20. European Council Foodstuffs Regulation (EC) 852/2004
21. European Council Infant Formulae Regulations S.I. No. 243/1998
22. European Council Medicinal Products Directive 2004/27/EC
23. European Council Cosmetic Products Directive 76/768/EEC,
24. Treaty of Europe
25. Treaty on the Functioning Of the European Union
26. The Maastricht Treaty Of EU
27. The Treaty Of Lisbon
28. Council of Europe Convention for the Protection of Human Rights and Fundamental Freedoms
29. Council of Europe Convention for the Protection of Human Rights and Biomedicine1997
30. Council of Europe Convention on the Protection of the Environment through Criminal Law
31. The Rio Declaration on Environment and Development
32. United Nations Convention On Biological Diversity
33. European Community Environmental Policies 1973-2112

In the context of existing EU and national regulatory legislation it has been found that the policy of water fluoridation, as practised, contravenes thirteen EU Directives, three European Food Regulations, four Statutory Regulatory Instruments, one EU Medical Directive, One EU Product Directive, seven International Treaties, three European Conventions and six European action policies; totaling thirty eight individual legislative acts of law or policy.

It is beyond any reasonable doubt that a water policy involving fluoridation of drinking water supplies could never have been enacted in Ireland after it joined the European Union, as it would have been in violation of the very Treaty of European Union itself. Similarly, it is beyond any doubt that such a policy should remain in force given its continued breach of EU Law and its implications for public safety and the environment.

In the area of good governance and environmental stewardship, the Government of Ireland must adhere to their commitments stipulated by the Charter of Fundamental Rights of the European Union, European Environmental Directives, Council Decisions, international treaties and European policy decisions.

Environmental decision-making has to be done in the interest – and thus with the participation – of the general public. This is particularly so when a Government mandates legislation on what people must consume regardless of their human rights and fundamental freedoms or their health or development status as consumers or care providers.

The 'precautionary principle' forms the cornerstone of European and International law. This report clearly demonstrates how in the past the Government of Ireland has consistently violated this principle by pursing a dangerous and untested policy of injecting known enzymatic poisons into the public drinking water supply, without adequate due diligence or risk assessment.

It is for this reason that the author of this report has submitted this document to the current Government in the knowledge that in the year 2000 Fine Gael, as an oppositional political party in the Oireachtas, called for a ban on water fluoridation.

10.1 The Charter for Fundamental Rights of the European Union

The Charter for Fundamental Rights mandates that European citizens have a right to a high level of environmental protection and that the improvement of the environment must be integrated into the policies of the Union. The Lisbon Treaty made this Charter legally binding on EU institutions and on the member states when they are implementing EU law. Enjoyment of these rights entails responsibilities and duties with regard to member states protecting their communities and future generations.

Article 3 specifies that all citizens have the right to respect for his or her physical and mental integrity and in the fields of medicine and biology, the free and informed consent of the person concerned, must be obtained according to the procedures laid down by law.

> Fluoridation of public drinking water exposes the consumer to known health risks that threatens their physical and mental wellbeing. The legislation does not allow for informed consent.

Article 6 secures the right to liberty and security for every person.

> It is clear that the injection of fluoride and silicafluoride compounds into water poses significant public health risks and certain individuals such as infants and diabetes have been identified as a high risk group to its toxicological impacts.

The Charter particularly targets the rights of children to protection and care as is necessary for their well-being in *Article 24* and states that "in all actions relating to children, whether taken by public authorities or private institutions, the child's best interests must be a primary consideration".

> It is a fact that infants who are bottle-fed using formula constituted with fluoridated water exceed the recommended daily tolerable intake for fluoride by orders of magnitude above safe limits.

The requirement of member states to ensure a high level of human health protection is mandated in *Article 35*.

> Fluoridation of public drinking water exposes the most vulnerable in society to known health risks that threatens their physical and mental wellbeing.

The requirement of a member sstate to ensure a high level of environmental protection and the improvement of the quality of the environment is mandated in *Article 37*. Environmental protection and improvement in the quality of the environment must be integrated into policies and ensured in accordance with the principle of sustainable development.

Fluoridation of public drinking water results in continuous discharges of a known environmental toxin into the environment at concentrations that are known to be harmful to ecosystems. The consequence of this is an accumulation of the toxin in the environment representing an upward contaminant trend within the environment. Furthermore, the policy of water fluoridation is not sustainable as 99.5% of the compound used for the original purpose of reducing dental decay is not used for its intended purpose and is discharged into the environment.

The requirement of a member state to ensure a high level of consumer protection is mandated under *Article 38*.

It is clear that the policy of water fluoridation provides no protection to consumers in reducing their daily intake of the toxin fluoride. In fact the policy places consumers at a high risk of a wide variety of serious health threats and diseases as a consequence of fluoride over-exposure. The *EU Scientific Panel on Dietetic Products, Nutrition and Allergies* has found that fluoride is not not essential for human growth and development.· *The European Scientific Committee on Health and Environmental Risks* could not find any scientific study to support the continued policy of water fluoridation.

Ireland remains the only country within Europe that supports such a policy without undertaking any risk assessment of the potential health, social or economic costs to consumers. It is documented that up to 40% of the population of Ireland may suffer from chronic fluoride over-exposure. It is clear that the Government of Ireland, in supporting the mandatory fluoridation of public drinking water supplies, is in breach of *Articles 3,6, 24, 28 35 and 37of* the Charter for Fundamental Rights of the European union. It is important to note that Ireland is the only state in the European Union that implements such a policy.

10.2 European Council Directive on the Protection of Groundwater against Pollution Caused By Certain Dangerous Substances (80/68/EEC).

Fluoride is a List ii substance under the Council Directive 80/68/EEC[968]. Council Directive 80/68/EEC aims to prevent the discharge of certain toxic, persistent and bio-accumulable substances into groundwater. There are two lists of dangerous substances within this directive that are drawn up for the protection of groundwater.

Article 3 of the Groundwater Directive requires member states to take the necessary steps to ensure the effective protection of groundwater in the community. It is necessary to prevent the discharge of List i substances and limit the discharge of substances in List ii so as to avoid pollution of groundwater by certain substances including fluoride.

All indirect discharges of substances in List i and all direct or indirect discharges of substances in List ii are subject to prior authorisation. Such authorisation is granted after an investigation into the receiving environment for a limited period and subject to regular review. Certain conditions have to be met for discharges. If they have not been or cannot be met, the authorisation is withdrawn or refused.

It is clear that by continuing with the policy of water fluoridation the State cannot comply with the conditions of the Groundwater Directive.

> Monitoring of compliance with this Directive and of the effects of discharges on groundwater is the responsibility of the competent authorities of the member States. Monitoring the effects or environmental impact of fluoride discharges into the environment from leaking infrastructure has not been undertaken by the Irish State to date. The EPA has acknowledged[969] that fluoride pollution from leakage in drinking water infrastructure is contaminating groundwater and poses a significant source of fluoride pollution in the environment.

It is clear that the Government of Ireland cannot comply with *Article 3* of this Directive by continuing with the policy of water fluoridation.

[968] Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances.
[969] Clenaghan, C,. O'Neill N, Page, D., Dangerous Substances Regulations National Implementation Report, 2005 Under the Water Quality (Dangerous Substances) Regulations, 2001 (S.I. No. 12 of 2001), Environmental Protection Agency, 2006.

Fluoridated water distributed through the water infrastructure in Ireland is an expensive, unscientific and inefficient mechanism that results in unnecessary health risks for the public as well as pollution of soil, groundwater and surface waters by the uncontrolled release of fluoride into the environment.

Fluoride is not only discharged through leaking water distribution systems into soil and groundwater but from wastewater treatment plants into rivers. It is bioaccumulated in foods grown on soil used for sewage or processed sludge disposal and incorporated into foods and beverages made in areas where water is fluoridated. Food cooked in water with added fluorides will also have increased fluoride levels[970].

It is clear that water fluoridation undertaken using the existing water infrastructure cannot comply with *Article 5* of this Directive. *Article 5* requires that a member state take all appropriate measures to limit all indirect discharge of substances in List ii due to activities on or in the ground.

The only appropriate measure to control the discharge of List ii substances is to limit them at source. This requires termination of the policy of water fluoridation. In the absence of such action, the only other appropriate measure would require the Government of Ireland to immediately replace the tens of thousands of kilometres of leaking infrastructure throughout the State and take measure to prevent such leakages into the future. Until such time as this occurs, toxic persistent and bio-accumulative substances will continue to pollute groundwater.

10.3 European Council Directive on Pollution Caused by Certain Dangerous Substances Discharged into the Aquatic Environment (2006/11/EC)

In order to ensure effective protection of the aquatic environment of the Community, this Directive establish a first list, called List i, of certain individual substances selected mainly on the basis of their toxicity, persistence and bioaccumulation and a second list, called List ii, containing substances which have a deleterious effect on the aquatic environment.

Pollution through the discharge of the various dangerous substances within List i must be eliminated. It is necessary to reduce water pollution caused by the substances within List ii. Any discharge of these substances should be subject to prior authorization which specifies emission standards.

Fluoride is a List ii substance under Council Directive 2006/11/EC. It should be noted that sodium fluoride was found to be an equivocal carcinogen by the

[970] Toxicological profile for fluorides, hydrogen fluoride, and fluorine, Agency for Toxic Substances and Disease Registry U.S. Public Health Service, April 1993

U.S. National Cancer Institute Toxicology Program[971]. It is accepted, alarmingly, that no toxicological or carcinogenesis studies have been undertaken on hexafluorosilicic acid and hexafluorosilicictes compounds used for fluoridating drinking water in Ireland.

It is acknowledged that epidemiological studies and case reports of humans exposed to fluoride or fluoride compounds in drinking water, or occupationally, have demonstrated that exposure to fluoride and inorganic fluoride compounds increases the risk of cancer. It is accepted that fluoride and arsenic are classified as a List ii substance in Directive 2006/11EC. The U.S. National Cancer Institute Toxicology Programme have classified arsenic and inorganic arsenic compounds as *known to be human carcinogens* based on sufficient evidence of carcinogenicity in humans. The classification of arsenic as a carcinogen must reclassify it as List i substance in accordance with the terms of this Directive.

Similarly fluoride or inorganic silicafluoride compounds may in the future be regarded as a List i substance based on the substantial international evidence as documented elsewhere in this report.

The Government of Ireland cannot comply with the terms of this Directive by continuing to facilitate the fluoridation of drinking water with a substance that may be classified as a List i substance. To continue to support this policy would breach European Law.

Where fluoride or inorganic fluoride compounds used in water fluoridation are regarded as List ii substances the Government of Ireland has a legal obligation to reduce water pollution caused by these substances.

All discharges of effluents into the environment containing fluoridated water require prior authorisation under the terms of this Directive. The Government of Ireland is in clear breach of the terms of this Directive by continuing to support the policy of water fluoridation and cannot in any manner comply with this Directive by continuing with a policy of water fluoridation.

[971] NTP (National Toxicology Program) (1990). Technical Report on the toxicology and carcinogenesis studies of sodium fluoride in F344/N rats and B6C3F1 mice (Drinking water studies), Technical Report Series No 393

10.4 European Council Groundwater Directive 2006/118/EC

This new directive establishes a regime which sets underground water quality standards and introduces measures to prevent or limit inputs of pollutants into groundwater.

The Directive establishes quality criteria that takes into account local characteristics and allows for further improvements to be made based on monitoring data and new scientific knowledge. The Directive thus represents a proportionate and scientifically sound response to the requirements of the Water Framework Directive (WFD) as it relates to assessments on chemical status of groundwater and the identification and reversal of significant and sustained upward trends in pollutant concentrations. Member states are required to establish standards at the most appropriate level and take into account local or regional conditions.

In accordance with the requirement of the Water Framework Directive, the EPA commissioned a study to establish the Natural Background Level (NBL) concentration for a range of parameters including fluoride. Median fluoride natural background Level in groundwater was estimated to be 0.1mg/l with a 90th percentile upper level of 0.21mg/l.

The EPA has also reported that leaking drinking water distribution mains are a significant potential source of fluoride particularly in urban areas. [972] Artificially fluoridated drinking water contains fluoride at levels of between 8 and up to 1.50mg/l.[973] This has resulted in emissions of fluoride into groundwater at concentrations in excess of seven times the natural background level.

It is uncertain, as to why the EPA have not set threshold values for fluoride, given its toxicity, ability to bioaccumulate in the environment, its reported impact on groundwater quality, surface water ecology and increasing concentrations in the environment as a consequence of fluoridation of drinking water supplies, as examined in this report.

Evidence from Camargo[974] demonstrated that net-spinning caddisfly larvae are harmed at fluoride levels as low as 0.2mg/L and how fluoride is an endocrine disruptor in the environment[975]. It has been demonstrated that freshwater animals are, in general, more sensitive to fluoride toxicity than freshwater algae and macrophytes and among freshwater animals, net-spinning caddisfly larvae and upstream-migrating adult salmons seem to be

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[972] Clenaghan, C,. O'Neill N, Page, D., Dangerous Substances Regulations National Implementation Report, 2005 / Water Quality (Dangerous Substances) Regulations, 2001 (S.I. No. 12 of 2001), Environmental Protection Agency, 2006.

[973] Hayes N,. Page D,. Sweeney L,. O'Leary G,. The Provision and Quality of Drinking Water in Ireland A Report for the Year 2010, Environmental Protection Agency, 2011.

[974] Camargo, J.A., Garc_ıa de Jal_on, D., Mu~noz, M.J., Tarazona, J.V., 1992b. Sublethal effects of sodium fluoride (NaF) on net-spinning caddisflies (Trichoptera). Aquatic Insects 14, 23–30.

[975] Camargo JA. Fluoride toxicity to aquatic organisms: a review. Chemosphere. 2003 Jan;50(3):251-64

the most sensitive[976],[977],[978]. In consequence, safe levels of fluoride for aquatic life must be primarily based on the tolerance of net-spinning caddisfly larvae and upstream-migrating adult salmons. Because a fluoride concentration as low as 0.5 mg F_/l can adversely affect net-spinning caddisfly larvae and upstream-migrating adult salmons inhabiting soft waters with low ionic content, safe levels below this fluoride concentration are recommended in order to protect these freshwater animals from fluoride pollution[979].

10.5 European Council Directive on the Conservation of Natural Habitats and of Wild Fauna and Flora (92/43/EEC)

The main aim of this Directive is to promote the maintenance of biodiversity within the general principles of supporting sustainable development.

Under *Article* 6 of 92/43/EEC member states are required to take appropriate steps to avoid, in special areas of conservation, the deterioration of natural habitats and the habitats of species as well as disturbance of the species for which the areas have been designated.

Fluoride emissions from wastewater treatment facilities pose a significant threat to freshwater ecosystems in particular freshwater salmon, freshwater trout and freshwater pearl as well as aquatic invertebrates such as caddisfly.

The policy of water fluoridation violates the provisions of Directive 92/43/EEC by allowing a known toxin to be emitted into surface and groundwaters at concentrations that are harmful to protected fisheries.

It is known that fluoride levels less than 0.5ppm are harmful to freshwater salmon and concentrations less than this are known to be harmful to caddisfly, the principle food supply for salmon.

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[976] Camargo, J.A., La Point, T.W., 1995. Fluoride toxicity to aquatic life: a proposal of safe concentrations for five species of Palearctic freshwater invertebrates. Arch. Environ. Contam. Toxicol. 29, 159–163.
[977] Camargo, J.A., 1996b. Estimating safe concentrations of fluoride for three species of Nearctic freshwater invertebrates: multifactor probit analysis. Bull. Environ. Contam. Toxicol. 56, 643–648
[978] Damkaer, D.M., Dey, D.B., 1989. Evidence for fluoride effects on salmon passage at John Day dam, Columbia river, 1982– 1986. N. Am. J. Fish. Manag. 9, 154–162.
[979] Camargo, J, A., Fluoride toxicity to aquatic organisms: A Review. Chemosphere 50 (2003) 251–264

10.6 European Council Water Framework Directive (S.I No. 722/2003 EU Regulations 2003)

The purpose of the Water Framework Directive (WFD) 2000/60/EC is to establish a framework for the protection of inland surface waters, transitional waters, coastal waters and groundwater. The Water Framework Directive deals with both the sustainable use of water resources and conservation and enhancement of freshwater ecosystem biodiversity.

The WFD was transposed in Ireland by S.I. No. 722 of 2003. The aim of the WFD is to prevent further deterioration and to protect and enhance the status of aquatic ecosystems and, with regard to their water needs, terrestrial ecosystems and wetlands directly depending on the aquatic ecosystems. The Directive promotes sustainable water-use based on the long-term protection of available water resources and aims at enhanced protection and improvement of the aquatic environment, inter alia, through specific measures for the progressive reduction of discharges, emissions and losses of priority substances and the cessation or phasing-out of discharges, emissions and losses of the priority hazardous substances.

The WFD ensures the progressive reduction of pollution of groundwater and prevents its further pollution.

The State cannot comply with the terms of the Water Framework Directive with uncontrolled discharges of treated fluoridated drinking water into the environment. The policy of water fluoridation furthermore does not promote sustainable water-use or long-term protection of aquatic ecosystems and results in increases in discharges of list ii substances into the environment.

The Government of Ireland has a requirement in deriving environmental quality standards for pollutants listed in points 1 to 9 of Annex VIII of the directive for the protection of aquatic biota. Standards may be set for water, sediment or biota using *both acute and chronic data. No such data exists for the fluoride product used in water fluoridation in Ireland.*

The WFD also requires that member states shall set appropriate safety factors consistent with the guidance given in Section 3.3.1 of Part II of the *"Technical guidance document in support of Commission Directive 93/67/EEC on risk assessment for new notified substances and Commission Regulation (EC) No 1488/94 on risk assessment for existing substances*". The WFD requires that the developed standard should be compared with any evidence from field studies. Where anomalies appear, the derivation shall be reviewed to allow a more precise safety factor to be calculated. No risk assessment or safety factors based on scientific study have been developed by the state for water fluoridation products.

The SCHER review[980] clearly noted that the toxicology of hexafluorosilicic acid & hexafluorosilicictes compounds is incompletely investigated and that additional epidemiology, toxicology, clinical medicine and environmental exposure assessments need to be undertaken in order to fill data gaps in the hazard profile, the health effects and the exposure assessment of fluoride.

In addition, further research was recommended by the NRC[981] and SCHER in assessing the health and environmental risks that may be associated with the use of the most common drinking water fluoridation agents like silicofluorides, taking into account their hazard profiles, their mode of use in water fluoridation, their physio chemical behaviour when diluted in water, and the possible adverse effects they may have in exacerbating fluoride health effects as reported in some scientific studies. In the absence of such measures and the development of such standards the State cannot comply with the WFD. It is noted that in the development of such standards the directive requires that the standard derived shall be subject to peer-review and public consultation. No such public consultation or scientific review has taken place.

In accordance with the requirement of the WFD, the State must design surveillance monitoring programmes to provide information for the assessment of long-term changes in natural conditions and the assessment of long-term changes resulting from widespread anthropogenic activity. No monitoring programme exists for the assessment of anthropogenic discharges of fluoride on long-term natural conditions. It is however, certain that by continuing with the policy of water fluoridation direct and indirect discharges of fluoride to the environment will continue and lead to further increases in fluoride within the natural environment.[982]

ANNEX VIII of the WFD provides an indicative list of the main pollutants including organohalogen compounds and substances which may form such compounds in the aquatic environment. Organophosphorous compounds, organotin compounds and substances and preparations, or the breakdown products of such, which have been proved to possess carcinogenic or mutagenic properties or properties which may affect steroidogenic, thyroid, reproduction or other endocrine-related functions in or via the aquatic environment. It is noted in studies examined by both the NRC and SCHER that fluoride may affect steroidogenic, thyroid, reproductive or other endocrine-related functions in or via the aquatic environment. Given the lack of detailed toxicological or ecological risk assessments to prove otherwise it may in future, subject to further investigation, be classified as a priority hazardous

[980] Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, Scientific Committee on Health and Environmental Risks, Director General for Health & Consumers 2010

[981] U.S National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C.

[982] U.S. EPA Technical Support Document for the Round Two Sewage Sludge Pollutants, Office of Water, EPA-822-R-96-003, August 1996.

substance. It is obvious therefore thta the State should adopt the precautionary approach and that the policy of water fluoridation should cease.

The ultimate aim of this Directive is to achieve the elimination of priority hazardous substances and contribute to achieving concentrations in the marine environment near background values for naturally occurring substances. Most of the fluoride in the marine environment comes from surface water discharges. According to the EPA naturally elevated levels of fluoride are quite rare in Ireland and any exceedances in drinking water standards reported by the Agency are due almost entirely to public water supplies being dosed with fluoride at levels in excess of the legally permitted dose.[983] The Water Framework Directive allows for the principle of recovery of the costs of water services, including environmental and resource costs associated with damage or negative impact on the aquatic environment. The Directive requires that these should be taken into account in accordance with, in particular, the polluter-pays principle.

It would seem logical that where households with sensitive sub-groups incur costs to remove fluoride from their water supply, that these costs could and should be passed onto the State placing a further burden on the Exchequer. There are an estimated 400,000 individuals in Ireland that may have renal complications due to diabetes, in addition over 70,000 households may have an infant under 6 months, and in both cases households require access to non-fluoridated water in accordance with WHO recommendations.[984] It is clear that the cost of treating fluoridated water to meet acceptable standards has not been calculated nor has the potential cost to the aquatic environment or public health been investigated. Some of the direct costs to public health of fluoridation can be determined and may be estimated to be in the tens of millions due to the massive increase in fluorosis alone requiring future dental treatment and long-term care.

The WFD states "*with regard to pollution prevention and control, community water policy should be based on a combined approach using control of pollution at source*". It is clear that this can only be achieved by not adding fluoridating agents to water, the only way this can be controlled is by not adding fluoridation products to water. The WFD further notes that in identifying priority hazardous substances, account "*should be taken of the precautionary principle, relying in particular on the determination of any potentially adverse effects of the product and on a scientific assessment of the risk*".

983 The Provision and Quality of Drinking Water in Ireland, a Report for the Year 2010, Environmental Protection Agency.

984 Nutrients in Drinking Water, Water, Sanitation and Health Protection and the Human Environment World Health Organization, Geneva, 2005.

It is evident that the State has not adhered to the precautionary principle and is in violation of EU law. It is certain that the State has not determined the potential adverse effects of fluoridation products or their derivative compounds on human health. It is clear also that the State has not undertaken any adequate scientific assessment of the environmental impact of fluoridation on natural ecosystems or biodiversity protection. While the WFD provides that a member state should adopt measures to eliminate pollution of surface water by priority substances and progressively reduce pollution by other substances, the continuing policy of water fluoridation prevents Ireland from achieving these or other objectives noted in the Directive. The only manner in which the Government of Ireland can comply with the terms of the WFD is by fully implementing and enforcing existing environmental legislation for the protection of waters. This requires an end to the policy of anthropogenic emissions of fluoride from wastewater treatment facilities.

10.7 European Council Directive on the Quality of Water Intended for Human Consumption (98/83/EEC).

EU Directive (98/83/EEC) concerns the quality of water intended for human consumption. The objective of this Directive is to protect human health from the adverse effects of any contamination of water intended for human consumption by ensuring that it is wholesome and clean.

Fluoride is listed as an undesirable substance in Annex 1 of Directive (98/83/EEC. A maximum admissible concentration (MAC) of 1.5mg/l is provided. Under *Article 2, w*ater intended for human consumption means all water used for that purpose, either in its original state or after treatment, regardless of origin. This includes boiled water used for infant food or other foodstuffs. Consequently it is the concentration of fluoride post treatment[985] (including boiling) that must be monitored.

Astonishingly it was reported[986] by the EU Scientific committee examining the safety of fluoride compounds in oral health care products that fluoride in infant formula became a problem as a consequence of water fluoridation. Yet this information was never circulated or provided by either the Food Safety Authority, the Health Service Executive or the Forum for Fluoridation in Ireland to warm parents or child care providers of the risks.

It is apparent that no comprehensive prepared feed infant product sampling has been undertaken by the health or food safety authorities in Ireland to monitor the risk of chronic fluoride intake by infants. A small scale unpublished study[987] on fluoride dose and children undertaken by *Dublin Dental School and Hospital* concluded, however, that baby formulae should be reconstituted with non-fluoridated water and that parents should be made aware of the additive effects from fluoride sources such as tap water, in an effort to avoid dental fluorosis or potential toxicity.

In countries where water fluoridation is practised, estimating the fluoride intake is particularly problematic as research has documented how variable changes in fluoride concentrations were observed in water boiled in cooking vessels of different surface compositions. Studies[988],[989] have demonstrated

[985] Article 6 (d) Directive 80/788/EEC.

[986] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

[987] To examine the Fluoride content of popular formulae and milk and to study the fluoride dose that infants are receiving from these sources. Dublin Dental School and Hospital 1997-1998.

[988] Effect of Cooking Vessel Composition on Fluoride, C.A. Full and Frederick M. Parkins, Department of Pedodontics, University of Iowa, College of Dentistry, Journal of Dental Research 1975 54: 192, DOI: 10.1177/00220345750540012501

[989] Archives of oral biology Volume 12 issue 1, 1967 pages 61-71,

that the extent of the alterations in fluoride content appeared large enough to alter the quantity of fluoride incorporated into foods prepared in the four types of containers. The magnitude of the composition effects seen with the aluminium and Teflon in contrast to the Pyrex and stainless steel containers suggests that significant alterations in fluoride uptake into boiled foods may occur.

It is a fact that boiling fluoridated water results in concentrating fluoride in water. By fluoridating water the State therefore has no means of protecting the consumer by ensuring that the maximum admissible concentration of 1.5mg/l is not exceeded in treated water. The State subsequently cannot ensure the wholesomeness of any foodstuffs produced in its finished form in Ireland unless the State has the capacity to test the final product individually within the household or within larger food manufacturing facilities. This applies particularly to the making of infant feed or processed foods for infants or sensitive sub-groups of the population, which is the major cause for concern.

Recent scientific evidence[990] suggests that mixing powdered or liquid infant formula concentrate with fluoridated water on a regular basis may increase the chance of a child developing enamel fluorosis. The U.S. EPA reported that adults exposed to excessive consumption of fluoride over a lifetime may have increased likelihood of bone fractures, as well as effects on bone leading to pain and tenderness. For effects to teeth, children are most likely to be affected by excessive exposure to fluoride because it impacts teeth while they are still in formative phases. Children aged 8 years and younger who are exposed to excessive amounts of fluoride have an increased chance of developing pits in the tooth enamel, along with a range of cosmetic effects to teeth.

To comply with *Article 8* of Directive 80/778/EEC, the State is required to take all the necessary measures to ensure that any substances used in the preparation of water for human consumption do not remain in concentrations higher than the maximum admissible concentration and that they do not, either directly or indirectly, constitute a public health hazard.

It is clear that the State cannot comply with the requirement of this directive by continuing with the policy of water fluoridation. It is only by cessation of mandatory water fluoridation of public water supplies that the Government of Ireland can comply with the terms and conditions of Directive 80/778/EEC.

The concentration of fluoride by boiling J.H. Fremlin, J. Mathieson. Department of Physics, The University, Birmingham, 15, England.

[990] U.S EPA. Office of Water (4606M) EPA 815-F-11-001 January 2011

Importantly the directive outlines how the establishment of parametric values, applicable to water intended for human consumption, should be based on public-health considerations and on a method of assessing risk, preventing a potential danger to human health and where such danger may exist the supply of such water should be prohibited or its use restricted.

It has been reported by the NRC and SCHER, and in peer-reviewed scientific journals, that fluoridation of drinking water exposes a significant sector of the population to unnecesssary risk; in particular bottle-fed babies, young boys (pre-adolescent boys who drink fluoridated water are at a seven-fold increased risk of osteosarcoma), patients with renal impairments, individuals with diabetes, individuals at risk of bone fractures or bone development problems, individuals with sensitive gastrointestional systems and individuals who are immunocompromised who could be at greater risk of the immunologic effects of fluoride, individuals with Alzeimer's disease or dementia and children with special healthcare needs who have a developmental, mental, sensory, behavioural, cognitive, or emotional impairment related to neurological development disorders.

The only option for the State to prevent a potential danger to human health is to end the policy of water fluoridation or alternatively to provide alternative supplies of clean low fluoride content water to consumers and individual households.

Directive 80/778/EEC also outlines how the State should take remedial action to restore the quality of the water where that is necessary to protect human health. To comply with this requirement the State would need to provide consumers with reverse osmosis kits or other technology to remove or reduce fluoride to acceptable levels. This would be unreasonably expensive compared to the direct cost of ending water fluoridation which rectifies the problem at source in accordance with Article 130r(2) of the EEC Treaty.[991]

Directive 80/778/EEC requires that consumers should be adequately and appropriately informed of the quality of water intended for human consumption (this includes bottle-fed infants) and that consideration should be given both to the technical and statistical needs of the Commission.

The European Commission's Scientific Commitee on Health and Environmental Risk (SCHER),who has acknowledged limited evidence from epidemiological studies, points towards adverse health effects following systemic fluoride consumption, e.g., carcinogenicity, developmental neurotoxicity and reproductive toxicity but that insufficient evidence (due to a lack of toxicological studies) is availalble to prove this conclusively. The Commitee also state that water fluoridation could induce fluorosis with a very narrow margin of exposure, that there is a risk of dental fluorosis in children in EU countries with systemic fluoride exposure, such as Ireland, and an increased risk of dental and bone fluorosis generally.

[991] Treaty of Amsterdam amending the Treaty of the European Union, the Treaties establishing the European Communities and certain related acts

Consumers in Ireland have not been notified of the findings of either the NRC or SCHER studies, the findings of the York Review were misrepresented in the 2002 Fluoridation Report and to the knowledge of this author the Department of Health have not issued circulars to health professionals or the public informing them of the potential risks of water fluoridation based on current scientific knowledge. While the U.S. EPA and U.S. Public Health Bodies[992] have notified the public of their concerns regarding this risk to public health no such warning has been provided to Irish consumers.

It would further appear that Department officials have incorrectly advisedthe Minister of State at the Department of Health (Deputy Róisín Shortall T.D.) of the findings of the EU Scientific Committee on Health and Environmental Risks (SCHER) report. [993]

The *U.S. Centre for Disease Control and Prevention* and the WHO recommend that it is important to know the fluoride content of your drinking water, particularly if you have children. As noted by the EPA in their most recent review on the quality of drinking water in Ireland there were many incidences of operator error resulting in contamination of sixty two water supplies with inappropriate levels of fluoride. The population was not informed of these incidences when they occurred. Since fluoride has no taste, colour or odour, the only way to determine its concentration is by laboratory analysis. The EPA also noted that a large number of boiled water notices were issued for microbiological contamination requiring the public to boil treated fluoridated water before use. Boiling water increases the concentration of fluoride in the liquid resulting in further exposure of the population to the toxic risks of fluoride.

Knowledge of the fluoride content is critical when recommending fluoride supplementation to avoid fluorosis of the dentition.[994]

In the U.S., the recommended fluoride level in drinking water for good oral health is 0.7 mg/L which is similar to that recommended in Ireland. A minimal risk level of 0.05 mg/kg/day for fluoride has been calculated by the *Agency for Toxic Substances and Disease Registry*. [995]

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[992] New Jersey Water Education Booklet 15. Mandatory fluoridation of public water supplies, State of New Jersey Public Sector Announcement, Do not mix fluoridated water with infant formula

[993] Dáil Éireann Debate *Vol. 746 No. 1*

[994] Fluoride content of some bottled waters and recommendations for fluoride supplementation. Stannard J; Rovero J; Tsamtsouris A; Gavris V The Journal of pedodontics, (1990 Winter) Vol. 14, No. 2, pp. 103-7. Journal code: 7702327. ISSN: 0145-5508. L-ISSN: 0145-5508.

[995] *Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine (F).* Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta 1993.

At the reported concentration of fluoride in drinking water in Ireland the minimum exposure dose for infants and the maximum exposure dose for infants, children and adults in Ireland would be significantly higher than the 0.05 mg/kg/day recommended, which is the minimum risk level (MRL) estimated by the *Agency for Toxic Substances and Disease Registry* (ATSDR).

The measured concentration of fluoride in fluoridated drinking water in Ireland ranges from 0.7-1.5mg/l generally. [996]

When evaluating the total fluoride exposure of the population, the consumption or total fluoride intake of an individual must account for factors such as:

- The quantity of water consumed per day.
- The fluoride intake from other beverages consumed such as tea, coffee or soft drinks.
- The fluoride intake from other sources such as toothpaste ingestion or high fluoride foods.
- Usage of boiled water in meal or drink preparation, baby formula and infant food dilution.

Based on a review of fluoride intake and prevalence of dental fluorosis in September 2000, it was concluded that fluoride intakes of infants and children have shown a steady increase since 1930.[997] It was also found that exposure is likely to continue to increase, and will be associated with further increase in the prevalence of enamel fluorosis unless intervention measures are instituted. The review concluded that "*the most important intervention measure that should be undertaken is to use, when feasible, water low in fluoride for dilution of infant formulas*". [998]

When examining the exposure to fluoride and dietary intake from drinking water it is necessary to calculate the exposure from boiled treated water in addition to tap water. [999] The source of water for bottle fed infants is boiled water (which has a higher concentration of fluoride than tap water) used for preparing milk formula. The same approach should also apply to adults as one of the main sources of consumption of liquids for adults is through drinking hot beverages. In boiled water, fluoride levels increase proportionally to the

[996] The Provision and Quality of Drinking Water in Ireland, A Report for the Year 2010, Environmental Protection Agency.
[997] Fluoride Intake and Prevalence of Dental Fluorosis: Trends in Fluoride Intake with Special Attention to Infants: Samuel J. Fomon MD, Jan Ekstrand DDS, PhD, Ekhard E. Ziegler MD, Journal of Public Health Dentistry Volume 60, Issue 3, pages 131–139, September 2000.
[998] Fluoride Intake and Prevalence of Dental Fluorosis: Trends in Fluoride Intake with Special Attention to Infants: Samuel J. Fomon MD, Jan Ekstrand DDS, PhD, Ekhard E. Ziegler MD, Journal of Public Health Dentistry Volume 60, Issue 3, pages 131–139, September 2000.
[999] Research Report: Assessment Of The Exposure To Fluoride From Drinking Water In Durango, Mexico. Dr Díaz-Barriga et al. Facultad de Medicina, Universidad Autónoma de San Luis Potosí, SLP, Mexico. International Society For Fluoride Research, Fluoride 31 (4) 1998 , pp 183-187

loss of volume, therefore in calculations the concentration of fluoride in tap water should conservatively be doubled.[1000]

In Ireland, the fluoride level has been demonstrated to be in the range of approximately 0.8-1.5 mg/L. Consequently to allow for an adequate safety margin for the purposes of calculating fluoride intake; the concentration of fluoride in tap water is doubled to 1.4 and 3mg/l respectively.

The exposure duration and infant body weight over the exposure period differ over the period of bottle feeding. For that reason the exposure scenarios should also examine the implications of different body weights.[1001]

Exposure doses can be calculated by the generic equation

ED=CxWI/BW

Where ED = exposure dose (mg/kg/day) WI= water intake (L/D)
C= fluoride concentration (mg/L) BW= body weight (KG)

For example, an infant with a body weight of :

- 6kg (first semester of life) would be exposed to a toxic level of fluoride estimated to be between 0.23 and 0.5mg/kg/day based on consuming one litre of liquid a day.
- 9.1kg (an average for babies for 6 to 11 months old) would be exposed to a toxic level of fluoride estimated to be between 0.15 and 0.33mg/kg/day, based on consuming one litre of liquid a day.
- 11.3kg (average for one year old babies) would be exposed to a toxic level of fluoride estimated to be between 0.12 and 0.26mg/kg/day, based on consuming one litre of liquid a day.

The safe dose estimated for this group was between 0.5 mg/kg/day and 0.12 mg/kg/day. At 0.5mg/kg/day this would be in the order of ten times the minimum risk level (MRL) estimated by the *Agency for Toxic Substances and Disease Registry* (ATSDR). At the above levels, a clear and immediate risk for dental fluorosis is evident. These levels would also constitute a clear public health risk. The lower range of exposure represents a doubling of the MRL. These lower levels would also present a clear risk for dental fluorosis. It is alarming that the maximum exposure dose to fluoride for infants living in Ireland, drinking fluoridated water, could be in the order of 10 times the ATSDR's MRL of 0.05 mg/kg/day for chronic oral exposure. The calculation above does not take into account other potential sources of fluoride and therefore the real exposure doses could be higher than the figures presented here. How serious a health risk such a dose represents is a question that deserves immediate action and further research.

[1000] Source: Grimaldo M, Borja V, Ramírez AL, Ponce M, Rosas M, Díaz-Barriga F. Endemic fluorosis in San Luis Potosí, Mexico. I. Identification of risk factors associated with human exposure to fluoride. Environmental Research 68 25-30, 1995.
[1001] National centre for health statistics (1987) (cited in U.S. EPA, 1994B

Fluoride has been subject to a series of acute, short- and long-term studies involving animals but given the limited character of these studies and the large body of data on the toxic effects of fluoride in humans the latter data have priority in the derivation of long-term tolerable intakes for humans.

Skeletal fluorosis consists of adverse changes in bone structure due to continuous deposition of fluoride in the bone. The minimum dose required for production of skeletal fluorosis in its various degrees is not known exactly. [1002] Furthermore there is little or no available data examining carcinogenicity of fluoride products for drinking water[1003]; this is especially the case in Ireland where little if any data on risk assessments is available.

In light of this, and the results outlined above, an urgent revision of the public health programme on water fluoridation is required. It is clear that a source of non-fluoridated potable water has to be made available to the population. This requirement is especially important since preliminary analysis of bottled water sold within the EU found that bottled water can also have high levels of fluoride.

This has resulted in Commission Directive 2003/40/EC to allow operators to comply with this directive and the authorization of a treatment to remove fluoride from natural mineral waters and spring waters by using activated alumina.

A recent survey[1004] of bottled water marketed in Ireland demonstrated that none of the products sampled displayed the fluoride content of the water on the labels. Therefore, the consumer currently cannot determine what the fluoride content in bottled water is as manufacturers do not provide information on fluoride content. The author of this study requested data on fluoride levels in bottled waters on sale in Ireland from the Food Safety Authority but no such data was available from them.

It is reported that the fluoride content of bottled waters can be highly variable with concentrations ranging from very low in distilled waters to over 4ppm in a mineral water. While manufacturers are encouraged to list the nutritional contents of their products, labels stating the fluoride levels of bottled water are not provided unless the mineral water has a concentration of fluoride greater than 1.5mg/L. At the higher range, this may have oral health implications for those individuals, and especially children, who drink

[1002] Opinion On Arsenic, Barium, Fluoride, Boron And Manganese In Natural Mineral Waters (Expressed On 13 December 1996), Scientific committee on Food, European Commission.
[1003] International Journal of Paediatric Dentistry/the British Paedodontic Society [&] the International Association of Dentistry for Children, (1991 Dec) Vol. 1, No. 3, pp. 143-6. Journal code: 9107511. ISSN: 0960-7439. L-ISSN: 0960-7439.
[1004] A consumer survey of spring, mineral or distilled waters, sold in plastic bottles was undertaken in November 2011 by the author of this report.

bottled water as their primary source of drinking water. Due to the high content of fluoride, some of these waters are unacceptable for a child's diet[1005]. As previously noted, despite the inherent public health risks, no data on chemical surveillance for fluoride concentrations on bottled waters either marketed, produced or sold in Ireland are available from the Food Safety Authority (FSA). This is a matter that should be addressed by the Minister for Agriculture and Food and the FSA of Ireland to ensure, in particular, the adequate supply of safe drinking water for the preparation of infant foods where no other supply of low fluoride water is available. Remarkably, the FSA have also found that the effects of formula feeding on infant health have not been investigated in Ireland.[1006]

10.8 European Council Directive Concerning Urban Waste Water Treatment (91/271/EEC)

This directive concerns the collection, treatment and discharge of urban wastewater and the treatment and discharge of wastewater from certain industrial sectors. Its objective is to prevent the environment from being adversely affected by the disposal of insufficiently-treated urban wastewater.

The directive requires member states to monitor treatment plants, receiving waters and the disposal of sludge to ensure that the environment is protected from the adverse effects of the discharge of wastewaters. Under *Article 6* of this directive urban wastewater discharges must be subject to comprehensive studies to demonstrate that discharges will not adversely affect the environment. Under *Article 14* sludge arising from wastewater treatment shall be re-used whenever appropriate. Disposal routes shall minimize the adverse effects on the environment and member states shall ensure that the total amount of toxic, persistent or bio-accumulable materials in sludge is progressively reduced. It is obvious that the policy of water fluoridation results in clear violations of the Urban Wastewater Directive. Over 99.5% of the fluoride compounds added to drinking water are not used for the purpose intended and end up in wastewater treatment systems. Wastewater treatment has been shown to be largely ineffective in removing the pollutant fluoride from wastewaters. This is resulting in significant discharges of a persistent bio-accumulative pollutant into the environment at concentrations that are harmful to freshwater fish and invertebrates.

> The only way to reduce the concentrations of fluoride released into the environment is to end the policy of water fluoridation.

[1006] Food Safety Authority of Ireland, Recommendations for a national infant feeding policy 1999.

10.9 European Council Directive on the Assessment of the Effects of Certain Public and Private Projects on the Environment (85/337/EEC)

The Environmental Action Programmes (EAP) of the European Communities on the environment, approved by the Council of the European Communities and the representatives of the Governments of the member states, stress that the best environmental policy consists in preventing the creation of pollution or nuisances at source, rather than subsequently trying to counteract their effects.

The EAPs also affirm the need to take effects on the environment into account at the earliest possible stage in all the technical planning and decision-making processes.

To that end, provision is made for the implementation of procedures to evaluate environmental effects through this directive.

The directive outlines how the effects of a project on the environment must be assessed in order to take account of concerns to protect human health, to contribute by means of a better environment to the quality of life, ensure maintenance of the diversity of species and to maintain the reproductive capacity of the ecosystem as a basic resource for life.

Facilities such as wastewater treatment facilities have significant effects on the environment, the impact of such infrastructure according to this directive must as a rule be subject to systematic assessment. This must, but has not yet to date in Ireland, include assessment of the impact of anthropogenic fluoride emissions.

It is apparent that the State has not complied with Articles 1 and 2 of this directive by not examining the impact of anthropogenic fluoride emissions from wastewater treatment facilities resulting from artificial fluoridation of drinking water on either the freshwater or marine environment.

10.10 European Council Directive on Environmental Liability with Regard to the Prevention and Remedying of Environmental Damage (2004/35/CE)

The Environmental Liability Directive (ELD) came into force in Ireland in 2009. The ELD is about preventing and remedying environmental damage. It aims to hold operators whose activities have caused environmental damage financially liable for remedying this damage, and it aims to hold those whose activities have caused an imminent threat of environmental damage liable for taking preventive actions.

The main objectives of the ELD include the application of the "polluter pays" principle; this directive establishes a common framework for liability with a view to preventing and remedying damage to animals, plants, natural habitats and water resources, and damage affecting the land.

The liability scheme applies to certain specified occupational activities and to other activities in cases where the operator is at fault or negligent. The public authorities are also responsible for ensuring that the operators responsible take or finance the necessary preventative or remedial measures themselves.

The prevention and remedying of environmental damage should be implemented through the furtherance of the "polluter pays" principle, as indicated in the Treaty and in line with the principle of sustainable development. The fundamental principle of this directive should therefore be that an operator, whose activity has caused the environmental damage, or the imminent threat of such damage, is to be held financially liable.

This directive should apply, as far as environmental damage is concerned, to occupational activities which present a risk for human health or the environment. This directive should also apply, as regards damage to protected species and natural habitats, the operator should only be liable under this directive whenever he is at fault or negligent.

According to the "polluter-pays" principle, an operator causing environmental damage or creating an imminent threat of such damage should, in principle, bear the cost of the necessary preventative or remedial measures.

Persons adversely affected or likely to be adversely affected by environmental damage should be entitled to ask the competent authority to take action. This includes non-governmental organisations promoting environmental protection or angling clubs or organisations. Such organisations must be given the opportunity to properly contribute to the effective implementation of this directive.

It is clear that through the policy of water fluoridation that the State is contributing to water damage as defined by this directive. The Liability Directive defines this as any damage that significantly adversely affects the

ecological, chemical and/or quantitative status and/or ecological potential of waters.

It is clear that through the policy of water fluoridation the State is contributing to environmental damage as defined by this directive. The Liability Directive defines environmental damage as damage to protected species and natural habitats including the favourable conservation status of protected habitats or species. In accordance with the requirement of this Directive the significance of any such effects are to be assessed with reference to the baseline condition.

It is clear that the State has not examined the impact of anthropogenic fluoride emission on the aquatic environment particularly of protected species such as freshwater salmon. It is known that concentrations of less than 0.5mg F/L are harmful to salmon and that concentrations of 0.25mg F/L are harmful to the caddisfly. The implication of fluoridation of water on the health and sustainability of inland fisheries has never been examined. The ecological importance of soft waters is a key requirement for salmonid fisheries worldwide. It is known that the effects of fluoride or inorganic fluoride compounds are more harmful in low calcium waters. It is plausible that water fluoridation and emissions of fluoride from wastewater treatment plants have been a major contributory factor to the decline of freshwater salmon stocks in Ireland. It is noteworthy that the timeline for the decline in stocks coincides with the commencement of fluoridation.

This Liability Directive applies to damage caused by an emission, event or incident resulting in discharges into inland surface water and pollution caused by certain dangerous substances, discharged into the aquatic environment of the Community. While EU law requires that all discharge or injection of pollutants into surface water or groundwater which require a permit, authorisation or registration in pursuance of Directive 2000/60/EC, it is clear that the Government of Ireland has not applied the legal requirement of EU Directives to fluoridation of water. All public water supplies are fluoridated in Ireland regardless of end use. Fluoride and inorganic fluoride compounds are discharged in significant quantities into the environment by the leakage in water infrastructure, emergency fire services, car washing and any other activity requiring use of public water. Alarmingly, the EPA in Ireland has reported that there are numerous incidences of accidental over-exposure of fluoride in drinking water due to operator error. This has major public health and environmental concerns yet no data is available on the actions taken to minimise pollution or warn the public of risks when they occurred. Local authorities remain liable for any damages caused under the Liabilities Directive.

No action has ever been taken against local authorities for the accidental release of fluoride into the public water supplies. While there have been documented cases of human fatalities and serious health consequences for communities who have suffered fluoride poisoning due to operator errors in water treatment plants in other western developed countries, no public health studies have ever, to this author's knowledge, been undertaken to examine this in Ireland.

10.11 European Council Directive on the Quality of Fresh Waters Needing Protection or Improvement in Order to Support Fish Life (2006/44/EEC)

The protection and improvement of the environment necessitates concrete measures to protect waters against pollution, including freshwaters capable of supporting fish life.

It is necessary from the ecological and economic viewpoint to safeguard fish populations from various harmful consequences resulting from the discharge of pollutant substances into the waters, such as, in particular, the reduction in the number of fish belonging to a certain species and even in some cases the disappearance of a number of these species.

Under *Article 5* of this Directive member states shall establish programmes in order to reduce pollution.

Article 9 mandates that member states may at any time set more stringent values for designated waters than those laid down in this Directive. They may also lay down provisions relating to parameters other than those provided for in this Directive.

While fluoride is not listed as a parameter requiring monitoring, fluoride under this Directive is now known to be a persistent toxic enzymatic poison in the aquatic environment.

It has been demonstrated to be an endocrine disruptor to fish. Salmonid species have been demonstrated to be particularly sensitive to this pollutant at concentrations as low as 0.5ppm. Similarly caddisfly are known to be affected at concentrations of 0.25ppm.

The regulatory authorities therefore are required to ensure that discharges of this pollutant are reduced and monitored in the aquatic environment.

It is clear that the State cannot protect or improve the quality of freshwater to protect fish life without ending the policy of water fluoridation.

10.12 European Council Regulation on the Conservation and Sustainable Exploitation of Fisheries Resources under the Common Fisheries Policy (2371/2002).

The Common Fisheries Policy (CFP) was formally created in 1983. The member states are responsible for the implementation and control of fisheries policy. The CFP Regulation is secondary legislation and is subordinate to Treaty provisions. It is now necessary to include the environmental element of sustainable development in the CFP.

The basic CFP Regulation Article 1(1) describes the scope of the CFP as encompassing 'conservation, management and exploitation of living aquatic resources, aquaculture, and the processing and marketing of fishery and aquaculture products'.

Article 2(1) also requires a precautionary approach to be taken in 'taking measures designed to protect and conserve living aquatic resource and provide for their sustainable exploitation'. The CFP also requires the EU and member states to aim at a progressive implementation of an ecosystem-based approach to fisheries management.

The CFP is also required to achieve EU environmental objectives in accordance with the integration objective under Article 11, TFEU and under international law to comply with requirements of the Marine Strategy Framework Directive, which in turn is based on the Environment Chapter, and especially on Article 191(2) (which includes, in particular, the application of the precautionary principle, principle of preventative action, rectification at source and polluter pays principle, and which requires fisheries to comply with and meet its aims and objectives). The CFP must comply with international law requirements relating to marine environmental protection and conservation; be consistent with other Community policies, including on the environment (according to Article 2(2)(d), Basic CFP Regulation).

In regard to pollution caused by the release of fluoride into the aquatic environment, little if any management measures have been taken to protect fisheries. In practice, the policy of water fluoridation poses a significant threat to the health and long-term viability of a number of protected freshwater species listed in the Habitats Directive. Consequently the Government of Ireland remains in breach of numerous EU environmental laws and policies.

Until now, there has been an almost complete lack of any fisheries management or fisheries conservation measures to protect the aquatic environment from fluoride pollution resulting from the policy of fluoridation of water. The EPA has acknowledged that fluoride pollution occurs in receiving waters downstream of wastewater treatment facilities. While fluoride is known to be harmful to fisheries at relatively low concentrations, no impact or risk assessment to examine the impact of fluoride on fisheries has been undertaken in Ireland.

It is clear therefore that any fisheries' management measures aimed at general environmental conservation and protection must address this persistent toxin that has been found to be an enzymatic poison to sensitive fish species, including the endangered salmon species, particularly in the freshwater aquatic environment. Fluoride is also know to effect algae and may be a contributory factor to the development of algae blooms in Irish waters.

It is a fundamental characteristic of environmental policy that member states are allowed to maintain or introduce more stringent protective measures under Article 193 of the TFEU.

Given that Ireland is the only member country of the European Union that mandates for the artificial fluoridation of public water supplies, it is a significant contributor of the persistent pollutant fluoride into the freshwater system and ultimately the marine environment. The Government of Ireland must address the lack of sustainability and pollution effects of this toxin in the aquatic environment. As with other nations outside Europe, such as Canada, more stringent measures may be taken to protect fisheries such as salmon from the harmful effects of fluoride. The province of British Colombia has set a recommended guideline value of 0.2mg F/L for the protection of inland fisheries including salmon and trout.

10.13 Fisheries (Consolidation) Act, 1959

Human activities such as discharges of fluoridated wastewater from urban wastewater treatment plants may cause significant increases in the fluoride concentration of surface waters.[1007],[1008],[1009],[1010],[1011],[1012],[1013],[1014],[1015],[1016],[1017],[1018],[1019],[1020],[1021]

[1007] Wright, D.A., Davison, A.W., 1975. The accumulation of fluoride by marine and intertidal animals. Environ. Pollut. 8, 1–13.
[1008] Oliveira, L., Antia, N.J., Bisalputra, T., 1978. Culture studies on the effects from fluoride pollution on the growth of marine phytoplankters. J. Fish Res. Board Can. 35, 1500–1504
[1009] Pankhurst, N.V., Boyden, C.R., Wilson, J.B., 1980. The effect of a fluoride effluent on marine organisms. Environ. Pollut. 23, 299–312.
[1010] Ares, J., Villa, A., Gayoso, A.M., 1983. Chemical and biological indicators of fluoride input in the marine environment near an industrial source (Argentina). Arch. Environ. Contam. Toxicol. 12, 589–602.
[1011] Somashekar, R.K., Ramaswamy, S.N., 1983. Fluoride concentration in the water of the river Cauvery, Karnataka, India. Int. J. Environ. Stud. 21, 325–327.
[1012] Sparks, R.E., Sandusky, M.J., Paparo, A.A., 1983. Identification of the water quality factors which prevent fingernail clams from recolonizing the Illinois River phase III. Water Resource Centre, University of Illinois at Urbana-Champaign, Urbana, IL.
[1013] Van Craenenbroeck, W., Marivoet, J., 1987. A comparison of simple methods for estimating the mass flow of fluoride discharged into rivers. Water Sci. Technol. 19, 729–740
[1014] Zingde, M.D., Mandalia, A.V., 1988. Study of fluoride in polluted and unpolluted estuarine environments. Estuarine Coastal Shelf Science 27, 707–712.
[1015] Camargo, J.A., Ward, J.V., Martin, K.L., 1992a. The relative sensitivity of competing hydropsychid species to fluoride toxicity in the Cache la Poudre River (Colorado).

Consequently, and eventhough safe levels of fluoride for aquatic life have not yet been determined (USEPA, 1986), it should be evident from the data presented in this review that discharges from anthropogenic sources (i.e., fluoride pollution) may result in a serious ecological risk for aquatic organisms.[1022] Aquatic organisms living in soft waters with low ionic content may be more adversely affected by fluoride pollution than those living in hard or seawaters because the bio-availability of fluoride ions is reduced with increasing water content of calcium and chloride.[1023],[1024],[1025],[1026],[1027]

Freshwater animals are, in general, more sensitive to fluoride toxicity than freshwater algae and macrophytes and among freshwater animals, net-spinning caddisfly larvae and upstream-migrating adult salmons seem to be the most sensitive.[1028],[1029],[1030] In consequence, safe levels of fluoride for aquatic life must be primarily based on the tolerance of net-spinning caddisfly larvae and upstream-migrating adult salmons. Because a fluoride concentration as low as 0.5 mg F/l can adversely affect net-spinning caddisfly

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Arch. Environ. Contam. Toxicol. 22, 107– 113.

[1016] Karunagaran, V.M., Subramanian, A., 1992. Fluoride pollution in the Uppanar Estuary, Cuddalore, South India. Mar. Pollut. Bull. 24, 515–517

[1017] Warrington, P.D., 1992. Lower Kitimat River and Kitimat Arm: water quality assessment and objectives. Ministry of Environment and Parks, BC, Canada.

[1018] Canadian Environmental Protection Act, 1994. Priority Substances List Supporting Document for Inorganic Fluorides. Prepared by Eco-Health Branch & Environment Canada, Ottawa (Ontario).

[1019] Skjelkv_ale, B.L., 1994. Water chemistry in areas with high deposition of fluoride. Sci. Total Environ. 152, 105–112.

[1020] Camargo, J.A., 1996a. Comparing levels of pollutants in regulated rivers with safe concentrations of pollutants for fishes: a case study. Chemosphere 33, 81–90

[1021] Clenaghan, C,. O'Neill N, Page, D., Dangerous Substances Regulations National Implementation Report, 2005 Under the Water Quality (Dangerous Substances) Regulations, 2001 (S.I. No. 12 of 2001), Environmental Protection Agency, 2006.

[1022] Camargo, J, A., Fluoride toxicity to aquatic organisms: A Review. Chemosphere 50 (2003) 251–264.

[1023] Neuhold, J.M., Sigler, W.F., 1962. Chlorides affect the toxicity of fluoride in rainbow trout. Science 135, 732–733.

[1024] Sigler, W.F., Neuhold, J.M., 1972. Fluoride intoxication in fish: A review. J. Wildlife Diseases 8, 252–254.

[1025] Pimentel, R., Bulkley, R.V., 1983. Influence of water hardness on fluoride toxicity to rainbow trout. Environ. Toxicol. Chem. 2, 381–386.

[1026] Smith, L.R., Holsen, T.M., Ibay, N.C., Block, R.M., Leon, A.B., 1985. Studies on the acute toxicity of fluoride ion to stickleback, fathead minnow and rainbow trout. Chemosphere 14, 1383–1389.

[1027] Camargo, J.A., 2002. Ameliorating effects of body size and sodium chloride on the intraspecific tolerance of netspinning caddisfly larvae to fluoride toxicity. Bull. Environ. Contam. Toxicol. (in revision).

[1028] Camargo, J.A., La Point, T.W., 1995. Fluoride toxicity to aquatic life: a proposal of safe concentrations for five species of Palearctic freshwater invertebrates. Arch. Environ. Contam. Toxicol. 29, 159–163.

[1029] Camargo, J.A., 1996b. Estimating safe concentrations of fluoride for three species of Nearctic freshwater invertebrates: multifactor probit analysis. Bull. Environ. Contam. Toxicol. 56, 643–648

[1030] Damkaer, D.M., Dey, D.B., 1989. Evidence for fluoride effects on salmon passage at John Day dam, Columbia river, 1982– 1986. N. Am. J. Fish. Manag. 9, 154–162.

larvae and upstream-migrating adult salmons, inhabiting soft waters with low ionic content; safe levels below this fluoride concentration are recommended in order to protect these freshwater animals from fluoride pollution.[1031] The EPA have acknowledged that in Ireland potential waters at risk from fluoride pollutant include receiving waters located downstream of drinking- and wastewater treatment plants and areas where there is significant leakage from the drinking water distribution system.[1032] The EPA in Ireland have reported a number of exceedances of the standard for fluoride in surface waters associated with the infiltration of drinking water and discharges from urban wastewater treatment plants.[1033] The impact of wastewater discharges on fluoride concentrations in receiving waters was also reported by Sparks et al. and Camargo where '*discharges of fluoridated municipal waters also cause significant increases (about five times the natural background level) in the fluoride concentration of recipient rivers.*'[1034,1035]

Evidence from the study by Daemker and Dey[1036] indicates that some species of fish (salmon) are harmed at levels of about 0.25mg/L, while evidence from Camargo[1037] demonstrates that net-spinning caddisfly larvae are harmed at fluoride levels as low as 0.2mg/L. It is further known that the toxic action of fluoride on the health of aquatic (and terrestrial) animals resides in the fact that fluoride ions act as enzymatic poisons, inhibiting enzyme activity (e.g., phosphatases, hexokinase, enolase, succinic dehydrogenase, pyruvic oxidase) and, ultimately, interrupting metabolic processes such as glycolysis and synthesis of proteins.[1038]

[1031] Camargo, J, A., Fluoride toxicity to aquatic organisms: A Review. Chemosphere 50 (2003) 251–264

[1032] Clenaghan, C,. O'Neill N, Page, D., Dangerous Substances Regulations National Implementation Report, 2005 Under the Water Quality (Dangerous Substances) Regulations, 2001 (S.I. No. 12 of 2001), Environmental Protection Agency, 2006.

[1033] Clenaghan, C,. O'Neill N, Page, D., Dangerous Substances Regulations National Implementation Report, 2005 Under the Water Quality (Dangerous Substances) Regulations, 2001 (S.I. No. 12 of 2001), Environmental Protection Agency, 2006.

[1034] Sparks, R.E., Sandusky, M.J., Paparo, A.A., 1983. Identification of the water quality factors which prevent fingernail clams from recolonizing the Illinois River phase III. Water Resource Centre, University of Illinois at Urbana-Champaign, Urbana, IL.

[1035] Camargo JA. Fluoride toxicity to aquatic organisms: a review. Chemosphere. 2003 Jan;50(3):251-64)).

[1036] Daemker, DM, Dey, DB. Evidence for fluoride effects on salmon passage at John Day Dam, Columbia River 1982-1986, North American Journal of fisheries management, 1989, 9, 154-162

[1037] Camargo, J.A., Garc_ıa de Jal_on, D., Mu~noz, M.J., Tarazona, J.V., 1992b. Sublethal effects of sodium fluoride (NaF) on net-spinning caddisflies (Trichoptera). Aquatic Insects 14, 23–30.

[1038] Kessabi, M., 1984. M_etabolisme et biochimie toxicologique du fluor: Une revue. Rev. M_ed. V_et. 135, 497–510.

Camargo similarly reported how fluoride was demonstrated to be an endocrine disruptor in the environment and how relatively low levels of fluoride below 4ppm were observed to stimulate female fecundity (reproductive fertility).[1039],[1040],[1041] Similar findings have also been observed in human studies.[1042],[1043]

Warrington[1044], in British Columbia, where the softness of major salmonid watercourses is the rule, combined the findings of Angelovic[1045], and of Pimental; and Bulkley[1046] to calculate that the chronic threshold for rainbow trout at 12 degrees and water hardness of 10 mg/L (calcium carbonate) is 0.2 mgF/L.

It is evident therefore, that the use of fluorosilicictes as additives in drinking water treatment results in the discharge of wastewaters with higher than background levels of the pollutant fluoride, at concentrations that are known to be harmful to salmon and their food supply. This would constitute a violation of the Fisheries Act 1956 under Sections 131,171 b), 172, 173 and Section 111 subsection 1(j) of Fisheries (Amendment) Act, 1999.

[1039] Connell, A.D., Airey, D.D., 1982. The chronic effects of fluoride on the estuarine amphipods Grandidierella lutosa and G. lignorum. Water Res. 16, 1313–1317.
[1040] McClurg, T.P., 1984. Effects of fluoride, cadmium and mercury on the estuarine prawn Penaeus indicus. Water SA 10, 40–45.
[1041] Camargo JA. Fluoride toxicity to aquatic organisms: a review. Chemosphere. 2003 Jan;50(3):251-64
[1042] Gupta, S. K.; Khan, T. I.; Gupta, R. C.; Gupta, A. B.; Gupta, K. C.; Jain, P., and Gupta, A. Compensatory hyperparathyroidism following high fluoride ingestion - a clinico - biochemical correlation. Indian Pediatr. 2001 Feb; 38(2):139-46
[1043] Trivedi, N.; Mithal, A.; Gupta, S. K., and Godbole, M. M. Reversible impairment of glucose tolerance in patients with endemic fluorosis. Fluoride Collaborative Study Group. Diabetologia. 1993 Sep; 36(9):826-8
[1044] Warrington PD. *Ambient Water Quality Criteria for Fluoride. Technical Appendix.* British Columbia Ministry Of Environment. 1990.
[1045] Angelovic JW, Sigler WF, Neuhold JM. Temperature and fluorosis in Rainbow trout. *Journal. Water Pollution Control Federation* 33 371-381 1961.
[1046] Pimental R. Bulkley RB. Influence of-water hardness on fluoride toxicity to Rainbow trout. *Environmental Toxicology and Chemistry* 2 381-386 1983.

10.14 European Council Marine Strategy Framework Directive 2008/56/EC

This Directive promotes the integration of environmental considerations into all relevant policy areas with the overall aim of promoting sustainable use of the seas and conserving marine ecosystems. The Directive includes coastal waters.

The Directive aims to prevent and reduce inputs in the marine environment, with a view to phasing out pollution so as to ensure that there are no significant impacts on or risks to marine biodiversity, marine ecosystems, human health or legitimate uses of the sea (Article 1.2).

The Directive requires that marine strategies shall apply an ecosystem-based approach to the management of human activities, ensuring that the collective pressure of such activities is kept within levels compatible with the achievement of good environmental status (Article 1.3). Member states are required to determine, for marine and coastal waters, a set of characteristics for good environmental status on the basis of the qualitative descriptors listed in the Directive.

This requires member states to ensure amongst other criteria that:

a) human-induced eutrophication is minimised, especially adverse effects thereof, such as losses in biodiversity, ecosystem degradation, harmful algae blooms and oxygen deficiency in bottom waters.

> It has been found that anthropogenic fluoride emissions may be a contributor to the development of harmful algae blooms.

b) concentrations of contaminants are at levels not giving rise to pollution effects.

> It has been demonstrated that anthropogenic releases of fluoride from wastewater treatment plants give rise to pollution effects and are harmful to freshwater fisheries and impact on the quality and environmental status of coastal waters impairing their sustainable use and impacting on biodiversity.

10.15 Local Government (Water Pollution) Regulations, 1992.

Fluoride is listed as a Harmful Substance in second schedule of the Local Government (Water Pollution) Regulations of 1992.

It is widely acknowledged that high silica fluosilicic acids have escaped general recognition. It is also accepted that there exists a paucity of toxicological data on hexafluorosilicic acid and that persons drinking fluoridated water may be exposed to compounds that have not been thoroughly tested for toxicity. Alarmingly and remarkably, given that this has been highlighted for many years, no data is available on mutagenic effects, teratogenic effects, development toxicity, cytotoxicity, carcinogenic effects, cogenotoxicity, short-term and sub-chronic exposure or synergistic/antagonistic effects of fluosilicic acid or its compounds. When fluorosilicictes are added to water they dissociate to form fluorosilicicte ions [SiF6] 2- with two negative electrical charges, accompanied by either two individual ions of hydrogen H+ (from fluorosilicic acid) or of sodium (Na+) (from sodium fluorosilicicte). The individual elements, silicon (Si) and fluorine (F) in the fluorosilicicte ion cannot move independently - at neutral pH they act as the complex substance fluorosilicicte. At around the normal pH of 7, approximately 97% of the fluorine in fluorosilicicte added to the water is present in the form of ionised fluoride, F+ . At the very slightly acidic pH of 6, only 27% of the fluorine in fluorosilicicte is present as fluoride - the rest is associated with other ions, and forms a number of complex and unstable compounds and ions that change over variable periods of time and at different pH values.

Analysis of samples of fluosilicic acid, reported in 1936, showed the silica component to be present as a primary silica component denoted by the formula H2SiF6 as well as the secondary silica component with its own characteristic chemical behaviour (fluosilicic acid).[1047] This was confirmed by Westendorf who found that under physiological conditions, dissociation of silicofluorides was no more than 66% in the concentration range considered optimum for fluoridated water. The partially dissociated residue would be the ion [SiF2(OH)4]2- which would then be present in the water at the same concentration as the originally introduced SiF.[1048]

This matter is of relevance as toxic or persistent organic compounds of silicon and substances which may cause the formation of such compounds in water are classified as harmful substances. The presence of such compounds and their potential toxicity and behaiour in either drinking water or wastewater has yet been examined in Ireland.

[1047] Thomsen, Milton S, High-silica fluosilicic acids : specific reactions and the equilibrium with silica, Am, Chem, Soc. 74 : 1690-1692

[1048] Westendorf J. The kinetics of acetylcholinesterase inhibition and the influence of fluoride and fluoride complexes on the permeability of erythrocyte membranes. Ph.D. Dissertation in Chemistry, University of Hamburg, Germany, 1975

The presence of such harmful substances as fluoride in sewage effluent or trade effluent is required to be controlled by licence issued by the local authority. A licence application is required to be accompanied by the results of a prior investigation including an assessment of the environmental impact of alternative methods of disposal of the harmful substance. The assessment must also examine the human health and ecological impacts of the substance on aquatic ecosystems and demonstrate that it will not interfere with the use of the water for domestic, agricultural, fisheries, commercial, industrial or recreational purposes.

Only provided that all practical technical precautions are observed to prevent water in an aquifer being affected by that harmful substance through examining the human and ecological consequences in the manner set out above, can a local authority specify conditions in a licence to allow discharge. To the author's knowledge, no such investigation examining the impact of fluoride or fluorosilicicte compounds has ever taken place in Ireland or in any other jurisdiction.

10.16 Water Quality (Dangerous Substances) Regulations (S.I. NO. 12/2001)

The Water Quality (Dangerous Substances) Regulations (S.I. No. 12 of 2001) were introduced to give further effect to Council Directive 76/464/EEC on pollution caused by certain dangerous substances discharged into the aquatic environment and to support implementation of the Water Framework Directive (2000/60/EC).

The Regulations prescribe water quality standards in respect of 14 dangerous substances including fluoride in surface waters. Fluoride was selected by virtue of its toxicity, persistence and bioaccumulation . Fluoride is listed as a persistent toxic substance that poses a specific risk to human health. Substances are regarded as very toxic if there is the possibility of long-term exposure to substances, which could be fatal. If a substance is persistent in groundwater or freshwater and the substance is toxic to humans and bio-accumulative a substance is determined to be hazardous.[1049]

The author of this report has already highlighted the various violations in EU law pertaining to surface water quality, groundwater and fisheries protection as a consequence of fluoride emissions from both point sources and diffuse uncontrolled sources as a consequence of water fluoridation. Similarly the Irish EPA have acknowledged and reported that major anthropogenic

[1049] Classification Of Hazardous And Non-Hazardous Substances In Groundwater, Hydrometric And Groundwater Section, Office Of Environmental Assessment, Environmental Protection Agency, 2010

sources of fluoride include fluoridation of public water supplies, leakage of mains water from the drinking water distribution system and municipal wastewater treatment plants.[1050]

In the latter report, the Irish EPA have also acknowledged that potential waters at risk from fluoride pollutant include receiving waters located downstream of drinking water treatment plants, downstream of wastewater treatment plants and areas where there is significant leakage from the drinking water distribution system. As previously reported the Agency further reported a number of exceedances of the standard for fluoride associated with the infiltration of drinking water into surface waters as well as leaking drinking water distribution mains into groundwater aquifers which the agency regarded as a significant potential source of fluoride (as drinking water contains fluoride at levels of between 800 and 1000 μg/l particularly in urban areas).

Risk of Accidental Discharge and Increased Exposure

The risk to public health and safety from accidental over-exposure to silicafluorides in drinking is a major public health concern. Over the past few years there have been hundreds of incidences of fluoride over-exposure in drinking water supplies reported by the EPA. In their most recent report on drinking water quality the EPA alarmingly reported that the number of Water Supply Zones with exceedances for fluoride were sixty four. Exceedances were observed by the Agency to be almost entirely due to public water supplies being dosed with fluoride at levels in excess of the legally permitted dose. [1051]

It is evident therefore that all exceedances were due to operator error. Based on the polluter pays principle and the Environmental Liabilities Directive the local authorities where these incidences occurred are liable for any damages that may be caused as a consequence of these incidences.

Internationally several such instances of accidental over-fluoridation of public water supplies have been reported. Eight patients with renal insufficiency were dialysed with accidentally over fluoridated water (dose of 1 g fluoride) and became symptomatic because of virtually absent renal elimination of fluoride. One patient died from cardiac arrest. Postmortal fluoride concentration in blood was 4.9 mg/L.[1052],[1053]

[1050] Clenaghan, C,. O'Neill N, Page, D., Dangerous Substances Regulations National Implementation Report, 2005 Under the Water Quality (Dangerous Substances) Regulations, 2001 (S.I. No. 12 of 2001), Environmental Protection Agency, 2006.

[1051] Hayes N,. Page D,. Sweeney L,. O'Leary G,. The Provision and Quality of Drinking Water in Ireland A Report for the Year 2010, Environmental Protection Agency, 2011.

[1052] McIvor M, Baltazar RF, Beltran J, Mower MM, Wenk R, Lustgarten J, Salomon J (1983). Hyperkalemia and cardiac arrest from fluoride exposure during hemodialysis. Am J Cardiol 51: 901-902

[1053] Waldbott GL (1981). Mass intoxication from accidental overfluoridation of drinking water. Clin Toxicol 18: 531-541

The number of over-fluoridation incidences reported in Ireland is a major cause for concern. It is apparent that no public notice warnings were issued to communities when they occurred. It is likely that there may have been incidences where at risk individuals may have had medical complications as a consequence of the over-fluoridation.

The EPA has reported that a total of 51 public water supplies and 11 public group water schemes exceeded the legal fluoride concentration (1.5mg/L). This should be of some concern as this level is twice the current recommended fluoride concentration for drinking water which is 0.7mg/l.

No information was provided by the Agency on the population exposed, the location of the geographic areas, potential public health consequences, if there were reported incidences of ill-heath in the community, whether the public were informed of the risk and for what period of time the exceedances continued.

No information was provided on what action, if any, was taken against the local authorities responsible for non-compliance and subsequent to the event, what incidence reports were issued identifying the failures to comply with standard operating practices. Further information is required in order to improve risk management procedures given that over-fluoridation of drinking water supplies may have fatal consequences for high-risk individuals within the communities concerned.

10.17 Urban Wastewater Treatment Regulations, S.I. NO. 254/2001

The Urban Wastewater Treatment (UWWT) Regulations, S.I. No. 254 of 2001 transpose into Irish law the provisions of EU Council Directive 91/271/EEC concerning urban wastewater treatment.

The UWWT Regulations, 2001, incorporate and update the Environmental Protection Agency Act, 1992 (Urban Wastewater Treatment) Regulations, 1994 as amended in 1999.

The essential purpose of the 2001 Regulations is to tackle eutrophication of Irish waters by designating 30 water bodies (including river stretches, lakes and estuaries) as sensitive areas for the purpose of the UWWT Directive, in addition to the 10 water bodies, which were designated in 1994.

The Fourth Schedule of the Regulations mandates that discharges from treatment plants do not adversely affect the environment or prevent receiving waters from complying with other Community Directives. The Regulations require that sludge must be disposed of safely in an environmentally acceptable manner. The U.S. EPA undertook a hazard identification study of pollutants that may cause adverse effects to public health and the environment.[1054] Based on the results of this study, a list of 254 pollutants were identified and of these 31 were then evaluated in a comprehensive hazard identification study. The goal was to identify pollutants that may potentially cause human health or ecological risks. Based on the results of the risk assessments of the comprehensive hazard identification, twelve pollutants were found to have critical exposure pathways. Fluoride was one of the principal pollutants.

The regulations also require Sanitary Authorities to monitor surface waters, which receive discharges from urban wastewater treatment plants where it is anticipated that the receiving waters will be significantly affected, with implications for compliance with other Directives.

It is obvious that the policy of water fluoridation results in clear violations of the Urban Wastewater Regulations. Over 99.5% of the fluoride compounds added to drinking water are not used for the purpose intended and end up in wastewater treatment systems. Wastewater treatment has been shown to be largely ineffective in removing the pollutant fluoride from wastewaters. This is resulting in significant discharges of a persistent bio-accumulative pollutant into the environment at concentrations that are harmful to freshwater fish and invertebrates.

The only way to reduce the concentrations of fluoride released into the environment is to end the policy of water fluoridation.

[1054] U.S EPA Technical Support Document for the Round Two Sewage Sludge Pollutants, Office of Water, EPA-822-R-96-003, August 1996.

10.18 European Council Food Hygiene Regulation 178/2002

Regulation (EC) No 178/2002 of the European Parliament and of the Council of 28th January, 2002 establishes the general principles and requirements of food law within the European community.

The regulations specify that water is ingested directly or indirectly like other foods, thereby contributing to the overall exposure of a consumer to ingested substances, including chemical contaminants[1055]. It is acknowledged that the safety of drinking water has a direct influence on the health of consumers.[1056] It is acknowledged that the systemic exposure of the populationto fluoride by fluoridation of water may result in impair normal development of enamel in the pre-eruptive tooth and cause fluorosis.[1057]

It is generally accepted that the health of sensitive consumers is affected by the consumption of fluoridated water resulting in dental fluorosis. It is acknowledged that the incidence of dental fluorosis is increasing in Ireland demonstrating the growing over-exposure of the population to fluoride through consumption of drinking water.

The State has a legal obligation to ensure that the quality of water used in the preparation of food, such as infant formula feed, namely boiled tap water[1058] in addition to water emitted from the tap, does not present a risk to consumers.[1059] It was acknowledged[1060] by the European Commission in 2003 that the fluoride in infant formula became a problem as a consequence of water fluoridation, yet incredible this information wa s not acted on by the authorities in Ireland. In fact it would appear that the recommendation of theEuropean scientific committee were completely ignored.

According to European law a high level of protection of human life and health should be assured in the pursuit of Community policies. It is necessary to adopt measures aimed at guaranteeing that unsafe food is not placed on the market and at ensuring that systems exist to identify and respond to food safety problems in order to ensure the proper functioning of the internal

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[1055] Regulation (EC) No 178/2002 Of The European Parliament And Of The Council Of 28 January 2002

[1056] Drinking Water and Health. A Review and Guide for Population Health HSE. 2008

[1057] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

[1058] It is scientifically accepted that boiled fluoridated water contains higher concentrations of fluoride than tap water.

[1059] Council Directive 98/83/EC on the Quality of Water Intended for Human Consumption, Article 6, Point 1(d).

[1060] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

market and to protect human health. Food law is aimed at the reduction, elimination or avoidance of a risk to health as well as targeted measures or other actions to protect health.[1061] Fluoride is listed as a persistent toxic substance under EU law that poses a specific risk to human health.[1062]

The fluoride content of infant formulae made with fluoridated tap water ranges from about 0.7 to 1.4 ppm.[1063,1064] These levels are 100-fold higher than the levels found naturally in breast milk.[1065] The British Medical Council as far back as 2002 reported that individuals most likely to have supra-optimal fluoride intakes are formula-fed infants in fluoridated areas.[1066]

American paediatricians recommend a maximum fluoride supplement of 0.25mg for children in unfluoridated areas aged 0-2years.[1067] If fluoridated water is used as liquid for formula feed the exposure of infants to fluoride corresponds to **4-6 times the daily doses** currently recommended by American paediatricians.[1068] Based on average fluid intakes and body weights, many infants exceed intakes of 0.15 mg fluoride/kg/day.[1069] It has been documented that a daily dose exceeding 0.05 mg/kg/d can result in dental fluorosis.[1070] The long-term medical consequences of this level of fluoride intake in infant foods have never been studied.

It has been reported that the only way to limit fluoride intake in fluoridated communities, to acceptable levels, as recommended by the *American Society of Dentistry for Children*, is to avoid using fluoridated water to dilute powered infant milk.[1071]

This means consumers are faced with no choice, as the only way to remove fluoride in drinking water, apart from at source, is by reverse osmosis, and

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[1061] Regulation (EC) No 178/2002 Of The European Parliament And Of The Council Of 28 January 2002

[1062] The Water Quality (Dangerous Substances) Regulations (S.I. No. 12 of 2001)

[1063] McKnight-Hanes MC, Leverett DH, Adair SM, Shields CP. Fluoride content of infant formulas: soy-based formulas as a potential factor in dental fluorosis. *Pediatr Dent* 1988;10(3):189-94.

[1064] Silva M, Reynolds EC. Fluoride content of infant formulae in Australia. *Aust Dent J* 1996;41(1):37-42.

[1065] Fomon SJ, Ekstrand J. Fluoride intake by infants. *J Public Health Dent* 1999;59(4):229-34.

[1066] Medical Research Council working group report: Water fluoridation and health, September 2002

[1067] Committee on Nutrition Fluoride supplementation. Paediatrics 77, 758-761 (1986)

[1068] Murnane M. Artificial feeding. In: Clements A (ed). Infant and family health in Australia: a textbook for community health workers. Churchill livingstone, melbourne

[1069] Erdal S and Buchanan SN. A quantitative look at fluorosis, fluoride exposure, and intake in children using a health risk assessment approach. *Environ Health Perspect* 2005;113(1):111-7.

[1070] Whitford GM. The physiological and toxicological characteristics of fluoride. *J Dent Res* 1990;69 Spec No:539-49; discussion 556-7.

[1071] Buzalaf, M. A., Granjeiro, J. M., Damante, C. A., & de Ornelas, F. (2001). Fluoride content of infant formulas prepared with deionized, bottled mineral and fluoridated drinking water. American Society of Dentistry for Children. (ASDC) Journal of Dentistry for Children, 68(1), 37-41.

according to the WHO, de-ionized water is not suitable for human consumption and poses additional health risks to consumers.[1072]

Therefore parents and care providers are left with no option but to buy bottled water with low-fluoride levels to prepare formula feed for infants in order to protect the infant from the effects of fluoride intoxication. The only other alternative is for the State to stop fluoridating drinking water at source.

The precautionary principle has been invoked to ensure health protection in the European Union. The proper approach to risk assessment in toxicology and environment exposure is to identify the high risk groups in the community and to set safety standards for daily doses with sufficient margin to protect them with a high degree of certainty. Bottle-fed infants have been identified as a high risk group not just because they are at higher risk of developing dental fluorosis but also because the uptake of fluoride may be higher in infants and young children than in adults.[1073] The exposure of this group to fluoridated drinking water may be considered a contributor to skeletal fluorosis in older age.[1074] It is considered that skeletal fluorosis is likely to become prevalent among high risk groups in artificially fluoridated areas as people who have been exposed to fluoridated water from infancy reach middle age. Current research from Ireland appears to support this view.

High fluoride contents in infant foods are of concern also as fluoride interacts with both calcium and magnesium. Calcium and magnesium in water and food are known to have antitoxic activity. They can help prevent the absorption of some toxic elements such as lead and cadmium from the intestine into the blood, either via direct reaction leading to formation of an unabsorbable compound or via competition for binding sites.[1075,1076,1077,1078,1079,1080,1081,1082]

[1072] Kozisek, F. Health risks from drinking demineralised water, Rolling revision of the WHO Guidelines for drinking-water quality, World Health Organization Geneva, 2004

[1073] Zipkin L et al. Unnary fluoride levels associated with the use of fluoridated waters. Public Health Report 17, 767-772 (1956).

[1074] Mark Diesendorf, Ph.D. The Health Hazards of Fluoridation: A Re-examination, Human Sciences Program, Australian National University, Canberra. International Clinical Nutrition Review, April 1990 Volume 10, No 2.

[1075] Thompson, D.J. (1970) *Trace element in animal nutrition*. 3rd ed. Int. Minerals and Chem. Corp., Illinois.

[1076] Levander, O.A. (1977). Nutritional factors in relation to heavy metal toxicants. *Fed. Proc.* 36, 1683-1687

[1077] Oehme, F.W. (ed.) (1979). *Toxicity of heavy metals in the environment*. Part 1. M.Dekker, New York.

[1078] Nadeenko, V.G., Lenchenko, V.G. and Krasovskii, G.N. (1987) Combined effect of metals during their intake with drinking water. (In Russian.) *Gig. Sanit.* No.12 /1987 (volume not given), 9-12.

[1079] Durlach, J., Bara, M. and Guiet-Bara, A. (1989) Magnesium level in drinking water: its importance in cardiovascular risk. In *Magnesium in Health and Disease* (ed. Y.Itokawa and J.Durlach), pp. 173-182, J.Libbey & Co Ltd, London

[1080] Hopps, H.C. and Feder, G.L. (1986) Chemical qualities of water that contribute to human health in a positive way. *Sci. Total Environ.* 54, 207-216.

[1081] Plitman, S.I., Novikov, Yu.V., Tulakina, N.V., Metelskaya, G.N., Kochetkova, T.A. and

The toxic effect of the fluoride ion plays a key role in acute calcium and magnesium deficiency. The amount of fluoride assimilated by living organisms constantly increases as calcium and magnesium absorption diminishes.[1083]

As noted in the British Medical Bulletin,"(f)*luoride seeks out minerals such as magnesium and binds with it, making magnesium unavailable to the body and unable to do its work. The magnesium-fluoride mineral produced is called sellaite; it is almost insoluble and ends up taking the place of magnesium in hard tissues like bone and cartilage, but its brittleness makes the bone susceptible to fracture. The reduction in available magnesium causes a decrease in enzymatic action in the body*". [1084]

Sufficient evidence is now available to confirm the health risk from drinking water deficient in calcium or magnesium. These risks are exasperated with the presence of fluoride. In addition to an increased risk of sudden death, it has been suggested that intake of water low in magnesium and calcium may be associated with a higher risk of motor neuronal disease, pregnancy disorders (so-called pre-eclampsia, and sudden death in infants), a higher risk of fracture in children and some types of cancer.

Furthermore, the possible role of calcium and magnesium in the development of cardiovascular disease cannot be excluded. Calcium (Ca) and magnesium (Mg) are both essential elements while calcium is a substantial component of bones and teeth. In addition, it plays a role in neuromuscular excitability (i.e., decreases the proper function of the conducting myocardial system, heart and muscle contractility, intracellular information transmission and the coagulability of blood). Magnesium plays an important role as a cofactor and activator of more than 300 enzymatic reactions including glycolysis, ATP metabolism, transport of elements such as sodium, potassium, and calcium through membranes, synthesis of proteins and nucleic acids, neuromuscular excitability and muscle contraction.

It is obvious that any substance artificially added to drinking water that may interfere with the biological activity of essential elements such as magnesium or calcium in infants through ingestion of fluoride-contaminated baby-feed must cease for the health and well being of infants, who represent the future of this country. It is clear that the health significance of high concentrations of fluoride in infant food cannot be underestimated. Any associated risk such as Sudden Infant Death Syndrome cannot be ignored especially when recent

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Khvastunov, R.M. (1989) On the issue of correction of hygenic standards with account of drinking water hardness. (In Russian.) *Gig. Sanit.* No. 7/1989 (volume not given), 7-10

[1082] Kozisek, F. Health risks from Drinking Demineralised water, Rolling revision of the WHO Guidelines for drinking-water quality, World Health Organization Geneva, 2004

[1083] Marier J R. Observations and implications of the (Mg F) interrelations in bio-systems: a review and comments on magnesium intake and fluoride intake in the modern-day world. Proceedings of the Finnish Dental Society 76. 82-92, 93-102, 1980. (Abstracted in *Fluoride* 14, 142 1981

[1084] Crawford M.D. GARDNER. M.J. Morris J .N. Cardio Vascular Disease and the mineral content OF drinking Water. British Medical Bulletin, VOL 27 No 1. Pp 21-24

research has demonstrated that both partial breastfeeding and exclusive breastfeeding were associated with a significantly reduced risk of sudden infant death syndrome.[1085],[1086]

In other words bottle-fed babies were at higher risk of Sudden Infant Death Syndrome.

It is noted that in 2002 the Food Safety Authority of Ireland carried out a limited risk assessment of the fluoride intake of infants under four months consuming infant formula reconstituted with fluoridated water. The risk assessment found that infants would be at high risk of developing dental fluorosis.[1087] Fluoride accumulation in hard tissues such as teeth and bone may be viewed as a defence mechanism against fluoride intoxication because of the removal of fluoride from body circulation.[1088],[1089]

The implications for general health as discussed in this report were not examined in the risk assessment.

While the FSA risk assessment demonstrated risk to the public, the methodology undertaken was simplistic and incomplete in many areas. For example, it did not examine the potential impact of high fluoride levels on bone or other human organs in children or adults. It did not acknowledge the published research findings that demonstrate a seven fold increase in the incidence of certain cancers in young boys living in fluoridated communities, nor how increases in bone fluoride levels are associated with increased osteoid parameters and decreased bone micro-hardness.

That is, deposition of fluoride in bone diminishes bone micro-hardness by interfering with mineralization[1090] resulting in increased *brittleness* and susceptibility to fracture in both children and adults, especially elderly women.[1091],[1092]

[1085] Hauck Fern R., Thompson John,. O. Tanabe, Kawai MPHa,. Moon Rachel Y . Vennemann M, Breastfeeding and Reduced Risk of Sudden Infant Death Syndrome: A Meta-analysis, Pediatrics, June 13, 2011. DOI: 10.1542/peds.2010-3000.

[1086] Ip S, Chung M, Raman G, et al. Breastfeeding and maternal and infant health outcomes in developed countries. *Evid Rep Technol Assess (Full Rep)*. 2007;(153):1–186

[1087] Anderson, W, A,. Pratt I,. Ryan M.R., Flynn A. A Probabilistic Estimation of Fluoride Intake by Infants up to the Age of 4 Months from Infant Formula Reconstituted with Tap Water in the Fluoridated Regions of Ireland, *Caries Res* 2004;38:421-429

[1088] Sigler, W.F., Neuhold, J.M., 1972. Fluoride intoxication in fish: A review. J. Wildlife Diseases 8, 252–254.

[1089] Kessabi, M., 1984. M_etabolisme et biochimie toxicologique du fluor: Une revue. Rev. M_ed. V_et. 135, 497–510.

[1090] Ng AH, Hercz G, Kandel R, Grynpas MD. Association between fluoride, magnesium, aluminum and bone quality in renal osteodystrophy. Bone. 2004 Jan;34(1):216-24.

[1091] Crawford M.D. GARDNER. M.J. Morris J .N. Cardio Vascular Disease and the mineral content OF drinking Water. British Medical Bulletin, VOL 27 No 1. Pp 21-24

[1092] B. Lawrence Riggs, M.D., Stephen F. Hodgson, M.D., W. Michael O'Fallon, Ph.D., Edmund Y.S. Chao, Ph.D., Heinz W. Wahner, M.D., Joan M. Muhs, B.S.N., Sandra L.

Research has also demonstrated how low levels of fluoride result in inhibition of cellulose digestion causing partial blocking of normal cellulose degradation.[1093] The study did not examine the inter-action of fluoride with other common compounds such as aluminium and the bio-availability and consequence of adding fluoride to water with low calcium and magnesium levels.

Astonishingly, the FSA did not issue any guidance or public health warning to healthcare professionals or parents of the risk of infants developing fluorosis from drinking infant formula reconstituted with fluoridated water. The FSA, furthermore, did apply the precautionary approach to their methodology which demands conclusive evidence of no harm. The FSA demonstrated harm yet did not advise of risks to consumers.

The potential health risks are examined in some detail in this report. It is clear even with the most obvious health risks such as dental fluorosis (caused from fluoridation of drinking water supplies) that any policy supported or enacted by the Government that would increase risk to health, such as injecting silicofluorides into drinking water supplies, is unacceptable. This is particularly so for high-risk groups such as bottle-fed infants in fluoridated communities who are being exposed to extremely high concentrations of a dangerous element at concentrations of up to 200 times above the natural background levels found in normal breast-milk.

Given that Ireland has the lowest level of breastfeeding in Europe[1094] and that it is the only European country with a statutory policy of water fluoridation of all public water supplies, it is evident that children and consumers in Ireland remain the most high-risk of all European citizens to dental and skeletal fluorosis as well as any of the other numerous potential health implications associated with fluoride intoxication, from ingestion of contaminated food and water and resultant exposure to harmful levels of this dangerous substance. It is no surprise, considering these facts, that the citizens of Ireland have one of the highest incidences of neurological diseases, coronary vascular disease, diabetes, endemic fluorosis of the population and incidences of osteoporosis in the world. Coronary heart disease, stroke and fragility fractures remain a huge part of the current health and social care expenditure which amounted to 14.8 billion in 2010.

It is clear that in order to comply with EU food law the Government of Ireland must adopt appropriate risk minimization and end the policy of water fluoridation immediately. Failure to do so or to comply with the relevant provision of the EC Regulations is a criminal offence.[1095]

Cedel, M.S., and L. Joseph Melon, III, M.D. Effect of Fluoride Treatment on the Fracture Rate in Postmenopausal Women with Osteoporosis, (N Engl J Med 1990; 322:802–9.)

[1093] C. C. Chamberlain and Wise Burroughs, Effect of Fluoride, Magnesium and Manganese Ions on in Vitro *Journal of Animal Science* 1962, 21:428-432.

[1094] Euro Growth Study, Conducted at 22 European Centres from 1992-1996

[1095] S.I. No. 747 of 2007 European Communities (General Food Law) Regulations 2007,

10.19 European Council Regulation (852/2004/EEC) on the Hygiene of Foodstuffs

Under Regulation 852/2004 of the European Parliament there is a requirement in European law to ensure that "*there is to be an adequate supply of potable water supply, which is to be used whenever necessary to ensure that foodstuffs are not contaminated*". [1096]

It is clear, as discussed in this report, that the policy of water fluoridation results in contamination of infant foods. It is likewise obvious that any foodstuffs washed or cooked in fluoridated water will be contaminated with fluoride. Furthermore, any beverages requiring boiled water will result in further contamination as fluoride concentrates in boiled water.

The use of fluoridated drinking water for irrigation of plants results directly in the bio-accumulation of fluorides in food.

Any foodstuffs washed, prepared or cooked in fluoridated water will result in contamination of foodstuffs. Boiling fluoridated water will further increase the level of contamination.

The disposal of wastewater treatment sludges on land has been found by the U.S. EPA[1097] to be a significant environmental risk and direct exposure pathway for the bioaccumulation of fluoride, a known persistent toxin in the environment and within the human food chain.

By continuing with a policy of injecting silicofluorides into drinking water the Government of Ireland cannot comply with EU regulations on the hygiene of foodstuffs for the protection of consumers.

Part 3, Section 22

[1096] Regulation (EC) No 852/2004 of The European Parliament and of the Council of 29 April 2004 on the hygiene of foodstuffs. Annex II Chapter VII, Paragraph 1 (a)

[1097] U.S EPA Technical Support Document for the Round Two Sewage Sludge Pollutants, Office of Water, EPA-822-R-96-003, August 1996.

10.20 European Council Regulations (S.I. No. 243/1998) for Infant Formulae and Follow-On Formulae

The regulations regarding infant formula are very strict and based on generally accepted established scientific data. The use of food ingredients is subject to the prohibitations and limitations specified in Schedule I and II of these regulations. No substance other than a substance specified in Schedule III may be used in the manufacture of infant formulae and follow-on formulae for the purposes of satisfying the requirements on mineral substances, vitamins, amino acids and other nitrogen compounds and other substances having a particular nutritional purpose.

The production of infant formula requires the addition of water in the manufacturing process as well as in the preparation of the final product before consumption. Boiled drinking water is required for the preparation of formula milk. Ireland remains the only country within the EU that legislates for water fluoridation, a process that requires the introduction of fluoride into drinking water at concentrations considerably above ambient background levels in the surface or groundwater.

Fluoride therefore constitutes a substance that is added within the definitions of these regulations. Fluoride is not included in the list of minerals permitted to be added to infant formula or follow-on formula.

The regulations provide for strict requirements for labelling, advertising and presentation of infant formula and follow-on formula. The labelling of infant formula and follow-on formula is required to bear amongst other items instructions for appropriate preparation of the product and a warning against the health hazards of inappropriate preparation. The manufacturer is required to provide informational and educational materials including, *inter alia,* written and audiovisual materials in relation to the feeding of infants.

The regulations require manufacturers to ensure information is provided to pregnant women, and mothers of infants and young children, and shall include clear information on, in addition to other items, the proper use of infant formulae, whether manufactured industrially or home-prepared, the health hazards of inappropriate foods or feeding methods and the health hazards of improper use of infant formulae.

To my knowledge no such information of the potential risks associated with preparation of infant formula using fluoridated water or bottled water is provided by either the manufacturer or by public health authorities in Ireland.

This is despite the fact the fact that a European Commissison scientific committee reported[1098] in 2003 that water fluoridation was a problem as it resulted in contamination of infant formula.

[1098] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

10.21 European Council Directive on Medicinal Products for Human Use (2004/27/EC)

The Health (Fluoridation of Water Supplies) Act, 1960 is a public health measure in the field of preventative dentistry that legislates for fluoride or fluoride products to be added to waters to prevent disease in human beings. [1099]

The legal definition of a medicinal product in the European Union (Codified Pharmaceutical Directive 2004/27/EC, Article 1.2) is "any substance or combination of substances presented as having properties for treating or preventing disease in human beings; or which may be used in or administered to human beings either with a view to restoring, correcting or modifying physiological functions by exerting a pharmacological, immunological or metabolic action."

Under Article 1(3) a substance is defined as any matter irrespective of origin which may include-chemical, e.g. elements (i.e. Fluoride), naturally occurring chemical materials and chemical products obtained by chemical change or synthesis (this includes fluoride products including hexafluorosilicic acid & hexafluorosilicictes compounds).

Legal precidence exists[1100] in Europe establishing clearly that fluoridated water is defined as a medicinal product. In 1983, a judge ruled that fluoridated water fell within the Medicines Act 1968, "*Section 130 defines 'medicinal product' and I am satisfied that fluoride in whatever form it is ultimately purchased by the respondents falls within that definition.*"

A judgment given in a European Union (EU) country is to be recognised without special proceedings, unless the recognition is contested. To the author's knowledge the judgement was never contested.

Directive 2004/27/EC outlines that the concepts of harmfulness and therapeutic efficacy can only be examined in relation to each other and have only a relative significance depending on the progress of scientific knowledge and the use for which the medicinal product is intended. The particulars and documents which must accompany an application for marketing authorization for a medicinal product demonstrate that potential risks are outweighed by the therapeutic efficacy of the product.

The Government of Ireland or its agencies have never undertaken risk assessments on the fluoridation products in use in Ireland. This would constitute a flagrant and serious violation of Directive 2001/83/EC.

1099 Source: Circular 6/2007: Code of Practice on Fluoridation of Drinking Water 2007

1100 Lord Jauncey. Opinion of Lord Jauncey in cause Mrs Catherine McColl (A.P) against Strathclyde Regional Council. The Court of Session, Edinburgh, 1983.

The State is required under EU law for the protection of public health to undertake detailed risk assessment and performance of tests and clinical trials including toxicological and pharmacological tests to demonstrate the effectiveness and risks associated with water fluoridation.

No toxicological or pharmacological trials on the fluoridation products have been undertaken by the State. The Government of Ireland is required to undertake a risk benefit analysis of all medical products including artificially fluoridated water before fluoridation becomes public policy. No such risk benefit analysis has been undertaken by the Government of Ireland.

The Government of Ireland, or any member EU state, is required to undertake an environmental impact assessment of the impact of water fluoridation on the environment before any such public policy should commence. No such environmental impact assessment has been undertaken.

According to fundamental principles of medical ethics, it is quite wrong to subject any competent individual to a medical intervention he or she neither needs nor wants. Article 5 of the *European Convention on Human Rights and Biomedicine* States: "*An intervention in the health field may only be carried out after the person concerned has given free and informed consent to it.*" Ergo: Water Fluoridation is an unethical act of mass-medication.

Regardless of the ethics of water fluoridation the State legislated for mandatory fluoridation and remains today the only country within the European community that has a legislative mandate to continue water fluoridation. It is not unreasonable for Irish citizens of the European Union to demand harmonization of Irish policy with European policy for water management that excludes the fluoridation of water.

The Government of Ireland by introducing legislation to improve public health have never demonstrated that fluoridation has been proven to be medically beneficial. In 2000, 55 years after fluoridation began in the USA; the York Review Expert Committee was unable to identify one high-quality study to show that the practice is effective or safe. Likewise the SCHER and NRC reviews have not demonstrated conclusively that fluoridation is safe or beneficial. Both studies concluded similarly that there was limited evidence from epidemiological studies pointing towards adverse health effects following systemic fluoride consumption, e.g., carcinogenicity, developmental neurotoxicity and reproductive toxicity. In addition, it was noted that there were insufficient human risk assessment studies to substantiate that the policy was safe. The onus of responsibility, however, clearly lies with the State to firstly provide sufficient proof that silicafluoride additives are safe before injecting them into the public water supply. It is unlawful, if not a criminal act, for the government of Ireland to continue with such a policy without providing legal standards of proof that the fluoridation product and treated drinking water is safe.

10.22 European Council Directive on Cosmetic Products for Human Use (76/768/EEC)

As the State has never proven fluoridation of drinking water to be beneficial it may argue that artificially fluoridated water is not a medical product. If it is not a medical product therefore it must be a cosmetic product. If the State or manufacturer of the fluoridation product does not make any 'medical claim' (i.e. a claim for a health effect) for their product, then that product (by default) is regarded as a cosmetic product. 'Products for the care of the teeth and the mouth' (Directive 76/768/EEC. Annex I) are regarded as cosmetic rather than medicinal products. A company cannot put a cosmetic product on the market unless it is 'safe'. The company must have documentation attesting to the safety of their product. To my knowledge no such documentation exists.

The definition in the EU of a 'cosmetic product' is '*..any substance or preparation intended to be placed in contact with the various external parts of the human body (epidermis, hair system, nails, lips and external genital organs) or with the teeth and the mucous membranes of the oral cavity with a view exclusively or mainly to cleaning them, perfuming them, changing their appearance and/or correcting body odours and/or protecting them or keeping them in good condition.*' (Article 1 Cosmetics Directive 76/768/EEC). The latter definition could apply to fluoride products added to drinking water as fluoride compounds are added with the aim of protecting teeth, or keeping teeth in good condition. Article 2 of that same Directive specifies that a cosmetic product "*must not cause damage to human health when applied under normal or reasonably foreseeable conditions of use*. "

Currently twenty fluorine compounds are approved as ingredients in cosmetic products and listed in Annex III Part 1 of this Directive, fluorosilicic acid, the product used in Ireland for fluoridation of water, is not included on the list as an approved substance. The Directive outlines how particular attention is needed for long-term safety aspects, since cosmetic products may be used extensively over a large part of the human lifespan. Therefore, the safety-in use of cosmetic products has been established in Europe by controlling the ingredients, their chemical structures, toxicity profiles, and exposure patterns [93/35/EEC[1101]].

In Annex III part 1 of the amended Council Directive 76/768/EEC on the approximation of the laws of the member states relating to cosmetic products, 20 fluoride compounds are listed which may be used in oral hygiene products up to a maximum concentration in the finished products of 0.15% (1500 ppm), calculated as fluorine. Fluorosilicic acid or hydrofluorosilicic acid (H2SiF6), the compound used for drinking water fluoridation in Ireland, is not a listed compound and is therefore not permitted for use in oral hygiene products. Fluoridated drinking water may be classified as an oral hygiene

[1101] Council Directive 93/35/EEC of 14 June 1993 amending for the sixth time Directive 76/768/EEC on the approximation of the laws of the Member States relating to cosmetic products. *Official Journal L 151, 23/06/1993 p.32*.

drinking product. It is noted that an assessment of the safety for human health of the finished product must be undertaken before placing a cosmetic product on the European market. It is acknowledged that no such assessment has been undertaken for water fluoridation products.[1102] [1103], It has been further demonstrated that water fluoridation can cause damage to human health through dental and skeletal fluorosis.[1104],[110511061107] The only medical or cosmetics claim that can now be applied to water fluoridation within the EU is that defined by SCHER.

SCHER concluded that water fluoridation "*appears to prevent caries, primarily on permanent dentition*" and "*no obvious advantage appears in favour of water fluoridation compared with topical prevention. The continued systemic exposure of fluoride from whatever source is questionable once the permanent teeth have erupted*". It is incredible that after half a century of water fluoridation that neither the NHS York Review nor the most recent SCHER critical assessment could find one scientific study that could conclusively prove that fluoridation of water supplies is an effective public health policy. At present, safety evaluation of cosmetic ingredients is carried out by the SCCP using data obtained from animal studies (*in vivo*), *in vitro* experiments, QSAR (quantitative structure activity relationship) calculations, clinical studies, epidemiological studies and accidents. No such safety studies have been undertaken examining the impact of fluoride or Hexafluorosilicic acid on human health. With the implementation of Directive 2003/15/EC[1108], the need for appropriate clinical tests for the safety evaluation of cosmetic ingredients and products becomes a legal requirement. This is particularly important for fluoride products used for ststemic dosing of public water supplies. In the absence of such tests the Government of Ireland must observe the most appropriate risk preventative policy which is to cease the fluoridation of water supplies immediately.

[1102] Scientific Committee on Health and Environmental Risks, European Commission, Director General Health and Consumers, Critical Review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, May 2010.

[1103] USA National Research Council, Fluoride in Drinking Water: A Scientific Review of EPA's Standards, Committee on Fluoride in Drinking Water, (2006)

[1104] Scientific Committee on Health and Environmental Risks, European Commission, Director General Health and Consumers, Critical Review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, May 2010.

[1105] USA National Research Council, Fluoride in Drinking Water: A Scientific Review of EPA's Standards, Committee on Fluoride in Drinking Water, (2006)

[1106] NHS Centre for review and Dissemination, A systematic review of water fluoridation, The University of York, Report 18

[1107] Opinion of the European Commission Scientific Committee on Cosmetic Products and non-food products (SCCNFP intended for consumers) concerning the Safety of Fluorine Compounds in Oral Hygiene Products for Children under the age of 6 years. SCCNFP/0653/03, Final, June 2003

[1108] Directive 2003/15/EC of the European Parliament and of the Council of 27 February 2003 amending Council Directive 76/768/EEC on the approximation of the laws of the Member States relating to cosmetic products. *Official Journal L66, 11/03/2003 p.26*

10.23 Treaty of Europe- Implications for Environment

The preservation, protection and improvement of the quality of the environment, including the conservation of natural habitats and of wild fauna and flora, are an essential objective pursued by the Community, as stated in the Treaty of Europe (TEU).

Article 3.3 TEU states that the Union "shall work for the sustainable development of Europe and introduces specific reference to the promotion of scientific and technological progress, which could also have implications for environmental protection.

In its relations with the wider world, the EU shall uphold and promote its values and contribute to the "sustainable development of the Earth." To this end, the Union will work towards the adoption of "international measures to preserve and improve the quality of the environment and the sustainable management of global natural resources" (Art. 21.2 TEU).

Article 174 of the Treaty provides that Community policy on the environment is to contribute to the preservation, protection and improvement of the quality of the environment, the protection of human health and the prudent and rational utilisation of natural resources and that it is to be based on the precautionary principle. According to the Treaty of European Union, the precautionary principle may only be invoked when the following **three preliminary conditions** are met:

- identification of potentially adverse effects;
- evaluation of the scientific data available;
- the extent of scientific uncertainty.

It is accepted that water fluoridation causes a toxic effect on the human body most commonly represented by dental fluorosis. Many other potentially adverse health and environmental effects have been reported and presented in this report. The FSA have reported that the effects of formula feeding on infant health have not been investigated in Ireland.[1109] In addition the EU Scientific Committee on Health and Environmental Risks has reported[1110] that the toxicology of hexafluorosilicic acid and hexafluorosilicictes compounds are incompletely investigated.

The NHS YORK Review, U.S. NRC Scientific Review (2005) and E.U SCHER Critical Review (2010) highlighted the risks associated with water fluoridation including the immediate and delayed effects on health, the cumulative toxic effects of the pollutant, the sensitivity of certain consumers to this compound and the lack of credible data to prove conclusively that fluoridation of water supplies is safe. Each scientific review highlighted the lack of adequate risk

[1109] Food Safety Authority of Ireland, Recommendations for a national infant feeding policy 1999.

[1110] Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, Scientific Committee on Health and Environmental Risks, Director General for Health & Consumers (2010

assessments and in particular the risk posed to infants and certain sensitive sub-groups of the population. In each review the presence of risk was identified. Further risks and adverse effects have been identified and discussed in this report.

In accordance with EU and international law the Government of Ireland has a legal duty of obligation to comply with the precautionary principle. In light of recent scientific developments the authorities must act to protect the consumer and the environment from unnecessary risk. The authorities must end the policy of water fluoridation immediately or prove beyond any doubt the absence of danger to consumers.

10.24 Treaty on the Functioning of the European Union

While the preservation, protection and improvement of the quality of the environment, including the conservation of natural habitats and of wild fauna and flora, are an essential objective pursued by the Community, as stated in the Treaty of Europe (TEU), the Treaty on the Functioning of the European Union (TFEU) reaffirms the EU's commitment to regional and worldwide environmental protection.

The EU's policy in relation to the environment is subject to shared competence between the EU and member states (see Article 4(2)(e), TFEU). Shared competence means that member states can exercise their competence to the extent that the Union has not exercised its competence or has decided to stop exercising it. Thus, member states should now be able to pass and implement national fisheries' management measures in areas that are not specifically covered by EU legislation, This will be extremely significant for law and policy development in a number of different areas, particularly the interrelationship between environmental law, fisheries protection and the potential reduction of specific pollutants harmful to fisheries and the environment.

10.25 The Maastricht Treaty - Implications for the Environment

The Maastricht Treaty of EU entered into force in 1993 and strengthened the EC's commitment to environmental protection. It included as a principal objective the promotion of sustainable growth respecting the environment (Art. 2). Central themes of the Maastricht Treaty are to support sustainable development and to take measures to curb environmental degradation.

The Maastricht Treaty includes among the activities of the Union a policy in the sphere of the environment specifying that this policy must aim at a high level of protection, be based on the precautionary principle and that environmental protection requirements must be integrated into the definition and implementation of other EU policies.

10.26 The Treaty of Lisbon - Implications for the Environment

The Treaty of Lisbon entered into force on 1st December 2009. While the main principles and objectives of EU environmental policy remain largely unchanged, the Treaty reinforces the EU's commitment to sustainable development. The Treaty of Lisbon reinforces the Union's pledge to pursue this objective both within and beyond its borders. The Lisbon Treaty sets out explicitly the EU's values and objectives – and among these are improving the quality of the environment.

The Treaty's new provisions on public participation and engagement are relevant. The Lisbon Treaty provides for the possibility of a 'citizens' initiative', an opportunity that is particularly relevant to environmental interest groups and anti-fluoridation groups. This report may be seen as part of this new right of initiative to raise local and national environmental issues in the context of European debate.

In accordance with EU and international law the Government of Ireland has a legal duty of obligation to comply with the precautionary principle. The authorities must act to protect the environment from unnecessary risk. The Government of Ireland cannot comply with the requirements of these international treaties by continuing to support a policy of water fluoridation. The State has not assessed the environmental impacts of this policy. The fluoridation of water supplies has resulted in significant quantities of a toxic enzymatic poison being released into the natural environment without any risk assessment on ecological impacts. The pollutant fluoride is known to be harmful to protected freshwater species of fish and their habitats. It is known to have significant adverse effects on the environment.

10.27 Council of Europe Convention for the Protection of Human Rights and Fundamental Freedoms

The European Convention on Human Rights and Fundamental Freedoms sets forth a number of fundamental rights and freedoms for the protection of the individual including security of person, health and life. The Convention has legal force in Ireland as it was enacted by the Oireachtas, with the publication of the European Convention on Human Rights Act 2003.

It is accepted that Fluoride is a dangerous toxin that can cause serious harm to the individual through over-exposure by ingesting fluoridated water and fluoride contaminated foods or beverages that may lead to bio-accumulation of fluoride in the human body and bllod plasma with unforeseen medical consequences.

It has been identified that high-risk groups of the population include infants, individuals with diabetes or cardiovascular disease and communities living in geographic areas with naturally low calcium and magnesium levels in drinking water.

As a result of the policy of water fluoridation of all public water supplies, healthcare professionals and care-providers have no access to non-fluoridated water. Consumers are forced to consume drinking water contaminated with a cumulative toxin that may cause actual bodily harm to their person and may have unforeseen toxicological and genetic implications for future generations.

Article 5 of the European Convention of Human Rights and Fundamental Freedoms establishes right of the individual to security of person. The Government of Ireland, by pursuing a policy that facilities the injection of known harmful substances into the public water supply that may cause harm to individuals, is violating the provisions of this Convention.

10.28 Council of Europe Convention for the Protection of Human Rights and Biomedicine 1997

The Council of Europe which represents all the democratic countries of Europe established the *Convention* for the Protection of *Human Rights and Biomedicine* with the aim of achieving a greater unity between its members states by pursuing a basic set of principles on human rights and fundamental freedoms for all EU citizens with regard to the misuse of biological or medical science.

The Convention on Human Rights and Biomedicine is one of the most important international agreements on bioethics from the point of view of international policy and law. In 1997, the Council of Europe's Convention on Human Rights and Biomedicine was signed by 21 member states in Oviedo, Spain. Its emphasis on the principles of protection of individual human rights, individual health needs, proper risk assessment and professional care and compliance with international law can be clearly observed in the following articles of the convention:

Article 1	The purpose and object of the Convention is defined in Article 1guaranteeing everyone, without discrimination, respect for their integrity and other rights and fundamental freedoms with regard to the application of biology and medicine.
Article 2	The rights of the individual are enshrined in Article 2 setting down the principle that the rights of the individual shall prevail over the sole interest of society or science.
Article 3	Establishes the requirement of State to take into account the individual health needs of citizens and provide equitable access to healthcare of appropriate quality.
Article 4	Establishes the requirement of the State to ensure professional due diligence, risk assessment and compliance with international best practice, operating standards and law.
Article 5	Establishes the legal requirement that any intervention in the health field may only be carried out after the individual has given free and informed consent. This may only be facilitated subject to the State providing information as to the purpose and nature of the intervention as well as on its consequences and risks. Under Article 5 the person concerned may freely withdraw consent at any time.
Article 6	Provides for the protection of persons not able to consent with particular reference to infants and establishes that authorisation by the child's parent or guardian is required to allow any health intervention.
Article 24	Establishes that any person who has suffered undue damage resulting from an intervention is entitled to fair compensation according to the conditions and procedures prescribed by law.

Since its inception in 1997, thirty five European countries have ratified the convention including Croatia, the Czech Republic, Estonia, Finland, France, Hungary, Iceland, Italy, Latvia, Lithuania, Luxembourg, Moldova, the Netherlands, Norway, Poland, Portugal, Romania, Spain, Sweden, Switzerland, the former Yugoslav Republic of Macedonia, Turkey, Bulgaria, Cyprus, Denmark, Greece, Slovakia, Albania, Georgia, Bosnia and Herzegovina, Montenegro, Serbia, Slovenia and Ukraine.

Ireland is one of the few remaining countries that has yet to ratify the Convention on Human Rights and Biomedicine. The Council of Europe Convention is binding on the Government of Ireland as a member of the Council of Europe, however, it does not form part of Irish law as the Government of Ireland did not sign or ratify the Convention and it was not enacted by the Houses of the Oireachtas.

It is clear that any mandatory national legislative policy such as water fluoridation of public drinking water supplies for medicinal needs as enacted in the Republic of Ireland is in violation of the convention on Human Rights and Biomedicine. Legal precedence has already established that water fluoridation is a medicinal product. It is further acknowledged that the Health (Fluoridation of Water Supplies) Act, 1960 is a public health measure in the field of preventative dentistry[1111] that legislates for fluoride or fluoride products to be added to waters to prevent disease in human beings.

The policy of injecting known toxic silicafluorides compounds into the public water supplies without adequate risk assessment, legal or professional due diligence, environmental impact assessment, toxicological and pharmacological tests and human risk assessment studies to prove beyond doubt that the chemical process is safe, is unlawful. The onus of responsibility, however, clearly lies with the Government of Ireland or its agencies to firstly provide sufficient proof that silicafluoride additives are safe **before** injecting them into the public water supply.

The NHS York Review (2000) demonstrated that water fluoridation is not safe. This view has been supported by the NRC review (2005), the SCHER review (2010) as well as other independent scientific reviews across Europe, USA, Australia and Canada.

This report further documents the numerous known and potential human health risks as well as the environmental and ecological consequences of water fluoridation. Ireland remains the only country within Europe with a mandated legislative policy on water fluoridation.

[1111] Source: Circular 6/2007: Code of Practice on Fluoridation of Drinking Water 2007

10.29 Council of Europe Convention on the Protection of the Environment through Criminal Law

The Council of Europe, which represents all the democratic countries of Europe, established the *Convention On The Protection Of The Environment Through Criminal Law* with the aim of achieving a greater unity between its members states by pursuing a basic set of principles for the protection of life and health of all EU citizens through protection of the environment by establishing criminal offences or other sanctions within domestic law for environmental violations.

The *Convention on the Protection of the Environment through Criminal Law* (Strasbourg, November 4, 1998) is the first international convention to criminalise acts causing or likely to cause environmental damage and it is a very important event in the development of environmental criminal law. The Convention has been concluded at the Council of Europe but it is also open to signature for non-member states.

The Convention is structured in four sections and holds legislative obligations for the member states. The first section, is dedicated to the "use of terms", it indicates that for the purposes of this Convention: a)"unlawful" means infringing a law, an administrative regulation or a decision taken by a competent authority, aiming at the protection of the environment; b)"water" means all kinds of groundwater and surface water including the water of lakes, rivers, oceans and seas (Article 1). Section II makes reference to "(m)easures taken at national level" (4) ; Section III to "(m)easures to be taken at international level" (5); and, finally, Section IV to "(f)inal clauses" (6) .

Article 3.1 of the Convention indicates that "(e)ach Party shall adopt such appropriate measures as may be necessary to establish as criminal offence under its domestic law, when committed with negligence. The Convention typifies risk misconducts. Risk is inevitable in industrialized societies. However, it is necessary that policies such as chemical treatment of public water supplies must be developed in a framework of adequate risk assessment and environmental security. In this sense, an environmental risk criminal prohibition may be necessary because the environment requires an anticipated protection and because environmental damages may be the consequence of a reiteration of accumulative acts which, at the individual level would, be harmless. This option is corroborated not only by the preventative finality of criminal law, but also by the precautionary principle of international law of the environment.

Principle 15 of The Rio Declaration indicates that "(i)n order to protect the environment, the precautionary approach shall be widely applied by states according to their capabilities. Where there are threats of environmental damage, lack of full scientific certainty shall not be used as a reason for continuing the policy or for postponing cost-effective measures to prevent risk to the consumers of the environment.

It has been clearly acknowledged that there is a lack of scientific risk assessment to prove without doubt that the policy of water fluoridation is safe. It has been acknowledged that certain health risks and environmental impacts are associated with water fluoridation and that it may act as a cumulative toxin in the human body and environment. It has been demonstrated that fluoride is a harmful ecotoxin in water and is released into the environment at concentrations that are known to be harmful to fisheries. Fluoride has been demonstrated to be an endocrine disruptor in the freshwater environment and levels as low as 0.25mg F/l have been scientifically demonstrated to be harmful to some species of fish.

It is plausible therefore that the policy of water fluoridation has had significant negative impact on the quality and health of inland fisheries in Ireland. No risk or environmental assessment has ever been undertaken to examine such a scenario by the authorities in Ireland.

With respect to criminal law, the convention typifies intentional and negligent offences. Article 4 of the Convention establishes criminal offences punishable by criminal sanctions under law when committed intentionally or with negligence. The sanction available shall include imprisonment, corporative liability and pecuniary sanctions and may include reinstatement of the environment.

This report has been submitted to the Government of Ireland and its regulatory agencies including the Health Service Executive, the Environmental Protection Agency, Inland Fisheries, the Food Safety Authority and the Chief Scientific Officer of the Republic of Ireland. This process facilitates the provision of detailed information that may allow for informed decisions. It is likely that if the Government of Ireland or its agencies were to decide to continue with the policy of fluoridation of drinking water, it could result in criminal sanctions on persons on whose behalf the offence was committed or their representatives.

10.30 European Community Environmental Policies

In total, there are six environmental action programmes of the European Communities on the environment namely which have been approved by the Council of the European Communities and the representatives of the Governments of the member states including Ireland.

They cover the period from 1973 to 2012 namely:

- First Environment Action Programme (1973-1977)
- Second Environment Action Programme (1978-1982)
- Third Environment Action Programme (1982-1986)
- Fourth Environment Action Programme (1987-1992)
- Fifth Environment Action Programme (1993-1999)
- Sixth Environment Action Programme (2002-2012)

The First Environment Action Programme (EAP) emphasized the need for a comprehensive assessment of the impacts of other policies (such as the policy of water fluoridation), in an effort to avoid damaging activities and devoted most of its attention to water protection and waste. The second EAP devoted much of its attention to nature protection. The third and fourth EAPs emphasized the potential risks and benefits of environmental policies and the need for waste avoidance and efficient resource use for environmental protection.

Part of this integrated approach was to reduce material inputs and to close cycles, so that waste streams could be minimised. Furthermore, pollution control was to systematically control all environmental media (water, air and soil) and involve an evaluation of the problem causing substances.

The fifth EAP focused on sustainable development, management of water resources, protection of nature and biodiversity and improved resource management as well as undertaking cost benefit analysis of policies with regard to the environment.

The sixth EAP establishes a 10 year framework for Community action on the environment up to 2012. Nature and biodiversity protection, pollution of freshwater, better waste management and research on the socio-economic value of biodiversity and ecosystem services were key pillars of the fifth and sixth EAPs.

The sixth EAP set out the EU's international commitments alongside its wider environmental objectives with the aim of promoting the consideration of environmental issues in policy development, supporting the integration of environmental considerations in sectoral debates and promoting sustainable use of the seas and conserving marine ecosystems.

One of the key considerations of the sixth EAP addresses so-called persistent environmental problems. Fluoride emissions from wastewater treatment of fluoridated drinking water supplies results in persistent environmental pollution of soil and water resources.

Overall the main objective of all the aforementioned programmes directed that the best environmental policy consists of preventing the creation of pollution or nuisances at source, rather than subsequently trying to counteract their effects. The action programmes affirm the need to take effects on the environment into account at the earliest possible stage in all the technical planning and decision-making processes; whereas to that end, they provide for the implementation of procedures to evaluate such effects.

It is clear that none of the objectives of the various Environmental Action Programmes covering the period 1973-2012 were ever applied to examination of the policy of water fluoridation in Ireland or its implications for protection of freshwater resources, biodiversity and minimizing waste emissions by preventing the creation of pollution nuisances at source.

It is clear that the consequential damages of water fluoridation to the environment were never examined when this policy was first introduced in the 1960's. It is obvious that policy or decision-makers at all levels continued to show a complete lack of appreciation for the impact of water fluoridation on the environment in subsequent years.

There is a need to change the attitudes of decision-makers at all levels so that they understand the toxicity of fluoride and silicafluoride compounds in the environment as well as their potential health risks for consumers, in order to protect the environment and to avoid future problems. Policy decisions must be based on the precautionary principle as defined in the European Treaty. It is clear that the policy of Water fluoridation violates the aims and objectives of each of the six European Environmental Action Programmes.

10.31 Rio Declaration on Environment and Development

The Rio Declaration was produced at the 1992 United Nations Conference on Environment and Development (UNCED), informally known as the Earth Summit. The Rio Declaration consists of 27 principles intended to guide environmental and development issues around the world. The Rio Declaration built on the previous Declaration of the United Nations Conference on the Human Environment which was adopted in Stockholm in 1972.

The Rio Declaration was signed by the Government of Ireland in 1992.

Principle 13 of the Rio Declaration requires signatory states to develop national law regarding liability and compensation for the victims of pollution and other environmental damage. It further requires that states shall also cooperate in an expeditious and more determined manner to develop further international law regarding liability and compensation for adverse effects of environmental damage caused by activities within their jurisdiction.

Principle 15 of the Rio Declaration establishes that signatory states shall apply the precautionary approach to protect the environment where there are threats of environmental damage and lack of full scientific certainty.

Principle 17 of the Rio Declaration requires signatory states to undertake an environmental impact assessment for activities that are likely to have a significant adverse impact on the environment.

The policy of water fluoridation has been demonstrated to cause actual bodily harm to individuals. Dental fluorosis is the most common toxic impact of water fluoridation.

It is internationally accepted that in developed countries where topical application of fluorides through toothpaste is widely available, that water fluoridation is a major contributor to the development of dental fluorosis.

It is accepted that all infants bottle-fed using milk formula reconstituted with fluoridated water are at a high risk of developing dental fluorosis.

In some cases it may result in moderate and severe dental fluorosis requiring significant dental surgery.

It is accepted that chronic ingestion of fluoride at concentration >1mg/l is a significant risk in developing skeletal fluorosis as well as musculoskeletal disorders.

It is accepted that individuals living in communities with low calcium and magnesium levels in drinking water are at higher risk of developing these and other serious diseases due to the interactive properties of fluoride added to drinking water.

It is accepted that individuals with diabetes or renal complications are at higher risk of developing these diseases due to impaired kidney function.

By continuing to pursue the policy of water fluoridation in light of this report the State is failing to protect its citizens from unnecessary harm and also violating the principles of the Rio Declaration.

It is clear that the public health authorities in Ireland have not informed consumers of the risks associated with water fluoridation.

It is accepted that adequate risk assessments to protect the consumer from the chemical hazards of fluoride or interactive fluoride compounds, such as aluminium fluoride, have not been undertaken.

It is accepted that despite scientific evidence demonstrating negative environmental impacts the State has not undertaken an environmental impact assessment of the risk posed by water fluoridation on freshwater of terrestrial ecosystems.

It is clear therefore that by continuing with the policy of water fluoridation in light of this report the Government of Ireland would be in clear violation of the principle of the Rio Declaration.

10.32 United Nations Convention on Biological Diversity

The Convention on Biological Diversity (CBD) entered into force on 29th December 1993 and was ratified by Ireland in 1996. The CBD is a global agreement which sets out commitments for maintaining the diversity and functionality of the world's ecosystems as countries proceed with economic development.

The Convention covers all ecosystems, species and genetic resources and recognises that they must be used for the benefit of humanity, but insists that this should be done in a way and at a rate that does not lead to the long-term decline of biological diversity. It underlines the importance of the precautionary principle where, if there is a threat of significant loss of biological diversity, lack of scientific certainty should not be used as a reason for postponing measures to avoid or minimise such a threat. The Convention requires parties to integrate, as far as possible and as appropriate, the conservation and sustainable use of biological diversity into relevant sectoral or cross-sectoral plans and programmes. The Convention requires parties to take measures for the conservation and sustainable use of biological diversity.

As a contracting party to the Convention, Ireland is committed to measures to conserve biodiversity including the conservation of ecosystems, habitats and species in their natural surroundings, both inside and outside protected areas, to impact assessment, identification and monitoring and sustainable use of ecosystems, species and other biological resources and to identify processes and categories of activities which have, or are likely to have, significant adverse impacts on the conservation and sustainable use of biological diversity and to monitor their effects through sampling and other techniques. Each member state is further required to introduce appropriate arrangements to ensure that the environmental consequences of its programmes and policies, that are likely to have significant adverse impacts on biological diversity, are duly taken into account.

The Government of Ireland cannot comply with the requirements of this international treaty by continuing to support the policy of water fluoridation. The State has not assessed the environmental impacts of this policy. The water fluoridation policy has resulted in significant quantities of a toxic enzymatic poison to be released into the natural environment without any risk assessment on its likely impacts. The pollutant fluoride is known to be harmful to protected freshwater species of fish and their habitats. It is known to have significant adverse effects on the environment.

In accordance with the Convention countries are obliged to prepare national strategies or plans in order to achieve the Convention's goals and objectives. One of the key targets set by Ireland in their Action for Biodiversity Plan 2011-2016 is to reduce pollutant pressures on terrestrial and freshwater ecosystems. Ireland cannot comply with this target by continuing to allow a known toxin to be discharged into the environment as a consequence of its policy of fluoridation of drinking water supplies.

11.0 COST OF FLUORIDATION OF WATER SUPPLIES

According to Oireachtas records the direct cost of fluoridation to the HSE in 2010 was €4.78 million.[1112] This figure does not include the fixed costs for equipment, further management and supervision costs associated with fluoridation including auditing costs for the 246 fluoridation plants throughout the country (total cost of €4.1million), upkeep and maintenance of plant and machinery, insurance and the financial costs of environmental monitoring and regulatory assessment.

An example of the overall operational costs of water fluoridation may be determined by examination of costs associated with other countries or large cities. Due to Ireland's unique position within Europe, as the only member state that has a policy of water fluoridation, no figures are available on a European basis. It is known, however, that New York City fluoridates its drinking water supplies at a cost of $34 million per annum. For further information on some of the health implications of water fluoridation on consumers in New York please review cancer studies undertaken by Harvard University. The cost of chemicals has been estimated to be $9million and $25million is spent on operational costs.[1113] While New York City may provide water for a population of 8million people it has very few water fluoridation plants in comparison to Ireland. It is expected therefore that the operational costs in comparison would be considerably higher for Ireland when taking manpower and equipment into consideration while chemical costs would be less. A plausible overall figure based on the current scenario would provide rough overall operational costs of €40million per annum including the cost of chemicals.

Additional operational costs would be associated with wastewater treatment to treat the pollutant fluoride present in wastewaters. Further costs would be associated with reducing the pollutant load in sewage sludge as required by EU Directives, though it is likely that this is not being complied with currently.

Additional costs are associated with the policy of fluoridation by the provision of various state-appointed dental bodies including the *Irish Expert Body on Fluorides and Health*, the Dental Council, the Post Graduate Medical and Dental Board, the Dublin Dental Hospital Board and the Cork University Dental School and Hospital Board, all of which are funded through the State Exchequer. While they have received significant funding for research from the State to date, no research has to our knowledge been undertaken on the most important consideration; that is the toxicology of silicafluorides and their co-toxicity within the human body with other compounds or substances.

It is reasonable to suggest that the work of the *Expert Body on Fluorides and Health*, who last published a report in 2002, has been overtaken by comprehensive assessments provided by the U.S. National Research Council and the European Commission's Scientific Committee on Health and

[1112] Deputy Róisín Shortall, Minister of State at the Department of Health and Children September 2011

[1113] Natural News April 20th 2011

Environmental Risks and the U.S. Environmental Protection Agency. The body of work undertaken by these expert international bodies provides relatively accurate and up-to-date, though incomplete, scientific information on fluoridation and should therefore negate the necessity for the State to continue to fund any national bodies in our current economic difficulties. It is noted that questions remain regarding the value for money, accuracy and transparency of the 2002 report given the published statements by the Chairman of the NHS YORK Review[1114] regarding widespread misrepresentation of the YORK scientific committee's findings.

Naturally if the policy of fluoridation is to cease there would be no requirement for this body and, at a minimum, financial costs to the Government would decrease providing further savings to the Exchequer.

11.1 The Potential Human Cost of Fluoridation

The potential human costs of water fluoridation are incalculable as inadequate toxicological risk assessments have been undertaken on synthetic based inorganic fluoride and its associated silica-fluoride compounds that are used in Ireland for water fluoridation. What is absolutely certain is that over the period of the past forty years consumers of fluoridated drinking water have been over-exposed to a dangerous and highly reactive substance that acts as an enzymatic poison on the human body.

There is no doubt from the data presented in this report that fluoridation of drinking water has contributed to the overall health burden in Ireland in recent years and this is likely to increase as long at the policy continues. Fluoridation of drinking water supplies while regulated by outdated national legislation is in breach of over thirty eight EU Directives and international treaties, each one mandating that the State must protect consumers, the environment and biodiversity. It is clear that the State has previously failed in its duty of care to protect the most vulnerable in society by ensuring that a 'one size fits all' approach with fluoridation of drinking water supplies exposes the entire population from newborn infants to high-risk individuals, such as diabetics, to multiples of the recommended daily tolerable intake of a dangerous substance.

This has been tolerated by state agencies who have failed in their duty to undertake adequate risk assessments or caution parents/care providers of the known short- and long-term health risks, particularly for bottle-fed infants. Consequently approximately 40% of the population of Ireland may now suffer from the effects of clinical fluoride over-exposure resulting in dental, skeletal and mental fluorosis. Fluorosis, dental or otherwise is a toxic effect that is consistent with prevailing risk assessment definitions of adverse health effects.

The estimate of costs provided above does not include the costs to the

[1114] NHS Centre for review and Dissemination, A systematic Review of water fluoridation, The University of York, Report 18

health service or society of adverse health effects from exposure of the human population to fluoride, in particular sensitive sub-groups. There are clearly enormous cost implications associated with fluoride over-exposure from consuming fluoridated drinking water. Indeed, there are much wider costs implications as the development of many diseases associated with fluoride over-exposure may impact on a person's ability to work. For example a report[1115] by the Irish Thoracic Society found there were almost 70 working days lost annually per recipient of illness benefit related to sarcoidosis. It is not unlikely that similar figures may be found for other major diseases associated with fluoride exposure. The potential financial burden therefore on the health system in Ireland and the economy as a whole is unquantifiable.

When one examines the information provided in this report on the medical and environmental implications of fluoride it is not implausible to suggest that the healthcare and social costs would be in the order of multiples of the estimated operational costs, and could easily approach or exceed one billion euros per annum, taking into consideration that over 750,000 people suffer from neurological conditions while upwards of 400,000 may suffer from diabetes. In addition, Ireland has the highest level of cardiovascular disease in Western Europe as well as epilepsy, osteoporosis, hip fractures and sarcoidosis. Add to this the extradinoraly high level of individuals in Ireland who suffer musculoskeletal pain, the most widely reported condition for individuals over 50 years of age with a prevalence of 40%.[1116] It is further estimated that there are approximately 585,000 people in Ireland who suffer from chronic pain representing 36% of all households in Ireland. It is also known that up to 50% of Type ii diabetes sufferers may develop chronic pain associated with nerve damage.[1117] It is now known that exposure to fluoride is a major risk factor associated with developing many chronic pain conditions. Add to this the estimated 45,000 individuals who may unknowingly be hypersensitive to fluoride and the 40% of the population with dental fluorosis as well as the unknown levels of skeletal or mental fluorosis[1118] within the community and the potential human and social costs for associated risks from water fluoridation become truly astounding.

It is important to highlight that the NRC recommended for health risk management strategies that all dental fluorosis patients with signs of renal impairment should have radiographic examinations of the skeletal system to ensure that they were not over-exposed to fluoride at levels that would contribute to long-term irreparable skeletal bone disorders.

The NRC reported that fluoride exposure may increase the severity of some types of diabetes and advised extreme caution should be used for patients on hemodialysis. If the Department of Health were to follow these recommendations, it would place an enormous financial cost and

[1115] Brennan NJ, McCormack S, O'Connor T. The INHALE Report: Irish Thoracic Society; February 2008.
[1116] Multimorbidity and Disability in the Older Population of Ireland, Longitudinal Study of Ageing (TILDA),2011.
[1117] Murphy, P. Neuropathic Pain, Chronic Pain, Oct 2008, Health Supplement Irish Indepenent Newspapers.
[1118] Mental Fluorosis: the impact of chronic fluoride exposure on normal brain function.

unmanageable burden on the health services at a time when they are already overstretched. It is quite clear given the current crisis in healthcare that the health system would not be able to provide this level of preventative care to the citizens of Ireland. For this reason alone, the only practicable preventative care that the State may undertake is to end the policy of water fluoridation immediately. Separate from the healthcare costs is the cost of environmental pollution to natural resources including inland fisheries and angling tourism, which is as a direct consequence of fluoride emissions from wastewater treatment plants and needs to be examined. The financial cost to the health and sustainability of this industry, and its dependent ecosystem, from fluoride contamination over the past forty years would be almost impossible to estimate.

The freshwater fisheries sector has seen an enormous decline in recent decades and while other factors may also be associated with this, fluoride is now known to be harmful to protected fisheries even at low concentrations. It is plausible therefore that those anthropogenic fluoride emissions may be a significant contributory factor in the decline of Ireland's inland fisheries resource during this period. This scenario, has to my knowledge, never been examined in Ireland.

The costs to the food sector also need to be critically examined as any food processed or prepared with fluoridated water is contaminated unnecessarily by fluoride. This will result in possible hypersensitivity reactions by certain sensitive consumers to certain food products produced in this country. The contamination of foodstuffs presents a unique problem to the marketing image for agriculture or food exports from Ireland as fluoridated water contaminates any product that it is in contact with. As consumers become aware of the importance of monitoring their total dietary intake of fluoride the most health conscientious consumers may avoid any produce produced in or exported from Ireland.

Separate to the cost to the consumers, health, environment, nature and food production, the financial cost of assurance in relation to undertaking and completing the extensive scientific and medical studies recommended by international and European technical scientific committees (addressed later in this report) to examine and quantify the risks to human health of water fluoridation and silicafluoride compounds is conservatively expected to be in the order of tens if not hundreds of millions of euro.

Without adhering to the basic principles of risk management it is clear that, subject to the Government of Ireland continuing to support the policy of mandatory fluoridation of public water supplies, such action will undoubtedly be challenged in a Court of Law. Good governance should aim to prevent this. The Government of Ireland must adhere to the precautionary principle of management as mandated by their commitment to various United Nation and European Legal Treaties. From the evidence examined in this report there may be further significant human, social and economic costs to the HSE and the consumer due to biological risks of fluoride in drinking water. Comprehensive toxicological studies have been recommended for many years in order to accurately assess the likely public health risks. To my

knowledge, none of these assessment have been undertaken. It is perhaps easier for the State to ignore scientific recommendations in order not to identity risk so as to minimise their exposure to potential liability. The opposite is the case. The longer the State continues with this policy the more culpable they become. One would hope that as a nation we would have learned from our recent past in our failure to protect children and the most vulnerable in our society.

Based on the lack of adequate toxicological risk assessment, as noted by SCHER and the NRC, it may prove legally difficult if not impossible to implement a charge on consumers for drinking water where they insist on being provided with non-fluoridated water, or demand a full risk assessment of the fluoridation policy in line with the recommendation of the NRC and SCHER (outlined elsewhere in this report). This will be particularly challenging for the State where some consumers and sensitive sub-groups of the population are burdened with the additional cost of removing fluoride from their drinking water.

It should also be noted that most of the work on public water supplies including maintenance and upkeep of fluoridation plants is borne by local authorities who remain under enormous financial pressures with limited labour resources. Continuing with the policy of fluoridation in effect requires local government to continue with burdensome procedures and inefficient operations in stark opposition to the most recent evidence-based observations of European scientific committees. In the current economic climate there cannot be any justification for continuing with such excessive and unjustifiable practices.

The regulatory state is built on the idea that it is the Government's role to protect the public from hazards. Where real and potential hazards have been identified, the Government and their agencies must create regulations that enforce a precautionary approach that is based on scientific knowledge or, where there is a lack of such information, provide for a significant safety buffer to guarantee protection. At the same time, the Government has an obligation to scrutinize existing regulations to evaluate their likely public benefit, either associated with direct impact or the averted potential for harm, as well as their potential burdens.

The scientific process of risk analysis and characterization is intended to be conducted without regard for political concerns. Both the NRC and SCHER scientific committees identified potential risks and the requirement for further urgent risk analysis on the fluoridation of drinking water supplies. The decision not to pursue with such risk analysis can be deemed political risk management, taking into account issues like costs, equity, feasibility, political will and societal concerns. It also has clear legal ramifications.

Agencies that undertake scientific review and analysis of regulatory information do so for a variety of reasons but in particular to protect public interest and public health. There is broad agreement that regulatory decisions about the environment, safety and health should be based on evidence and transparency.

The evidence, as examined by SCHER, demonstrates that there is no continued benefit to the continuation of fluoridation and the concerns raised by the York, SCHER and the NRC scientific committees clearly demonstrated the potential human health risks associated with adding untested substances to drinking water. The law and common sence should prevail and demand that no such substances be added to public water supplies without adequate proof of safety to humans and the environment.

The Government must therefore consider the likely costs, benefits and risks associated with fluoridation of water supplies, as addressed in this report, and determine whether the mandatory policy of fluoridation needs to be revised in light of the overwhelming evidence which convincingly shows that fluoridation is neither safe, effective nor necessary given the widespread sale and use of fluoridated toothpaste in Ireland.

It is notable that this work has most recently been undertaken by the European Commission, through the scientific committee of SCHER, who clearly advised the Commission and its member states that there are more risks than benefits associated with water fluoridation.

12.0 Governmental Risk Management

It is clear that in the recent past the Government has failed to protect the interests of citizens in Ireland in many areas from protection of children to economic security. When the Government of Ireland undertakes mandatory artificial fluoridation of drinking water supplies that affect every consumer, regardless of their individual health considerations, it has ethical and legal responsibilities to ensure that it has undertaken rigorous scientific examination of the risks of fluoridation products, for the protection and safety of all its citizens. It is clear that state agencies have failed to protect consumers from the risks posed by water fluoridation. At the most basic level, consider the following: fluoride is added to water so that it may make contact with teeth in an attempt to reduce the incidence of dental disease. It is now known that upwards of 40% of the population may suffer from dental fluorosis, a condition demonstrating chronic long-term biological poisoning where the level of fluoride in blood plasma, as a consequence of ingesting fluoridated drinking water, is so high that it causes visible physical structural damage to people's teeth. One can only imagine, regardless of the evidence provided in this report, how this highly reactive toxin may be affecting other organs and the general physical and mental wellbeing of the wider population.

Going forward and taking into consideration the Government of Irelands legal duty of care and requirement for due diligence, the State must ensure that all adequate measures have been taken to inform the consumer of potential health risks and that the regulatory authorities urgently implement adequate risk assessment of the fluoridation products and their co-toxicity with other water additives.

When one examines the most up-to-date peer-reviewed scientific documentation informing best practice in water management, environmental and public health policy in the USA[1119] and in Europe[1120],[1121] the information for the public and consumer is both alarming and disturbing. The information contained in the two most recent and reputable international publications, comprising the most comprehensive scientific assessments undertaken to date on fluoridation of drinking water, provides clear observations, recommendations and conclusions that surely must raise serious concerns for the State and local government. It is important to note in particular, as this has been widely misrepresented by some, that where the observation of inadequate evidence has been applied in certain instances, this does not mean that there is no impact but rather that there exists insufficient scientific assessment to draw a firm conclusion. In orther words,

[1119] U.S. National Research Council, Fluoride in Drinking Water: A Scientific Review of EPA's Standards, Committee on Fluoride in Drinking Water, (2006)

[1120] Scientific Committee on Health and Environmental Risks, European Commission, Director General Health and Consumers, Critical Review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, May 2010.

[1121] NHS Centre for review and Dissemination, A systematic Review of water fluoridation, The University of York, Report 18

there is no evidence to prove it is safe. At a fundamental level the precautionary principle requires that where there is potential risk but insufficient evidence to prove otherwise, one does not proceed in the interests of public health. Unfortunately this has never been applied to water fluoridation.

Within the aforementioned reports, in addition to other peer-reviewed publications, the advantages and disadvantages of fluoridation have been examined in detail. The data has been analyzed by experts and by peer-reviewed national and European scientific committees. Their observations conclude that the safety of water fluoridation remains uncertain and that there remain clear health risks for certain vulnerable sub-sections of the population. A large body of evidence shows that insufficient human studies have been undertaken to protect the public from the many documented risks and dangers associated with systemic dosing of the population through water fluoridation.

Public health is the science and art of disease prevention, prolonging life and promoting health and well-being through organized community effort for the sanitation of the environment, the control of communicable diseases, the organization of medical and nursing services for the early diagnosis and prevention of disease, the education of the individual in personal health and the development of the social machinery to ensure for everyone a standard of living adequate for the maintenance or improvement of health.[1122] The goal of public health is to fulfill every society's ambition to create conditions in which all people can be healthy. Public health addresses the health of the population as a whole rather than the treatment of individuals. WHO defines health as "*a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity*".[1123]

"Healthy people in healthy communities" is the ultimate goal of all public health interventions, which are aimed at promoting physical and mental health and preventing disease, injury and disability.[1124] Public health is particularly concerned with threats to the overall health of the community.[1125] This report documents and illustrates the large body of scientific research that demonstrates human toxicity to fluoride as well as the over-exposure of the population generally to this toxic pollutant. This report has highlighted the many scientifically documented peer reviewed referenced sources of information that clearly identify sensitive sub-sectors of the population such as infants and diabetics are particularly at risk from continuation of the policy of water fluoridation. The Health Service Executive, Food Safety Authority and the Environmental Protection Agency must prioritize strategies to not only

[1122] Winslow CEA. The untilled field of public health. *Modern Medicine*, 1920, 2:183–191.

[1123] Preamble to the Constitution of the World Health Organization as adopted by the International Health Conference, 1946. In: *Basic documents*, 45th ed. Geneva, World Health Organization, 2005

[1124] Breslow L et al. *Encyclopedia of public health*. New York, Macmillan Reference, 2002.

[1125] Neurological Disorders Public Health Challenges, World Health Organization 2006

reduce health risks for all citizens but especially the most vulnerable and high-risk in society. This can only be achieved by either providing non-fluoridated water to households or providing the technology to households to remove fluoride from their water supply. For that matter it is incorrectly assumed that typical granulated activated carbon water filters and ultra-filtration membranes will remove fluoride from drinking water as they do not. Only by providing either reverse osmosis units or ending water fluoridation at source can one reduce fluoride concentrations to acceptable levels. In environmental law, the polluter pays principle applies. It is acceptable therefore that households who invest in equipment to remove artificially injected fluoride from their drinking water supply in order to protect the health and welfare of its occupants by reducing the contaminant level to acceptable international recommended limits, should be able to claim compensation from the State. It is inconceivable that the Government of Ireland would consider charging them for supply of water which is otherwise unfit for human consumption.

In order to quantify the potential economic cost and public health risk from fluoridation of drinking water, in excess of fifty comprehensive epidemiology, toxicology, clinical medicine, and environmental exposure assessments were identified requiring urgent assessment by the U.S. National Research Council (NRC) and the European Commission's Scientific Committee on Health and Environmental Risks(SCHER). Some of these studies were regarded as of paramount importance for the protection of public health. None of these studies have, to this author's knowledge, been undertaken by state agencies in the Republic of Ireland or in any other jurisdiction. In the absence of proper risk assessment the Government of Ireland cannot provide any assurance in relation to water fluoridation or fluoridation products.

Given the numerous and potentially dangerous and hazardous risks that have been identified (regardless of the probability or level of risk), where such a risk exists, the potential cost to the State and society clearly outweighs any perceived public health benefit. This has been clearly demonstrated in the most recent SCHER report, which coincidentally does not recommend the continued practice of water fluoridation. The specific concerns referred to above are summarized in the Health section of this report.

The fact that the SCHER review concluded that there is no social or economic advantage to the continued fluoridation of drinking water supplies must be acknowledged by the Government of Ireland given that the remit of SCHER is to provide expert advice to the European Commission and its member states.

Furthermore, taking into consideration the recommended comprehensive epidemiological, toxicological, clinical medicine and environmental exposure assessments that are required to be undertaken to support the continued fluoridation of drinking water, a balance must be struck between the cost and benefits of adopting a measure to avoid or control risks, and its disadvantages.

Proper regulation requires that both the individual risks, as identified in the published scientific reports, and societal concerns engendered by a hazard must be addressed. In undertaking such a review, both the NRC and SCHER

studies examined some of the developments that have influenced our approach to decision-making in the past for fluoridation of water, recent developments and advances in science and knowledge on how people view risks, the changes in the regulatory environment and shifts in the values, preferences and expectations of our society.

There are a number of principles of good regulation that have evolved in adapting the best approach to risk management to take account of such developments; namely:

The targeting of action:	focusing on the most serious risks or where the hazards are less well controlled;
Consistency:	adopting a similar approach in similar circumstances to achieve similar ends;
Proportionality:	requiring action that is commensurate to the risks;
Transparency:	being open on how decisions are arrived at and what their implications are;
Accountability:	making clear, for all to see, who is accountable when things go wrong.

The meaning of risk must also encompass more than physical harm by taking into account other factors such as ethical, economic and social considerations. The legal definition of "risk" implies that approaches for managing risks must ensure that anything in an undertaking presenting the possibility of danger (or what conceptually is regarded as a hazard) has to be properly addressed.

Both the NRC and SCHER reports clearly demonstrate that the correct approaches to managing risks associated with fluoridation of water supplies have clearly not been addressed to date and thereby expose the consumer and public to unnecessary risk. To perform such a task requires a need to make transparent the factors that inform our decisions on how risks should be regulated and managed, for example; how account is taken of the scientific knowledge of the risks concerned, the technology available for controlling them, the resource implications of adopting the decisions, public attitudes towards the risks and the benefits they engender and how they shape the form and content that our regulations and guidance take. In pursuing such a strategy one must reassure the public that risks to society and high-risk individuals are properly addressed. It is also necessary in the current financial crisis that the State responds to economic pressures and scientific advice that is based on robust scientific examination. This is particularly important where scientific research doubts the validity of any potential benefits of fluoridation but rather demonstrates significant potential hazards from exposure of the public and the environment by its continued use.

Finally, in defining risk prevention strategies, such as that implemented in Ireland, to improve dental health and provide social equality one must examine the extent to which the risk factor would now exist in the absence of water fluoridation. Since the introduction of water fluoridation as a public health measure post-World War Two, fluoride has also been added to toothpaste. It has been further shown that the topical application of fluoride

through toothpaste is the most effective method of caries prevention. Abundant evidence also exists to show that in countries that ended the policy of water fluoridation, no increase in the incidence of dental decay resulted. All of this has been examined in detail in Chapter 4 of this report. Therefore it is known that other cost effective intervention measures now exist to address the original aims and objectives of the HSE to improve dental health. It is also known that none of the risks now known to be associated with water fluoridation were available when the policy originally commenced. It is also unclear why the HSE would attempt to pursue a policy within Europe where Ireland remains the only country to continue with water fluoridation[1126], especially given the known health risks associated with this practice.

There is an obligation by public health services to assess whether adequate information on the health effects of fluoride is available. Where adequate information is not available the State is required to initiate a programme of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of fluorides. Chapter 10 of this report highlights the categories of extensive research that have been recommended by the NRC and SCHER to reduce or eliminate the uncertainties of the impact of fluorides on human health. To the author's knowledge none of the assessments have been undertaken by the State.

In examining the policy the State and HSE must also acknowledge how societal values and preferences have changed since the 1960's and how the public, if they were made aware of the dangers and risks posed by water fluoridation, would demand that the practice were ended, as has occurred in every other country where reviews have recently taken place.

Despite this, it is clear that many informed members of the Irish public (as well as the public internationally) do not agree with or accept that fluoridation of water supplies is safe, a fact that is evident from the limited public consultation process undertaken in 2002.[1127] If the wider public were aware of the findings of recent scientific studies it is certain that public opposition would rise dramatically. It is hoped that this report will go some way to raise environmental awareness and promote public participation in environmental decision-making.

An important observation in regard to the aforementioned reports is that it would appear that the *Irish Expert Body on Fluorides and Health Forum on Fluoridation* report (2002) may have misrepresented the results of the York Review as did the British Medical Association and British Fluoridation Society.

The YORK Review was exceptional in this field in that it was conducted by an independent group to the highest international scientific standards and a

[1126] Scientific Committee on Health and Environmental Risks, European Commission, Director General Health and Consumers, Critical Review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, May 2010.
[1127] Forum on Fluoridation Report 2002

summary has been published in the British Medical Journal[1128]. This study aimed to provide a systematic review of the best available evidence on potential positive and negative effects of water fluoridation. The review observed that the impact of fluoridation on drinking water supplies depends on a number of major issues: the potential benefits (including improved dental health and reductions in dental health inequalities); the potential benefits over and above that offered by the use of alternative interventions and strategies (e.g. fluoridated toothpaste); and the potential negative impacts that fluoridation can cause (including dental fluorosis, bone fractures and bone development problems, genetic mutations, birth defects, cancer and hypersensitivity).

The Chairman of the NHS Advisory Group for the systematic review and its founding director, Professor Trevor A. Sheldon published a letter in 2001 stating that "*(i)t is particularly worrying then that Statements which mislead the public about the review's findings have been made in press releases and briefings by the British Dental Association, the British Medical Association, the National Alliance for Equity in Dental Health and the British Fluoridation Society*" in support of fluoridation.

While it is unfortunate that similar statements regarding the York Review appear to have been made by the Irish *Forum for Fluoridation* in their report published in 2002; it is alarming that these statements and misintrepretations were made after the publication of the letter by Professor Sheldon.[1129]

Professor Sheldon wished to correct these errors of misprepresentation by stating unequivocally that "*the review did not show fluoridation to be safe,*" that the review "*found water fluoridation to be significantly associated with high levels of dental fluorosis which was not characterised as "just a cosmetic issue*" and that the review found that " *there was little evidence to show that water fluoridation has reduced social inequalities in dental health*". The review concluded that "*until high quality studies are undertaken providing more definite evidence, there will continue to be legitimate scientific controversy over the likely effects and costs of water fluoridation.*"

It is apparent and inconceivable that none of this information was presented to the Government by the expert panel on fluoridation. This raises serious questions and doubts regarding accountability, lack of independence and objectivity of the panel. While one can forgive Government for political decisions based on recommendations from appointed panels, the seriousness of the health implications regarding support for such a policy demands the highest levels of independent oversight and risk assessment.

In contrast the U.S. National Research Council (NRC), comprising the *National Academies of Sciences, Medicine and Engineering,* in their rigorous and comprehensive report on fluoridation highlighted an alarming number of

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[1128] Systematic review of water fluoridation *BMJ* 2000; 321

1129 Professor Sheldon, Department of Health Studies, Innovation Centre, York Science Park, 3/1/2001.

potential adverse public health risks associated with the continued use of fluoridation. Furthermore the NRC documented the growing weight of toxicological or epidemiological evidence identifying clear public health risks associated with the addition of fluoride to public drinking water supplies. The NRC's remit is to improve government decision-making and public policy, increase public understanding and promote the acquisition and dissemination of knowledge in matters involving science, engineering, technology and health. The Research Council's independent, expert reports and other scientific activities inform policies and actions that have the power to improve the lives of people in the U.S. and around the world.

Subsequent to the NRC report, the European Commission Director General for Health and Consumers requested the European Commission's Scientific Committee on Health and Environmental Risks (SCHER) to undertake an independent critical review of the hazards and health effects of fluoridating agents and fluoride. The SCHER work in close collaboration with the *Scientific Committee on Consumer Products* (SCCP), *the European Food Safety Authority* (EFSA), *the Scientific Committee on Consumer Safety* (SCCS), *the Scientific Committee on Emerging and Newly Identified Health Risks* (SCENIHR), *the European Medicines Evaluation Agency* (EMEA), *the European Centre for Disease prevention and Control* (ECDC) and the *European Chemicals Agency* (ECHA). SCHER published its report in 2010.

It is noted that the Committee provides opinions on health and environmental risks related to pollutants in environmental media and other biological and physical factors which may have a negative impact on health and the environment. The Committee provides opinions on life-cycle environmental assessment and health and safety issues related to the toxicity and eco-toxicity of biocides. In addition, the Committee addresses questions relating to methodological aspects of the assessment of health and environmental risks of chemicals (including fluoride). It provides sound and consistent advice in its areas of competence and contributes to addressing relevant issues in close cooperation with other European agencies.

A critical finding of both the NRC and SCHER reports is that the the toxicology of hexafluorosilicic acid, and hexafluorosilicictes compounds used to fluoridate drinking water supplies, is incompletely investigated. The implications of this finding, given the potential hazards identified in both the SCHER and NRC reports, poses serious and unacceptable risks to the consumer.

Further information is provided from Harvard College's School of Dental Medicine, the international peer-reviewed journal *Toxicology*[1130] (the official journal of the British Toxicology Society and the German Toxicology Society), the Lancet medical journal[1131], the international peer-reviewed journal

[1130] *Toxicology*, an international journal, publishes only the highest quality original research and critical reviews dealing with the adverse effects of xenobiotics on the health of humans and animals. All manuscripts published in *Toxicology* are subject to rigorous peer review.

[1131] The *Lancet* is the world's leading general medical journal and has specialty journals in Oncology, Neurology and Infectious Diseases.

Neurotoxicology[1132], the journal of Epidemiology[1133], the journal of Biological Chemistry[1134], the journal Neurologia[1135], the journal Radiology[1136], the Australian & New Zealand Journal of Public Health, the Journal of the American Medical Association[1137], the Journal of Public Health Dentristy[1138], with additional observations and recommendations by a coalition of U.S. Environmental Protection Agency Unions[1139], the American Dental Association, the U.S. Department of Health and Human Services, the Centre for Disease Control and Prevention (CDC), the World Health Organisation, the United States Public Health Service and the Department of Health Studies, University of York, U.K and the Russion Academy of Sciences to name but a few. The most recent peer-reviewed Fluoride Risk Assessment and Relative Source Contribution studies undertaken by the U.S. EPA were also reviewed. The latter study[1140] undertaken by the U.S. EPA in response to the 2006 National Research Council (NRC) report: *Fluoride in Drinking Water* concluded that the main mechanism for fluoride intake in children is through ingestion of drinking water and diet. The agency reported how prolonged high intake of fluoride, at any age, can result in skeletal fluorosis, an irreversible condition which may increase bone brittleness, and a potential increase in risk of bone fracture. The most recent data collected by the U.S. Agency[1141] included estimating the fluoride dose resulting from drinking water. This resulted in age- and drinking water consumption-specific dose estimates that ranged from 0.04 mg/kg/day to 0.19 mg/kg/day. For mean water consumption rates, the doses ranged from 0.04 to 0.09 mg/kg/day for the different age groups. These values were compared to the dose level of 0.05 mg/kg/day which had been recommended as an Adequate Intake (AI) by the American Institute of Medicine (IOM, 1997) for optimal anticaries protection. This report demonstrates conclusively that fluoridation of drinking water contributes to excess fluoride intake above the recommended daily dose levels.

1132 *NeuroToxicology* specializes in publishing the best peer-reviewed original research papers dealing with the effects of toxic substances on the nervous system of humans and experimental animals of all ages.

1133 Epidemiology is the official journal of the International Society for Environmental Epidemiology (ISEE).

1134 The Journal of Biological Chemistry is published by the American Society for Biochemistry and molecular Biology.

1135 Neurología is the official scientific journal of the Spanish Neurology Society (Sociedad Española de Neurología, SEN).

1136 Radiology is a journal devoted to clinical radiology and allied sciences published by the Radiological Society of North America.

1137 The Journal of American Medical Association is an international peer-reviewed general medical journal and is the most widely circulated medical journal in the world.

1138 The Journal of Public Health Dentistry is devoted to the advancement of public health dentistry. It is published on behalf of the American Association for Public Health Dentistry.

1139 NTEU CHAPTER 280, U.S. Environmental Protection Agency, National Headquarters, A Statement of Concern on Fluoridation (2002).

1140 Fluoride: Dose-Response Analysis For Non-cancer Effects Health and Ecological Criteria Division Office of Water, Environmental Protection Agency, January, 2011.

1141 Fluoride: Dose-Response Analysis For Non-cancer Effects Health and Ecological Criteria Division Office of Water, Environmental Protection Agency, January, 2011.

It is widely acknowledged that policy makers and healthcare providers are unprepared to cope for the predicted rise in the prevalence of neurological and other chronic disorders such as dementia, Parkinson's disease, Alzheimer's disease, epilepsy, multiple sclerosis, neuroinfections, diabetes, pain associated with neurological disorders, arthritis, osteoporosis, chronic fatigue syndrome and autism.[1142]

It is obvious that where scientific studies indicate that environmental toxins could be associated with the onset of any of these conditions, it must be scientifically considered, regardless of how small the perceived risk, that fluoride may be a contributing factor to any of these conditions. This is particularly the case when a toxic and potentially dangerous substance such as hydrofluorosilicic acid is distributed through the public water supply system. To my knowledge, to date no such study has comprehensively been undertaken in Ireland.

The York, NRC, SCHER and most recent U.S. EPA studies acknowledge that many potential biological, neurological and toxicological risks are possible from exposure to fluoride. It is obvious that where there is any risk, no matter how small, in the absence of detailed risk assessments and given the concerns raised in scientific research the Government of Ireland must act to minimise any such risk and enact the precautionary principle by ending the policy of fluoridation of drinking water immediately.

[1142] Neurological Disorders Public Health Challenges, World Health Organization 2006

13.0 Fluoridation and Department of Health and Children

The Department of Health and Children is responsible for policy in relation to the Irish health services. It also has a role in the strategic planning of health services. This is carried out in conjunction with the Health Services Executive, voluntary service providers, government departments and other interested parties.

The Department's mission Statement is: *"To improve the health and well being of people in Ireland in a manner that promotes better health for everyone, fair access, responsive and appropriate care delivery, and high performance"*. The Department's mandate states *"The Minister for Health and Children is politically accountable for developing and articulating Government policy on health and personal social services, and for the overall performance of the health service. Our mandate is to support the Minister and the Ministers of State by advising on policy development and implementation, evaluating the performance of existing policies and service delivery, preparing legislation, and working with other Government Departments, the social partners and international organizations.*

The functions of the Department of Health and Children are:

- To develop policy across the full spectrum of health and personal social services, with a focus on quality, equity, access based on need, consistency and outcomes, and to support implementation of Government policy.
- To negotiate and report on the Health group of votes and analyse financial and service outturns, including value for money and adherence to governance and accountability standards.
- To provide a legislative and regulatory framework that helps protect the interests of service users and supports practitioners in working to the highest standards and to work with colleagues in other Government Departments and the social partners to ensure that the aim of improving health and social well-being is advanced effectively in other parts of the public service. [1143]

It is not possible, based on information provided in this report, for the Department to deliver an equitable service based on quality, access based on need, consistency and outcomes by supporting a policy of mandatory fluoridation of drinking water. While the practice of fluoridation of drinking water was intended to have a beneficial effect on caries[1144] prevention and reduce social inequalities in dental health there is now unequivocal evidence to show that the practice is now contributing to adverse public health risks, especialy in children. The public have always been assured that there was absolutely no possibility of any harm or risk from fluoridation of water. There is now unequivocal evidence that demonstrates that this is not the case. Recent robust international peer-reviewed scientific studies have

[1143] Department of Health and Children.
[1144] The medical term for tooth decay, Gale Encyclopaedia of Medicine.

demonstrated that the addition of fluoride to drinking water is not safe and that certain sub-groups of the population are at risk from water fluoridation.

As noted elsewhere in this report, it has been agreed by both opponents and supporters of fluoridation that there has been a dramatic increase in the number of cases of fluorosis in Ireland in recent years. It is a condition that alarmingly may now affect one in three children in the Republic of Ireland. It has been demonstrated scientifically that excessive ingestion of fluoride leads to dental and skeletal fluorosis. [1145] It is known that young children are most at risk as over-exposure to fluoride in the early childhood has irreversible consequences. For example, it is scientifically known that fluoride readily accumulates in the human pineal gland[1146] and that by old age, the pineal gland has a higher fluoride content than either bone, teeth or enamel.'[1147]

The main pineal hormone is melatonin. Melatonin has strong antioxidant effects with a particular role in the protection of nuclear and mitochondrial DNA. Preliminary evidence suggests that melatonin may help strengthen the immune system. Melatonin also helps control the timing and release of female reproductive hormones. Animal studies suggest that melatonin may be effective for treating Alzheimer's disease and in the mechanisms of learning and memory.[1148,1149,1150,1151] Pineal fluoride concentrations significantly correlate with pineal calcium. In fact, calcification of the developing enamel organs in children and the pineal gland occur concurrently. This could affect pineal metabolism in much the same way that high local concentrations of fluoride in the developing enamel organ affect ameloblast function (the developing enamel structure).[1152]

[1145] Defined as chronic fluoride poisoning, McGraw-Hill Concise Dictionary of Modern Medicine.

[1146] Luke J. (1997). The Effect of Fluoride on the Physiology of the Pineal Gland. Ph.D. Thesis. University of Surrey, Guildford.

[1147] Michotte Y, Lowenthal A, Knaepen L, Collard M, Massart DL: A morphological and chemical study of calcification of the pineal gland. J Neurol 1977;215:209-219.

[1148] Hardeland, RüDiger (2005). "Antioxidative Protection by Melatonin: Multiplicity of Mechanisms from Radical Detoxification to Radical Avoidance". *Endocrine* **27** (2): 119–30

[1149] Reiter, Russel J.; Acuña-Castroviejo, Dario; Tan, DUN-Xian; Burkhardt, Susanne (2006). "Free Radical-Mediated Molecular Damage". *Annals of the New York Academy of Sciences* 939: 200–15

[1150] Pappolla, MA; Sos, M; Omar, RA; Bick, RJ; Hickson-Bick, DL; Reiter, RJ; Efthimiopoulos, S; Robakis, NK (1997). "Melatonin prevents death of neuroblastoma cells exposed to the Alzheimer amyloid peptide". *The Journal of neuroscience : the official journal of the Society for Neuroscience* **17** (5): 1683–90

[1151] Larson, John; Jessen, Ruth E.; Uz, Tolga; Arslan, Ahmet D.; Kurtuncu, Murat; Imbesi, Marta; Manev, Hari (2006). "Impaired hippocampal long-term potentiation in melatonin MT2 receptor-deficient mice". *Neuroscience Letters* 393 (1): 23–6

1152 Luke J. (2001). Fluoride deposition in the aged human pineal gland. School of Biological Sciences, University of Surrey, Guildford, UK, Department of Obstetrics and Gynaecology, The Royal London Hospital, Caries Research 35:125-128.

There is consequently a risk for bottle-fed babies who ingest milk formula reconstituted with fluoridated water that fluoride will accumulate in the child's pineal. This is particularly significant given that large amounts of calcification have been demonstrated in the pineals from young children.[1153],[1154],[1155],[1156],[1157]

After finding that the pineal gland is a major target for fluoride accumulation in humans, Dr. Luke conducted preliminary animal experiments to determine if the accumulated fluoride could impact the functioning of the gland - particularly the gland's regulation of melatonin. Luke found that animals treated with fluoride had lower levels of circulating melatonin, as reflected by reduced levels of melatonin metabolites in the animals' urine. This reduced level of circulating melatonin was accompanied by an earlier onset of puberty in the fluoride-treated female animals. Luke summarized her human and animal findings as follows: *"In conclusion, the human pineal gland contains the highest concentration of fluoride in the body. Fluoride is associated with depressed pineal melatonin synthesis by prepubertal gerbils and an accelerated onset of sexual maturation in the female gerbil. The results strengthen the hypothesis that the pineal has a role in the timing of the onset of puberty. Whether or not fluoride interferes with pineal function in humans requires further investigation."* [1158]

The first step in assessing a health risk by a substance to humans is the identification of its harmful effects on animals. A health risk to humans is assessed using results from human epidemiological studies in conjunction with results from animal studies. The latter animal study indicates that fluoride is associated with an earlier onset of puberty in female gerbils while as far back as 1956 the Newburgh-Kingston Study identified that bone defects, anaemia and earlier female menstruation occur more often in children living in the fluoridated Newburgh than in unfluoridated Kingston.[1159]

Researchers have found that in recent years girls are menstruating more than earlier than they did 20 years ago. [1160] It is also known that early menstruation can lead to an increased risk of breast cancer, obesity, and miscarriage.[1161]

[1153] Cooper ERA: The human pineal gland and pineal cysts. J Anat (Lond) 1932;67:28-46.

[1154] Wurtman RJ: The pineal gland; in Endocrine Pathology. Baltimore, Williams & Wilkins, 1968, pp 117-132.

[1155] Kerényi NA, Sarkar K: The postnatal transformation of the pineal gland. Acta Morphol Acad Sci Hung 1968; 16:223-236.

[1156] Tapp E, Huxley M: The weight and degree of calcification of the pineal gland. J Pathol 197 1; 105:31-39

[1157] Doskocil M: Development of concrements in the human pineal body. Folia Morphol (Praha) 1984;32:16-26.

[1158] Luke J. (2001). Fluoride deposition in the aged human pineal gland. School of Biological Sciences, University of Surrey, Guildford, UK, Department of Obstetrics and Gynaecology, The Royal London Hospital, Caries Research 35:125-128.

[1159] Newburgh-Kingston caries-fluorine study. XIII. Pediatric findings after ten years. J Am Dent Assoc. 1956 Mar;52(3):296-306. Schlesinger ER, Overton DE, Chase HC, Cantwell KT

[1160] David S. Freedman, Laura Kettel Khan, Mary K. Serdula, William H. Dietz, Sathanur R. Srinivasan, and Gerald S. Berenson, Relation of Age at Menarche

The obvious risks from dietary fluoride exposure are consequently of particular significance especially where it may be shown to effect synthesis and secretion of melatonin or other functions of the pineal gland in humans. This was acknowledged by the U.S. National Research Council in their review when they observed that "recent information on the role of the pineal organ in humans suggests that any agent that affects pineal function could affect human health in a variety of ways, including effects on sexual maturation, calcium metabolism, parathyroid function, postmenopausal osteoporosis, cancer, and psychiatric disease."[1162]

As with the implications for coronary disease the potential consequences of disturbances to functions of the pineal gland and resultant human health impacts from increased absorption of fluoride through dietary intake from water fluoridation cannot be underestimated.

Water fluoridation also effects the fluoride concentrations of breast milk and blood plasma levels of mothers. Breast milk is the major dietary intake of infants in the early stage of life in most other countries apart from Ireland, which continues to have one of the lowest incidences of breastfeeding in the developed world. It has been demonstrated that the dietary intake of fluoride in breastfeeding mothers will affect the levels of fluoride in their breast milk. The lower the exposure of mothers to fluoride, the lower the concentration in breast milk. [1163] Regardless of the level of fluoride in water however, the level of fluoride absorbed and transmitted to an infant in breast milk is limited and minor in comparison to that found in formula milk reconstituted with fluoridated water which in themselves could be deemed critical especially regarding the potential dental fluorosis that may result from such high concentrations of dietary fluoride.[1164],[1165] Rahul et al found that fluoride concentrations of various commercially available infant milk formulations ranged from 1.95 ppm to 7.45 ppm.[1166]

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to Race, Time Period, and Anthropometric Dimensions: The Bogalusa Heart Study, Pediatrics 2002; 110:e43

[1161] Reproductive History and Breast Cancer Risk, Factsheet, U.S National Cancer Institute, National Institutes of Health, Department of Health and Human Services.

[1162] National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C. p221-22

[1163] Yamur Şener, DDS, PhD,a Gül Tosun, DDS, PhD,a Firdevs Kahveciolu, DDS, PhD,b Alparslan Gökalp, DDS, PhD,c and Hasan Koç, MD, Fluoride Levels of Human Plasma and Breast Milk, Eur J Dent. 2007 January; 1(1): 21–24. PMCID: PMC2612944

[1164] Fejerskow O, Manji F, Baelum V. The nature and mechanism of dental fluorosis in man. *J Dent Res.* 1990;69:699–700.

[1165] Ekstrand J, Fomon SJ, Ziegler EZ, Nelson SE. Fluoride pharmacokinetics in infancy. *Pediatr Res.* 1994;35:157–163

[1166] Rahul P, Hedge AM, Munshi AK. Estimation of the fluoride in human breast milk, cow's milk and infant formulate. *J Clin Pediatr Dent.* 2003;27:257–260

It is accepted that dental fluorosis does not develop after teeth have erupted in children; therefore the critical exposure pathway for infants is primarily through fluoridated water, and in particular, boiled fluoridated water used for infant formula feed. This report outlines the significant health dangers of using fluoridated water for sensitive sub-groups of the population in Ireland.

This report documents how infants are exposed to multiples of the recommended upper tolerable daily intake of fluoride at concentrations that are unacceptably high. The Department therefore has a duty of care to take action to inform the Minister, parents and public health officials of the potential risks associated with water fluoridation and infant care and provide households with low fluoride drinking water. The increased risk to health for infants has been addressed in detail in this report. The Government of Ireland have a legal duty of care to protect children from harm and the policy of fluoridating drinking water supplies with a dangerous substances that is known to be an enzymatic metabolic poison is both unlawful and in breach of the Charter for Fundamental Rights of the European Union. It is clear that the Government of Ireland, in supporting the mandatory fluoridation of public drinking water supplies, is in clear violation of Articles 3,6, 24, 28 35 and 37 of the Charter for Fundamental Rights of the European union. Article 24, in particular, states that "in all actions relating to children, whether taken by public authorities or private institutions, the child's best interests must be a primary consideration". The legal implications and non-compliance with numerous EU Directives and international treaties is addressed elsewhere within this report.

It is obvious that the Department of Health and Children must take measures to inform the public and consumer groups of the risk of systemic fluoride poisoning (over-exposure) of infants, in particular, from parents mixing powdered or liquid infant formula concentrate with fluoridated water. This risk is increased as the water is recommended to be boiled increasing the concentration of fluoride in the water further. In light of the findings addressed in this report and documentation of hundreds of scientific studies that demonstrate risk to human health, it is imperative that the public should be provided with non-fluoridated sources of drinking water. Bottled water is not a solution as this places an unnecessary financial burden on households and more especially as manufacturers do not provide information on fluoride content.

The report has further identified, in subsequent chapters, the enormous volume of data that is legally required on toxicological information of fluoridation compounds for the interest of public safety. Should the policy of fluoridation continue the Department has a responsibility to ensure that all these studies are conducted in full and the finance is made available to fund the necessary health surveillance programmes as recommended for consumer safety by independent expert scientific committees.

The author of this report is uncertain how Department of Health officials could inform the Minister of State at the Department of Health (Deputy Róisín Shortall) that "*the report of the EU Scientific Committee on Health and Environmental Risks (SCHER), published in June of this year, has not made any findings of negative health or environmental effects concerning fluoridation of water*". [1167]

This is clearly incorrect and a misrepresentation of the findings as presented by the scientific committee of SCHER and outlined elsewhere in this report. The main findings of the SCHER review are summarized in this report.

One of the more critical risk concerns to public health that SCHER observed is that there exists a lack of human studies to prove conclusively that fluoride intake from fluoridation of water does not affect the neurological development of children or lead to increased risk of bone fractures. SCHER agreed that some epidemiological studies seem to indicate a possible link between fluoride in drinking water and osteosarcoma. While SCHER may have observed that "*available human studies do not allow concluding firmly that fluoride intake hampers children's neurodevelopment*" it did not question the latest scientific findings or present any shortcomings regarding a recent study in which they reported that intake of fluoride in drinking water may contribute to the decreased intelligence in children. [1168] It is important to note that the authors of this particular study, Rocha-Amador et al. further concluded that "*the risk is particularly acute for children, whose brains are particularly sensitive to environmental toxins. Furthermore, it would be advisable to re-examine the benefits of F given the documented health risks.*"

While the health implications of fluoride and the findings of SCHER have been examined comprehensively in this report, it is important to acknowledge that the environmental review undertaken by SCHER was limited and simplistic, as accepted and acknowledged by SCHER themselves in their review.[1169]

To compensate for this, this report has examined in greater detail the risks to ecological receptors in the terrestrial and marine environments. It has discussed food chain bioaccumulation and exposure pathways within the human food chain from contamination of food prepared or processed with fluoridated water.[1170] This report has further examined the health implications for a sub-group of the population that has been documented as sensitive to fluoride.[1171] The impacts of the pollutant fluoride in wastewaters discharges on

[1167] Dáil Éireann Debate *Vol. 746 No. 1*

[1168] Rocha-Amador D, Navarro ME, Carrizales L, Morales R, Calderón J, Facultad de Medicina, Universidad Autónoma de San Luis Potosí, San Luis Potosí, México. Decreased intelligence in children and exposure to fluoride and arsenic in drinking water; Cad. Saúde Pública, Rio de Janeiro, 23 Sup 4:S579-S587 (2007).

[1169] Scientific Committee on Health and Environmental Risks, European Commission, Director General Health and Consumers, Critical Review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water, May 2010.

[1170] Cook HA. 1969. Fluoride in tea. Lancet 2:329.

[1171] USA National Research Council, Fluoride in Drinking Water: A Scientific Review of EPA's Standards, Committee on Fluoride in Drinking Water, (2006)

freshwater ecosystems, in particular juvenile salmon, rainbow trout and Daphnia magna the main food source for these species as reported in this study, will be of particular interest to the Department of Agriculture, Food and the Marine, the Department of the Communications, Energy and Natural Resources, the Department of Environment, Community and Local Government, Inland Fisheries Ireland and the Environmental Protection Agency requiring further detailed examination and assessment of the environmental impacts of fluoride on the environment and inland fisheries.

It is apparent, based on the information provided in this report, that the SCHER environmental assessment, while not the principle focus of the review, was totally inadequate, incomplete and based almost entirely on one desk-based study.[1172.] The observation and conclusion of this study itself clearly contradicted the subsequent findings of the SCHER committee. This is discussed in further detail within this report.

In considering whether to continue to support the policy of water fluoridation, in opposition to the recommendations of SCHER, the Department must examine the policy in the context of one of the key principles of the National Health Strategy in the Republic of Ireland, that of equity.[1173] This addresses the targeting of health inequalities and the treatment of people according to need. This report has clearly outlined how this principle cannot be upheld by a continuation of the existing water fluoridation policy. Ireland remains the only country in Europe with a nationally mandated policy for water fluoridation despite the introduction of topical fluoride based toothpaste products into this county in 1969.

This report has highlighted the increased risk of coronary artery disease (CAD) which is linked to fluoride exposure and how blood serum fluoride levels influence the rate of calcification in the human body. CAD is a progressive disease process that generally begins in childhood and manifests clinically in middle to late adulthood. Cardiovascular disease is the single biggest cause of death in Ireland accounting for over 40% of all deaths and 37% of deaths under 65 years of age in Ireland. This report has highlighted that Ireland has the highest mortality rate from ischaemic heart disease in males in the European Union.[1174] This report has further highlighted how despite improvement in diagnosis, medical healthcare and preventative medicine, Ireland remains the only country in western Europe with increasing mortality rates form coronary disease.

Blood fluoride levels were also observed to be significantly higher in patients with osteosarcoma.[1175] Research has also demonstrated that the

[1172] Camargo JA (2003) Fluoride toxicity to aquatic organisms: a review. Chemosphere 50: 251-64

[1173] Department of Health and Children. Quality and Fairness: A health system for you. Dublin: Stationery Office, 2001

[1174] 1996 World Health Statistics Annual, World Health Organisation, Geneva, 1998

[1175] Sandhu R, Lal H, Kundu ZS, Kharb S. Serum fluoride and sialic Acid levels in osteosarcoma. Biol Trace Elem Res. 2011 Dec;144(1-3):1-5. Epub 2009 Apr 24.

concentration of fluoride in the bones increased in an essentially linear fashion with an increase of fluoride in the drinking water.[1176]

It has been scientifically documented and accepted by international scientific committees that a small percentage of the population may be particularly sensitive to exposure to fluoride.[1177],[1178] This represents approximately 1% of the entire population living in Ireland or 45,800 individuals. A further 55,000 infants exceed the daily maximum tolerable recommended level for fluoride by ingesting formula milk made up with fluoridated water. Just with these two sub-groups alone over 100,000 Irish citizens are unfairly targeted by the continuation of the policy for water fluoridation.

The link between Vitamin C deficiency and fluoride, as discussed previously in this report, could also mean that water fluoridation may unintentionally target disportionately poorer income families that may have restricted dietary intake of Vitamin C.

This may be of particular interest to the Department as a national health survey has documented the inadequacy of Vitamin C intake in boys and girls aged between 5-12 years in Ireland.[1179] According to population estimates for 2007, there were 1,056,947 children under the age of 18 living in Ireland.[1180.] It is documented in the latter survey that at least 10% of these are deficient in Vitamin C. Therefore, it is plausible that the policy of water fluoridation may be adversely affecting a further 105,000 children in Ireland, ironically the same target population that the policy of water fluoridation was intended to protect.

Children represent the future of this nation; it is obvious given the available scientific data that the State cannot protect infants from the numerous potential health impacts associated with excessive intake of fluoride by continuing with the policy of water fluoridation. The most appropriate and cost effective policy is risk prevention at source by not artificially adding fluoride to drinking water in the first place.

[1176] Zipkin, I.; McClure, F. J.; Leone, N. C.; Lee, W. A. Fluoride deposition in human bones after prolonged ingestion of fluoride in drinking water, Public Health Reports (1958), 73, 732-40 CODEN: PHRPA6; ISSN: 0033-3549

[1177] Feltmnn, R. and Kosel, G., Prenatal and Postnatal Ingestion of Fluorides -- Fourteen Years of Investigation -- Final Report, Journal of Dental Medicine, 16:190, October 1961.

[1178] Fluoride in Drinking Water: A Scientific Review of EPA's Standards, Committee on Fluoride in Drinking Water, National Research Council (2006)

[1179] Irish Universities Nutrition Alliance (IUNA), 2001, Adequacy of micronutrient Intakes in Ireland, Results from the National Food Consumption Surveys

[1180] Department of Health and Children 2008

14.0 Risks and Observations noted in the U.S. National Research Council (NRC) Report 2006

According to the NRC *"it is apparent that fluorides have the ability to interfere with the functions of the brain and the body by direct and indirect means"*.[1181]

The NRC noted that the *Agency for Toxic Substances and Disease Registry* (ATSDR 2003) discussed four papers on thyroid effects and two papers on parathyroid effects and concluded that *"there are some data to suggest that fluoride does adversely affect some endocrine glands"*.

The NRC reported that *"the suggested mechanisms of action for the results reported to date in peer-reviewed literature include decreased production of thyroid hormone, effects on thyroid hormone transport in blood, and effects on peripheral conversion of T4 to T3 or on normal deiodination processes"*.

The NRC reported that two studies have examined the age of onset of menstruation (age of menarche) in girls in fluoridated areas (Schlesinger et al. 1956; Farkas et al. 1983) and both noted that postmenarcheal girls present at younger ages where drinking water is fluoridated compared to non-fluoridated areas.

On endocrine functions, the NRC committee concluded that "fluoride exposure appears to bring about increases in blood glucose or impaired glucose tolerance in some individuals and to increase the severity of some types of diabetes"[1182]. The NRC also highlighted the increased health risk to diabetic individuals who will often have higher than normal water intake, and consequently, will have higher than normal fluoride intake for a given concentration of fluoride in drinking water.

This presents a particular health management risk for the up to 200,000 patients diagnosed (and a further 200,000 undocumented that have the condition) with diabetes in Ireland[1183]; any role of fluoride exposure in the development of impaired glucose metabolism or diabetes is potentially significant.] According to the NRC, fluoride is an endocrine disruptor altering normal endocrine function or response, although probably not in the sense of mimicking a normal hormone. *"The mechanisms of action remain to be worked out and appear to include both direct and indirect mechanisms, for example, direct stimulation or inhibition of hormone secretion by interference with second messenger function, indirect stimulation or inhibition of hormone secretion by effects on things such as calcium balance, and inhibition of*

[1181] USA National Research Council, Fluoride in Drinking Water: A Scientific Review of EPA's Standards, Committee on Fluoride in Drinking Water, (2006)

[1182] National Research Council of the National Academies, *FLUORIDE IN DRINKING WATER*, *A Scientific Review of EPA's Standards,* Ch. 8. Effects on the Endocrine System, Pages 259-260:

[1183] Diabetes Federation of Ireland.

peripheral enzymes that are necessary for activation of the normal hormone".

The NRC reported that fluoridated water is known to elicit acute gastrointestional systems (GI) affecting the liver, kidney and immune system. The NRC reported how scientific studies[1184] have demonstrated that at least 1% of the population complains of GI symptoms after fluoridation is initiated.

The NRC reported how animal studies have provided some important information on the mechanisms involved in GI toxicity from fluoride. Fluoride can stimulate secretion of acid in the stomach (Assem and Wan 1982; Shayiq et al. 1984), reduce blood flow away from the stomach lining, dilate blood vessels, increase redness of the stomach lining (Fujii and Tamura 1989; Whitford et al. 1997) and cause cell death and desquamation of the GI tract epithelium.[1185], [1186],[1187],[1188] The NRC acknowledged that fluoride is a risk factor for kidney stones and that fluoride toxicity can affect kidney tissue and function. The NRC noted, in particular, the public health risk to individuals who have impaired kidney function. The NRC recommended *extreme caution* to be used for patients on hemodialysis and that additional studies be carried out to determine the incidence, prevalence and severity of renal osteodystrophy in patients with renal impairments where there is fluoridation of drinking water supplies. The NRC observed that the effect of low doses of fluoride on kidney and liver enzyme functions in humans needs to be carefully documented in communities exposed to different concentrations of fluoride in drinking water.

The NRC reported that the plausibility of the bladder as a target for fluoride is supported by the tendency of hydrogen fluoride to form under physiologically acid conditions, such as found in urine. Hydrogen fluoride is caustic and might increase the potential for cellular damage, including genotoxicity. The NRC reported how some patients complaining, for example, of oral ulcers, colitis, urticaria, skin rashes, nasal congestion and epigastric distress patients might be sensitive to the effects of silicofluorides. The NRC reported how in a recent study[1189], Machalinski et al. (2003) reported that the four different human leukaemic cell lines were more susceptible to the effects of sodium hexafluorosilicicte, the compound most often used in fluoridation.

[1184] Feltman, R., and G. Kosel. 1961. Prenatal and postnatal ingestion of fluorides—Fourteen years of investigation. Final report. J. Dent. Med. 16(Oct.):190-198.
[1185] Easmann, R.P., D.E. Steflik, D.H. Pashley, R.V. McKinney, and G.M. Whitford. 1984. Surface changes in rat gastric mucosa induced by sodium fluoride: A scanning electron microscopic study. J. Oral Pathol. 13(3):255-264.
[1186] Pashley, D.H., N.B. Allison, R.P. Easmann, R.V. McKinney, J.A. Horner, and G.M. Whitford. 1984. The effects of fluoride on the gastric mucosa of the rat. J. Oral Pathol. 13(5):535-545.
[1187] Susheela, A.K., and T.K. Das. 1988. Chronic fluoride toxicity: A scanning electron microscopic study of duodenal mucosa. J. Toxicol. Clin. Toxicol. 26(7):467-476.
[1188] Kertesz, P., T. Kerenyi, J. Kulka, and J. Banoczy. 1989. Comparison of the effects of NaF and CaF2 on rat gastric mucosa. A light-, scanning- and transmission electron microscopic study. Acta Morphol. Hung. 37(1-2):21-28.
[1189] Machali ÅLnski, B., M. Baskiewicz-Masiuk, B. Sadowska, A. Machalinska, M. Marchlewicz, B. Wiszniewska, and I. Stecewicz. 2003. The influence of sodium fluoride and sodium hexafluorosilicicte on human leukemic cell lines: Preliminary report. Fluoride 36(4):231-240.

The NRC noted how clinical trials reported an elevated risk of new nonvertebral fractures in populations exposed to drinking water containing fluoride. An increased risk of bone fracture was found among a subset of the trials.

The NRC reported that overall, there was consensus among the committee that there is scientific evidence that under certain conditions fluoride can weaken bone and increase the risk of fractures. It is acknowleged that hip fracture rates have increased dramatically in recent years, independently of the increasing age of populations.

Seven other studies have now reported this association between low water fluoride levels and hip fractures[1190],[1191],[1192],[1193],[1194],[1195] in support of the NRC findings.

It has been reported[1196] that since 1990, five major epidemiological studies from three countries-the United States, the United Kingdom and France - showed a higher rate of hip fractures in fluoridated regions. In another medical journal in the U.S., a significant increase in the risk of hip fracture in both men and women exposed to artificial fluoridation at 1 ppm[1197] was also documented.

The NRC reported how fluoride, usually in the millimolar range, has a number of effects on immune cells, including polymorphonuclear leukocytes, lymphocytes and neutrophils and acknowledged that there is no question but that fluoride can affect the cells involved in providing immune responses.

The NRC acknowledged that there exists the possibility that a small percentage of the population reacts systemically to fluoride, perhaps through changes in the immune system.

1190 Cooper C, Wickham CAC, Barker DJR, Jacobsen SJ. Letter. Journal of the American Medical Association 266 513-514 1991.

[1191] Jacobsen SJ, Goldberg J, Cooper C, Lockwood SA. The association between water fluoridation and hip fracture among white women and men aged 65 years and older. A national ecologic study. *Annals of Epidemiology* 2 617-626 1992

1192 Sowers MFR, Clark MK, Jannausch ML, Wallace RB. A prospective study of bone mineral content and fracture in communities with differential fluoride exposure. American Journal of Epidemiology 133 649-660 1991.

[1193] Jacqmin-Gadda H, Commenges D, Dartigues J-F. Fluorine concentration in drinking water and fractures in the elderly. *Journal of the American Medical Association* 273 775-776 1995.

[1194] Danielson C, Lyon JL, Egger M, Goodenough GK. Hip fractures and fluoridation in Utah's elderly population. *Journal of the American Medical Association* 268 746-748 1992.

1195 Keller C. Fluorides in drinking water. Paper presented at Workshop on Drinking Water Fluoride Influence on Hip Fractures and Bone Health. Bethesda MD, April 10 1991. May DS., Wilson MG. Hip fractures in relation to water fluoridation: an ecologic analysis. Paper presented at Workshop on Drinking Water Fluoride Influence on Hip Fractures and Bone Health. Bethesda MD, April 10, 1991.

1196 Australian & New Zealand Journal of Public Health, 1997 vol. 21 no. 24

[1197] Journal of the American Medical Association, August 1992

The NRC warned that from an immunologic standpoint, individuals who are immunocompromised (e.g. AIDS, transplant and bonemarrow- replacement patients) could be at greater risk of the immunologic effects of fluoride.

In regard to carcinogenicity and animal cancer studies, the NRC reported that the nature of uncertainties in the existing data could also be viewed as supporting a 'greater precaution' regarding the potential risk to humans.

The NRC reported how the Hoover et al[1198](1991) analyses of the Iowa and Seattle cancer registries indicated a consistent trend of kidney cancer incidence with duration of fluoridation.

Alarmingly, the NRC reported how the *U.S. Agency for Toxic Substances and Disease Registry* (ATSDR 2003) estimated that the ecological studies performed to date for fluoride and cancer did not have sensitivities to detect less than 10% and 20% increases in cancer raising serious concerns regarding the standard epidemiologic methods [this is of particular concern in Ireland given the dramatic increase in the percentage of kidney cancers as reported by National Cancer Registry Ireland (NCRI) and the relatively high percentage of kidney cancer incidence for women in Ireland (9th highest of 31 countries). The NCRI also report that bladder cancer incidence was relatively higher for women in Ireland (4th highest of 31 countries) and that prostate cancer incidence in Ireland ranked highest of all 30 European countries and was over 60% higher than the EU average[1199]].

The NRC reported how oral-pharyngeal cancers among females constituted the only site-gender category for which standardized mortality ratios in England were found to be significantly elevated in areas with naturally occurring high fluoride concentrations, defined as more than 1.0 mg/L.

The NRC reported an association of uterine cancer (combination of cervical and corpus uteri) with fluoridation as documented by Tohyama (1996), who observed mortality rates in Okinawa before and after fluoridation was terminated, controlling for socio-demographics.

In its summary of cancer epidemiology findings, the NRC acknowledged that the Harvard-based hospital multicenter osteosarcoma study (referenced below) would add important data to the current body of literature on fluoride risks for osteosarcoma because the study includes bone fluoride concentrations for cases and controls.

This study[1200] undertaken at Harvard observed that for males diagnosed before the age of 20 years, fluoride level in drinking water during growth was

[1198] Hoover, R.N., S.S. Devesa, K.P. Cantor, J.H. Lubin, and J.F. Fraumeni. 1991. Fluoridation of Drinking Water and Subsequent Cancer Incidence and Mortality. Appendix E in Review of Fluoride Benefits and Risks: Report of the Ad Hoc Subcommittee on Fluoride Committee of the Committee to Coordinate Environmental Health and Related Programs. Public Health Service, U.S. Department of Health and Human Services, Washington, DC.

[1199] National Cancer Registry Ireland data.

[1200] Fluoride & Osteosarcoma - Harvard Case-Control Study (2006).

associated with an increased risk of osteosarcoma, demonstrating a peak in the odds ratios from 6 to 8 years of age. All of their models were remarkably robust in showing this effect, which coincides with the mid-childhood growth spurt.

These finding were supported by Harvard College's School of Dental Medicine research[1201] which demonstrated that pre-adolescent boys who drink fluoridated water are at a seven-fold increased risk of osteosarcoma, an often fatal bone cancer.

This finding is further acknowledged by the *U.S. National Toxicology Program (NTP)*[1202] who say there is a "biological plausibility" of a link between fluoride exposure and osteosarcoma. The biological plausibility centers around three facts: 1) bone is the principal site of fluoride accumulation, particularly during the growth spurts of childhood; 2) fluoride is a mutagen when present at sufficient concentrations, and 3) fluoride can artificially stimulate the proliferation of bone cells (osteoblasts).

The NRC recommended that further research on a possible effect of fluoride on bladder cancer risk should be conducted. The NRC observed that since bladder cancer is relatively common (compared with osteosarcoma), both cohort and case-control designs would be feasible to address this question.

In examining neuro-chemical effects and mechanisms, the NRC reported that lipids and phospholipids, phosphohydrolases and phospholipase D and protein content were reduced in the brains of laboratory animals subsequent to fluoride exposure. The NRC reported that the greatest changes were found in phosphatidylethanolamine, phosphotidylcholine and phosphotidylserine and that fluorides were also shown to inhibit the activity of cholinesterases, including acetylcholinesterase.

The NRC reported that recently, the number of receptors for acetylcholine has been found to be reduced in regions of the brain thought to be most important for mental stability and for adequate retrieval of memories.

The NRC acknowledged that fluorides also increase the production of free radicals in the brain through several different biological pathways. These changes have a bearing on the possibility that fluorides act to increase the risk of developing Alzheimer's disease and dementia. Dementia affects almost 44,000 people and touches the lives of 50,000 carers and hundreds of thousands of family members therefore any role of fluoride exposure in the development of Alzheimer's or dementia is potentially significant.

[1201] Bassin EB, Wypij D, Davis RB, Mittleman MA. (2006). Age-specific Fluoride Exposure in Drinking Water and Osteosarcoma (United States). Cancer.

[1202] The NTP is an interagency program whose mission is to evaluate agents of public health concern by developing and applying tools of modern toxicology and molecular biology. The program maintains an objective, science-based approach in dealing with critical issues in toxicology and is committed to using the best science available to prioritize, design, conduct, and interpret its studies.

15.0 Risks and Observations noted in the EU Scientific Committee on Health and Environmental Risks (SCHER) Fluoride Report 2010.

The committee observed that there are no systematic data on the concentration of fluoride in natural drinking water in EU member states. The committee noted that In Europe, only Ireland and minor regions in the UK and Spain currently fluoridate drinking water at concentrations ranging from 0.8 to 1.2 mg/L. Rudimentary data shows large variations in fluoride concentrations in drinking water between countries, in particular Ireland (0.01-5.8 mg /L) compared to Finland 0.1- 3.0 mg/L, and Germany 0.1-1.1 mg/L.

While not as detailed or comprehensive as the published NRC study, the SCHER review nevertheless acknowledges many of the human risks from fluoride as previously documented in the NRC study. The human risks reported by SCHER can be summarised as follows;

SCHER stated that the toxicology of hexafluorosilicic acid and hexafluorosilicictes compounds are incompletely investigated. These are the most commonly used agents in drinking water fluoridation and it has been claimed that incomplete dissociation of these agents in drinking water may result in human exposure to these chemicals.

SCHER acknowledged that limited evidence from epidemiological studies points towards adverse health effects following systemic fluoride consumption, e.g., carcinogenicity, developmental neurotoxicity and reproductive toxicity.

SCHER observed that water fluoridation was intended to have a beneficial effect on caries prevention but could also induce fluorosis with a very narrow margin of exposure.

SCHER acknowledged that there is a risk dental fluorosis in children in EU countries with systemic fluoride exposure. In Ireland dental fluorosis affects up 30% of children in communities with fluoridated water compared to 1.5% in non-fluoridated areas. In fluoridated communities it was observed that 1% of children developed moderate dental fluorosis with a further 1% developing severe fluorosis. Neither moderate nor severe fluorosis was observed in children in non-fluoridated communities.[1203]

SCHER reported that the systemic exposure to fluoride in drinking water is associated with an increased risk of dental and bone fluorosis and noted that exposure to fluoride levels during tooth development can result in dental fluorosis and excess systemically absorbed fluoride may impair normal development of enamel in the pre-eruptive tooth.

[1203] Dental Fluorosis In Primary Teeth Of 5-Year-Olds In Ireland D.M. O'mullane1, M. Harding1, H.P. Whelton1, M.S. Cronin1, And J.J. Warren2, 1 University College Cork, Ireland, 2 University Of Iowa, USA

SCHER observed that enamel fluorosis seen in areas with fluoridated water has been attributed to inappropriate high fluoride intake. [1204], [1205]

SCHER observed that water fluoridation, as well as topical fluoride treatments (e.g. fluoridated toothpaste), appears to prevent caries, primarily on permanent dentition, but noted that topical application as opposed to fluoridation of drinking water is the more efficient measure.

SCHER concluded that no obvious advantage appears in favour of water fluoridation compared with topical prevention.

SCHER observed that topical application of fluoride is most effective in preventing tooth decay.

SCHER acknowledged that the benefits of fluoridation to adult and elderly populations in terms of reductions in coronal and root decay are limited.

SCHER stated that the continued systemic exposure of fluoride from whatever source is questionable once the permanent teeth have erupted. Therefore there is no medical benefit to the wider population. One should note that if the State were to claim a medical benefit from water fluoridation then it must comply with relevant EU legislation pertaining to medical products (Directive 2001/83/EC). The State has a legal obligation to ensure that any such product meets relevant health and safety standards.

The SCHER findings observed that the tolerable Upper intake Level (UL), is exceeded for the preparation of infant formula tap water when the maximum permitted fluoride level (1.5 mg/L) according to the Council Directive 98/83/EC of 3 November 1998 on the quality of water intended for human consumption, is used. If the infant's diet consists entirely of formulated food products the risk of developing dental fluorosis increases in areas with high level of fluoride in tap water.

SCHER observed that there is slight evidence that high level occupational exposure to fluoride affects male reproductive hormone levels and that a few studies on human populations have suggested that fluoride might be associated with alterations in reproductive hormones and fertility.

SCHER observed that most of the animal studies on the reproductive effects of fluoride exposure deal with the male reproductive system of mice and rats and that little or no data is available for human studies. SCHER acknowledged that animal studies consistently show an effect on spermatogenesis or male fertility.

1204 Ellewood R, Fejerskov O, Cury JA, Clarkson B. (2008) Fluorides in caries control. IN: Dental Caries. Eds O Fejerskov & E. Kidd. Blackwell & Munksgaard Chapter 18
1205 Forsman B (1977) Early supply of fluoride and enamel fluorosis. Scand J Dent Res. 85:22-30. Forum on Fluoridation Ireland Report (2002) Presented to Micheál Martin TD, Minister for Health and Children. ISBN: 0755-711-572

SCHER reported the recent Harvard case-control study,[1206] which found an association between fluoride exposure during childhood and the incidence of osteosarcoma among males, but not among females (Bassin, 2006).

SCHER accepted that some epidemiological studies seem to indicate a possible link between fluoride in drinking water and osteosarcoma disparities.

SCHER observed that there is no clear association of bone fracture with water fluoridation, however, SCHER reported that fluoride can weaken bone and increase the risk of bone fractures under certain conditions.[1207]

Of the more critical risk concerns to public health that SCHER observed is that there are insufficient human studies to prevent concluding firmly that fluoride intake hampers children's neurodevelopment, impairs IQ or leads to bone fractures. While SCHER may have observed that "*available human studies do not allow concluding firmly that fluoride intake hampers children's neurodevelopment*" it did not question the latest scientific findings or present any shortcomings regarding a recent study which SCHER reported concluded that intake of fluoride in drinking water may contribute to the decreased intelligence in children. [1208]

It is important to note that the authors of this particular study, Rocha-Amador et al. further concluded that "*the risk is particularly acute for children, whose brains are particularly sensitive to environmental toxins. Furthermore, it would be advisable to re-examine the benefits of F given the documented health risks.*" The findings of this study were not disputed by SCHER.

[1206] Bassin EB, Wypij D, Davis RB, Mittleman MA. (2006). Age-specific Fluoride Exposure in Drinking Water and Osteosarcoma (United States). Cancer Causes and Control 17:421-8
[1207] McDonagh M, Whiting P, Bradeley M, Cooper J, Sutton A, Chestnut I, Misso K, Wilson P, Treasure E, Kleijnen J (2000) A systematic report on public water fluoridation. NHS Center for Reviews and Dissemination. York, UK, University of York: 259.
[1208] Rocha-Amador D, Navarro ME, Carrizales L, Morales R, Calderón J, Facultad de Medicina, Universidad Autónoma de San Luis Potosí, San Luis Potosí, México. Decreased intelligence in children and exposure to fluoride and arsenic in drinking water; Cad. Saúde Pública, Rio de Janeiro, 23 Sup 4:S579-S587 (2007).

15.1 Limitations of SCHER Review regarding Examination of Environmental Impact of Fluoride on Ecosystems

In regard to the examination by SCHER of the environmental impacts of fluoride it is unfortunate that the review was so limiting and poorly researched especially given the volume of environmental legislation, the potential environmental impacts for both aquatic and terrestrial ecosystems in addition to overall resource management and sustainable development concerns. The environment is often least considered when water management or public health policies are developed or reviewed. The SCHER review in this regard is an example in kind and its authors themselves noted the study's limitations by stating that the environmental review was simplistic and based on just one published paper.[1209] It is apparent based on the information provided in this report that the SCHER environmental assessment, while not the principle focus of the review, was totally inadequate and its recommendations therefore remain highly contentious and questionable.

It is noted in particular that the observations and conclusion of the reference study on which the review itself was based clearly contradict the subsequent findings of the SCHER committee. In particular, Camargo observed that the toxic action of fluoride resides in the fact that fluoride ions act as enzymatic poisons, inhibiting enzyme activity and, ultimately, interrupting metabolic processes such as glycolysis and synthesis of proteins.

As reviewed elsewhere in this report Camargo observed that fluoride toxicity to aquatic invertebrates and fishes increases with increasing fluoride concentration, exposure time and water temperature, and decreases with increasing intraspecific body size and water content of calcium and chloride. Importantly, it was also observed that freshwater invertebrates and fishes, especially net-spinning caddis fly larvae and upstream-migrating adult salmons, appear to be more sensitive to fluoride toxicity than estuarine and marine animals.

It is expected that the findings of this research, in addition to the research by Neuhold(1960), Angelovic(1961), Damkaer (1989), Groth(1975) and Warrington (1990) as noted elsewhere in this report, highlight the potential impacts of fluoride in wastewater discharges on freshwater ecosystems, in particular juvenile salmon, rainbow trout and Daphnia magna, the main food source for these species. This study will be of particular interest to the Department of Agriculture, Food and the Marine, the Department of the Communications, Energy and Natural Resources, the Department of Environment, Community and Local Government, Inland Fisheries Ireland and the Environmental Protection Agency requiring further detailed examination and assessment of the environmental impacts of fluoride on the environment and inland fisheries given

[1209] Camargo JA (2003) Fluoride toxicity to aquatic organisms: a review. Chemosphere 50: 251-64

the number of wastewater effluent treatment plants located on important fisheries rivers. Anthropogenic fluoride emissions from waste water treatment plants are discharged to over one hundred Salmonid Rivers in Ireland from over 475 waste water treatment works.

Camargo recommended that because, in soft waters with low ionic content, a fluoride concentration as low as 0.5 mg F-/l can adversely affect invertebrates and fishes, safe levels below this fluoride/l concentration are needed to protect freshwater animals from fluoride pollution. It was also noted that aquatic organisms living in soft waters may be more adversely affected by fluoride pollution than those living in hard or seawaters because the bioavailability of fluoride ions is reduced with increasing water hardness.

Camargo also found that fluoride can either inhibit or enhance the population growth of algae, depending upon fluoride concentration, exposure time and algal species. This finding may be of interest to environmental resource managers investigating the unprecedented algae blooms experienced in coastal tidal waters in certain locations around Ireland over the past decade.

The SCHER review in examining the environmental impacts was particularly lacking in not examining the volume of scientific data included in this report, in particular, the information on lifecycle impacts of fluorides on the environment.

SCHER concluded incorrectly (as is evident from the comprehensive studies undertaken by the U.S. EPA and others) that the risks from fluoridation to soils do not give any cause for concern; in particular that sewage sludge is unlikely to become contaminated and, in turn, this means that the contamination of soils and terrestrial systems is unlikely. This has been proved otherwise conclusively by the U.S. EPA.[1210] Therefore it is reasonable to assume that the observations regarding the environmental impact of fluoride on the environment, as noted in the SCHER review, are flawed and incorrect.

In the SCHER mass balance modelling scenario for fluoride in the environment it is however interesting to note that the predicted no-effect concentrations (PNECs) for fluoride is 0.4mg/l for aquatic organisms. For intermittent releases the PNEC for aquatic organisms, was established at 0.146mg/l and for freshwater sediment dwelling organisms the PNEC was 0.313mg/kgwwt-1. As detailed elsewhere in this report, these limits would clearly be exceeded in any wastewater discharges from communities with fluoridation of drinking water.

Incredibly, the SCHER review provided no data for environmental exposure including for soil biota, concentrations in fish, bioaccumulation in earthworms or bioaccumulation factor for fish. Nor were data provided for exposure through intake of fish, leaf crops, root crops, meat or milk. The factor of emission directed to sludge at 3.74% is clearly incorrect given the findings of the comprehensive

[1210] EPA Technical Support Document for the Round Two Sewage Sludge Pollutants, Office of Water, EPA-822-R-96-003, August 1996.

U.S. EPA study which identified fluoride as a pollutant with critical land pollution application pathways for both livestock and soil organisms.

Alarmingly, in the model predictions no data for effects of fluoride on terrestrial organisms, birds and mammals or freshwater sediment organisms were provided. It is obvious that where no data is provided in the SCHER risk assessment, the quality for the predicted outcomes remains highly questionable and the findings unreliable and unscientific.

In the absence of such data, and given the peer-reviewed information reviewed in this report, one cannot conclude with any confidence that the environmental impact of water fluoridation and fluoride pollutants on the environment is not significant. The information presented in this report would conclude that water fluoridation provides numerous exposure pathways for bioaccumulation of fluoride in the environment with clear consequences for ecosystems.

16.0 FLUORIDE HUMAN TOXICITY ASSESSMENTS AND FURTHER RESEARCH IDENTIFIED TO BE UNDERTAKEN ON FLUORIDE

In quantifying the potential public health risk from fluoridation of drinking water, in excess of fifty comprehensive epidemiology, toxicology, clinical medicine, and environmental exposure assessments were identified by the U.S. National Research Council (NRC) and the European Commission's Scientific Committee on Health and Environmental Risks(SCHER). Some of these were regarded as of paramount importance for the protection of public health.

If the current government were to continue with the policy of mandatory fluoridation of drinking water supplies, a thorough examination of the scientific recommendations for further study would need to be undertaken requiring comprehensive and costly research. In light of the recommendations from SCHER which concluded that there is no obvious advantage in favour of water fluoridation and considering the lack of appropiate toxological assessment of fluoride products, the risks posed to the population and the length and cost of scientific, medical and epidemiological research to demonstrate appropiate standards of care, it cannot be justified and must be regarded in the current economic climate as a totally unnecessary cost.

However, should the Government consider that fluoridation policy must continue, disregarding the expert opinion of SCHER, the following studies would be required to be undertaken. In light of the findings and recommendation of the NRC and SCHER reports it would be unconscionable not to protect the public from undue risk.

16.1 Recommended Studies on Intake, Metabolism and Disposition of Fluoride

1. Determine and compare intake of fluoride from all sources, including fluoride-containing dental products, in communities with fluoridated and non-fluoridated water.

2. Determine the effects of factors that affect human acid-base balance and urinary pH on the metabolic characteristics, balance, and tissue concentrations of fluoride.

3. Determine the metabolic characteristics of fluoride in infants, young children, and the elderly.

4. Determine prospectively the metabolic characteristics of fluoride in patients with progressive renal disease.

5. Using preparative and analytical methods now available, determine soft-tissue fluoride concentrations and their relation to plasma fluoride concentrations. Consider the relation of tissue concentrations to

variables of interest, including past fluoride exposure and age.

6. Identify the compounds that compose the "organic fluoride pool" in human plasma and determine their sources, metabolic characteristics, fate, and biological importance.

16.2 Recommended Studies on Enamel Fluorosis

7. Identify sources of fluoride during the critical stages of tooth development in childhood and evaluate the contribution of each source to enamel fluorosis.

8. Conduct studies on the relation between water fluoride concentrations and enamel fluorosis.

9. Improved assessment of exposure to fluoride from all sources is needed for a variety of populations (e.g., different socioeconomic conditions).

10. To the extent possible, exposures should be characterized for individuals rather than communities, and epidemiologic studies should group individuals by exposure level rather than by source of exposure, location of residence, or fluoride concentration in drinking water. Intakes or exposures should be characterized with and without normalization for body weight.

11. Fluoride should be included in nationwide biomonitoring surveys and nutritional studies; in particular, analysis of fluoride in blood and urine samples taken in these surveys would be valuable.

12. Additional studies should be done in communities with water fluoride concentrations greater than 1 mg/L. These studies should focus on moderate and severe enamel fluorosis in relation to caries and in relation to psychological, behavioural, and social effects among affected children, their parents, and affected children after they become adults.[1211]

13. Methods should be developed and validated to objectively assess enamel fluorosis. Consideration should be given to distinguishing between staining or mottling of the anterior teeth and of the posterior teeth so that aesthetic consequences can be more easily assessed.

14. More research is needed on the relation between fluoride exposure and dentin fluorosis and delayed tooth eruption patterns.

[1211] The concentration of fluoride in drinking water increases when water is boiled.

16.3 Recommended Pharmacokinetic Studies

15. The concentrations of fluoride in human bone as a function of exposure concentration, exposure duration, age, sex, and health status should be studied.

16. Such studies would be greatly aided by non-invasive means of measuring bone fluoride.

17. Information is particularly needed on fluoride plasma and bone concentrations in people with small-to-moderate changes in renal function as well as in those with serious renal deficiency.

18. Improved and readily available pharmacokinetic models should be developed. Additional cross-species pharmacokinetic comparisons would help to validate such models.

16.4 Recommended Bone Fracture Studies

19. Evaluate the advantages and disadvantages of the various doses, treatments, laboratory animal models, weight-bearing versus non-weight-bearing bones, and testing methods for bone strength that can be used to determine the effects of fluoride on bone.

20. Conduct additional studies of hip and other fractures in geographic areas with high and low fluoride concentration in drinking water and make use of individual information about water consumption.

21. These studies also should collect individual information on bone fluoride concentrations and intake of fluoride from all sources, as well as reproductive history, past and current hormonal status, intake of dietary and supplemental calcium and other cations, bone density, and other factors that might influence the risk of hip fracture.

16.5 Recommended Further Bone Studies to be undertaken

22. A systematic study of clinical stage II and stage III skeletal fluorosis should be conducted to clarify the relationship between fluoride ingestion, fluoride concentration in bone, and clinical symptoms.

23. More studies of communities with drinking water containing fluoride at 2 mg/L or more are needed to assess potential bone fracture risk at these higher concentrations.

24. Quantitative measures of fracture, such as radiologic assessment of vertebral body collapse, should be used instead of self-reported fractures or hospital records. Moreover, if possible, bone fluoride concentrations should be measured in long-term residents.

16.6 Recommended Studies on Carcinogenicity

25. Conduct more highly focused, carefully designed analytical studies (case control or cohort) of the cancer sites that are most highly suspect, based on data from animal studies and the few suggestions of a carcinogenic effect reported in the epidemiologic literature. Such studies should be designed to gather information on individual study subjects so that adjustments can be made for the potential confounding effects of other risk factors in analyses of individuals.

26. Information on fluoride exposure from sources other than water must be obtained, and estimates of exposure from drinking water should be as accurate as possible. In addition, analysis of fluoride in bone samples from patients and controls would be valuable in inferring total lifetime exposures to fluoride.

27. Among the disease outcomes that warrant separate study are osteosarcomas and cancers of the buccal cavity, kidney, and bones and joints.

28. Further research on a possible effect of fluoride on bladder cancer risk should be conducted. Since bladder cancer is relatively common (compared with osteosarcoma), both cohort and case-control designs would be feasible to address this question. For example, valuable data might be yielded by analyses of cancer outcomes among the cohorts followed for other health outcomes, such as fractures.

16.7 Recommended Other Health Effects Requiring Assessment

29. Carefully conducted studies of exposure to fluoride and emerging health parameters of interest (e.g., endocrine effects and brain function) should be performed in populations exposed to various concentrations of fluoride. It is important that exposures be appropriately documented.
30. Further effort is necessary to characterize the direct and indirect mechanisms of fluoride's action on the endocrine system and the factors that determine the response, if any, in a given individual.

Such studies would address the following:

31. The in vivo effects of fluoride on second messenger function;
32. The in vivo effects of fluoride on various enzymes;
33. The integration of the endocrine system (both internally and with other systems such as the neurological system);
34. Identification of those factors, endogenous (e.g., age, sex, genetic factors, or preexisting disease) or exogenous (e.g., dietary calcium or iodine concentrations, malnutrition), associated with increased likelihood of effects of fluoride exposures in individuals;
35. Consideration of the impact of multiple contaminants (e.g., fluoride and perchlorate) that affect the same endocrine system or mechanism;

Important Exposure Aspects of such studies should include the following:

36. Examination of effects at several time points in the same individuals to identify any transient, reversible, or adaptive responses to fluoride exposure.
37. Better characterization of exposure to fluoride is needed in epidemiology studies investigating potential endocrine effects of fluoride.
38. Collecting data on general dietary status and dietary factors that could influence the response, such as calcium, iodine, selenium, and aluminium intakes.
39. Characterizing and grouping individuals by estimated (total) exposure, rather than by source of exposure, location of residence, fluoride concentration in drinking water, or other surrogates.
40. Reporting intakes or exposures with and without normalization for body weight (e.g., mg/day and mg/kg/day), to reduce some of the uncertainty associated with comparisons of separate studies.
41. Addressing uncertainties associated with exposure and response, including uncertainties in measurements of fluoride concentrations in bodily fluids and tissues and uncertainties in responses (e.g., hormone concentrations).
42. Reporting data in terms of individual correlations between intake and effect, differences in sub-groups, and differences in percentages of individuals showing an effect and not just differences in group or population means.
43. Examining a range of exposures, with normal or control groups taking into consideration mechanism of fluid intakes. In the United Kingdom, where tea drinking is more common as in Ireland, people can consume up to 9 mg of fluoride a day.[1212]
44. Having very low fluoride exposures (below those associated with 1 mg/L in drinking water for humans).
45. The effects of fluoride on various aspects of endocrine function should be examined further, particularly with respect to a possible role in the development of several diseases or mental States.

Major Areas for Investigation should include the following:

46. thyroid disease (especially in light of decreasing iodine intake by the U.S. population);
47. nutritional (calcium deficiency) rickets;
48. calcium metabolism (including measurements of both calcitonin and PTH);
49. pineal function (including, but not limited to, melatonin production); and development of glucose intolerance and diabetes.

[1212] Jenkins, G.N. 1991. Fluoride intake and its safety among heavy tea drinkers in a British fluoridated city. Proc. Finn. Dent. Soc. 87(4):571-579.

16.8 Recommended Studies on Neurochemical and Biochemical Change

50. On the basis of information largely derived from histological, chemical, and molecular studies, it is apparent that fluorides have the ability to interfere with the functions of the brain and the body by direct and indirect means.
51. To determine the possible adverse effects of fluoride, additional data from both the experimental and the clinical sciences are needed.

16.9 Recommended Studies on Renal and Hepatic Effects

52. There is a particular need to study patients with renal impairments.
53. Additional studies should be carried out to determine the incidence, prevalence, and severity of renal osteodystrophy in patients with renal impairments in areas where there is fluoridation of the drinking water.
54. The effect of low doses of fluoride on kidney and liver enzyme functions in humans needs to be carefully documented in communities exposed to different concentrations of fluoride in drinking water.

16.10 Recommended Studies on Immune Response

55. Epidemiologic studies should be carried out to determine whether there is a higher prevalence of hypersensitivity reactions in areas where there is fluoridation of drinking water.
56. In addition, studies could be conducted to determine what percentage of immunocompromised subjects have adverse reactions when exposed to fluoride in the range of 1-4 mg/L in drinking water (taking into consideration fluoride exposure through boiling fluoridinated drinking water and tea drinking).
57. More research is needed on the immunotoxic effects of fluoride in animals and humans to determine if fluoride accumulation can influence immune function.
58. It is paramount that careful biochemical studies be conducted to determine what fluoride concentrations occur in the bone and surrounding interstitial fluids from exposure to fluoride in drinking water, because bone marrow is the source of the progenitors that produce the immune system cells.

16.11 Recommended Studies in Genotoxicity

59. In vivo human genotoxicity studies in U.S. populations or other populations with nutritional and sociodemographic variables similar to those in the United States should be conducted.

17.0 CONCLUSION

It is abundantly clear from the assessment of documentation reviewed that the epidemiological evidence supporting continued fluoridation of water supplies appears weak and that the risks to public health and the environment, from the mandatory fluoridation of drinking water supplies, far outweigh any perceived benefits to dental health.

It has been extensively documented in published peer-reviewed scientific reports that gaps in the information on fluoride presents certain safety and public health risks that require further immediate research into the risks of fluoridation on the wider population.

The State must ensure that all adequate measures have been taken to inform the consumer of potential health risks associated with fluoridated water and that the regulatory authorities implement adequate health surveillance programmes to monitor the total fluoride intake of the population including infants and sensitive sub-groups.

The government and regulatory authorities must take into consideration their legal duties to ensure the health of the consumer and protection of the environment; in particular ensuring the safety of any product added to drinking water supplies for consumption by the public through robust scientific risk assessment and environmental impact assessment.

The State must take into consideration ethical, economic, environmental and societal concerns regarding continued fluoridation of drinking water supplies and, in particular, evolve public policy to keep pace with current scientific research and development.

The State has an obligation to develop and update public policy based on current scientific research. When you examine the most up-to-date peer-reviewed scientific documentation that informs water management, environmental and public health policy in the USA and in Europe, the information for the public and consumer is both alarming and disturbing. The information contained in the two most recent and reputable international publications, comprising the most comprehensive scientific assessments undertaken to date on the fluoridation of drinking water, provides clear observations, recommendations and conclusions that must raise serious concerns for the State and local government.

In determining the cost benefit analysis of fluoridation, one must examine the cost of assurance in relation to health protection, the absence of proper risk assessment of fluoridation products and the potential benefit of fluoridation of public drinking waters. Given the numerous and potentially dangerous and hazardous risks that have been identified (regardless of the probability or level of risk) where such a risk exists the cost to the State and society clearly outweighs any potential public health benefit.

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The fact that the SCHER review concluded that there is no social or economic advantage to the continued fluoridation of drinking water supplies must be acknowledged by the State given that the remit of SCHER is to provide expert advice to the European commission and its member states.

Where further research has been recommended by expert bodies or scientific committees the State must address targeting such research, to address potential concerns and gaps in information that currently exist. To date this has not been undertaken by the State and a substantial cost and economic burden would be involved in meeting this undertaking.

Where potential hazards have been documented and appropriate action recommended, the State is held to be accountable and liable for any potential liability that may result from continued mandatory fluoridation; including its avoidance to undertake the necessary rigorous scientific examination of the risks through adequate epidemiology, toxicology, clinical medicine and environmental exposure assessments.

The SCHER and NRC reports identified additional epidemiology, toxicology, clinical medicine, and environmental exposure assessments that need to be undertaken in order to fill data gaps in the hazard profile, the health effects and the exposure assessment of fluoride. In addition, further research was recommended in assessing the health and environmental risks that may be associated with the use of the most common drinking water fluoridation agents like silicofluorides, taking into account their hazard profiles, their mode of use in water fluoridation, their physical chemical behaviour when diluted in water and the possible adverse effects they may have in exacerbating fluoride health effects as reported in some scientific studies.

This information cannot be ignored and must be acted on by the Government of Ireland if it intends to continue with mandatory fluoridation of public water supplies.

The U.S. National Research Council [whose members are drawn from the councils of the National Academy of Sciences, the National Academy of Engineering and the Institute of Medicine], the professional unions representing the U.S. Environmental Protection Agency staff and the European Union's Scientific Committee on Health and Environmental Risks [comprising the Scientific Committee on Consumer Safety and the European Food Safety Authority]; in addition to other expert committees have scientifically examined and evaluated the practice of water fluoridation and concluded that fluoridation of drinking water supplies presents a public health risk, particularly for infants.

The SCHER and NRC review have demonstrated that the epidemiological evidence supporting continued fluoridation of water supplies is weak, that its continued practice is unnecessary and that the risks to public health may far outweigh its perceived benefits.

Against a background of exposure to multiple sources of fluoride, changes in the rates of dental decay and a rapidly increasing incidence of dental fluorosis on both a population and individual level in Ireland and given the perceived and known risks to sensitive sub-population groups, such as infants, patients at risk of diabetes or individuals managing diabetes and the high incidence of certain cancers, neurological conditions and coronary disease in Ireland; it is considered appropriate to use every caution to minimize the exposure to potentially dangerous compounds such as fluoride to acceptable levels.

It is obvious that the State cannot track each individual's response to mandatory medication through fluoridation of drinking water supplies and that this practice is resulting in over-exposure of the public to this anthropogenic contaminant, thereby exposing the population to increased health risks. The systemic exposure of the population to fluoride in drinking water, even in relatively low concentrations is now known to cause negative health effects and there is evidence to suggest that it may also be directly associated with a wide range of further much more serious medical conditions, some fatal. It is now sufficiently clear that the State can no longer continue to justify mandatory fluoridation of drinking water.

It is obvious that the State must accept its responsibility to protect the individual consumer and the environment (including soil quality, groundwater and surface water and their ecosystems) from fluoride contamination.

This cannot be achieved by continuing with a policy of water fluoridation. To continue with this policy in the absence of a full toxicological and environmental impact assessment of the fluoridation products would present liability for negligence by the State, especially where there are known risks associated with sensitive sub-population groups within society.

In undertaking its duty, the State must consider the potentially significant costs to ensure that adequate epidemiology, toxicology, clinical medicine and environmental exposure assessments are undertaken to protect the consumer as well as environmental impact assessment to protect the environment and ecosystems including fisheries.

It is obvious that the State must acknowledge the clear findings from the SCHER Review to the European Commissioner for Consumer Protection that the continued systemic exposure of the population to fluoride in drinking water is highly questionable and not without public health risk. The State must accept the findings that no obvious advantage appears in favour of water fluoridation to prevent dental caries compared with topical prevention and that topical application of fluoride is the most effective action in preventing tooth decay. The Government must also acknowledge that the ingestion of fluoride through drinking water has health risks as fluoride concentrates in human bone and can compromise the health and well-being of individuals with sensitive gastrointestional systems as well as individuals who are immunocompromised. The Government must accept that international scientific experts have concluded that water fluoridation can cause chronic fluoride poisoning in the population exposing infants to the highest risk factor that can result in

permanent and irreversible health issues. There are other potential health risks that may be associated with water fluoridation that require immediate research as outlined in the previous chapter.

The environmental risks of water fluoridation have also been examined in this report. It has been demonstrated that the policy of water fluoridation remains one of the most unsustainable and environmentally damaging public health policies ever implemented. The application of this policy in Ireland is resulting in numerous significant violations of various EU Directives for the protection of groundwater, surface waters, the environment and human health. These have been discussed in detail within this report. The policy of water fluoridation in Ireland results in the indirect release of the pollutant fluoride into soils, groundwater and surface waters in significant concentrations. Given that fluoride is classified as a persistent inorganic pollutant, an element that bioaccumulates within the environment and food chain, this degree of sustained pollution of surface water, groundwater and soils is entirely unacceptable.

In the age of sustainability with ever-growing pressures on the environment such an inept and inefficient policy is completely unnecessary, unsustainable and unacceptable. It would be considered that to continue to fluoridate drinking water based on the established scientific facts, as presented within this report and elsewhere, would be against the fundamental principles of toxicology, pharmacology, environmental sustainability and democratic ethics.

As previously noted, Ireland remains a lone country within Europe with its legislative policy of mandatory water fluoridation. If the State were to continue with the policy of mandatory fluoridation of drinking water supplies, it has at minimum a legal duty of care to ensure that the risk assessments and recommendations identified in this report to protect public health and well-being (as outlined in international peer-reviewed scientific studies) are acted on immediately by the health service authority in Ireland. This has not been undertaken to date by the State. Failure to continue to implement these recommendations to their fullest will undoubtedly have legal and financial ramifications for the State.

It is simply unconscionable that the State would continue to fluoridate public water supplies when the overwhelming weight of scientific and medical evidence clearly demonstrates that the practice has little or no positive benefit to public health but rather exposes the public to unnecessary risk and places an unacceptable environmental burden of pollution on the environment.

Given the controversial and alarming scientific findings presented in this and other reports and the continued exposure of the population to unnecessary risks, as identified by international scientific committees and scientific experts, it is appropriate that this policy be reviewed immediately.

Clearly the most obvious and economically prudent approach would be to cease fluoridation of drinking water in accordance with the recommendations of the EU's Scientific Committee on Health and Environmental Risks (SCHER) as

provided to the European Commission's Director General for Health & Consumers in 2010.

As it currently stands, the following western European countries have rejected water fluoridation: Austria, Belgium, Denmark, Finland, France, Germany, Iceland, Italy, Luxembourg, Netherlands, Norway, Sweden and Switzerland.

In the second decade of the 21st century Ireland remains the only member state within the European Community with a nationally mandated drinking water fluoridation programme, a public health programme that may be regarded as belonging to the 20th century. It is important to note that other European countries such as Denmark banned water fluoridation when its National Agency for Environmental Protection, after consulting the widest possible range of scientific sources, pointed out that the long-term effects of low-fluoride intakes on certain groups in the population (for example, persons with reduced kidney function), were insufficiently known[1213] .

Sweden also rejected water fluoridation on the recommendation of a special Fluoride Commission, which included among its reasons that: "(t)*he combined and long-term environmental effects of fluoride are insufficiently known*" [1214]. Holland banned fluoridation after a group of medical practitioners presented evidence that it caused reversible neuromuscular and gastrointestinal harm to some individuals in the population[1215.] Other European countries such as the Czech Republic ended water fluoridation in the early 1990's. Following a scientific and economic review in 2003 in the city of Basel, the only remaining city in Switzerland where water fluoridation occurred, the policy was terminated.

The Chairman of the UK Advisory Group for the Systematic Review of Fluoridation (YORK Review), Professor Trevor A. Sheldon confirmed unequivocally that: "*the review did not show fluoridation to be safe, found water fluoridation to be significantly associated with high levels of dental fluorosis which was not characterised as "just a cosmetic issue" and that there was little evidence to show that water fluoridation has reduced social inequalities in dental health*."

Like many post-World War Two countries in Europe, the Czech Republic undertook water fluoridation of water supplies for a period up to 1993 when the practice was terminated. A government spokeperson noted[1216] (below) that fluoridation of drinking water is no longer under consideration in the Czech Republic because this form of supplementation is considered:

- Uneconomical (only 0.54% of water is used for drinking)

[1213] *Nyt fra miljøstyrelsen* (Newsletter of National Agency of Environmental Protection, Denmark). Special issue (in English), February, 1977

[1214] *Flour i karies- förebyggande syfte* (Report of Swedish Fluoride Commission). Statens Offentliga Utredningar, Stockholm 1981. English-language summary pp 21-30

[1215] Grimbergen GW. A double blind test for determination of intolerance to fluoridated water (preliminary report). *Fluoride* 7 146-152 1974

[1216] Dr. B. Havlik, Ministerstvo Zdravotnictvi Ceske Republiky.

- Unecological (environmental load by a foreign substance).
- Unethical ("forced medication").
- Toxicologically and physiologically debatable (fluoridation represents an untargeted form of supplementation which disregards actual individual intake and requirements and may lead to excessive health-threatening intake in certain population groups; [and] complexation of fluoride in water into non-biological active forms of fluoride.

It is clear from this report that the previous observations apply equally to Ireland. The government must develop public policy based on current scientific knowledge. In the current economic crisis continuing with such an ineffective, harmful and environmentally unsound policy is a waste of much need public monies that could be spent on far more critical health or social programmes. In every single category from cost to public benefit, the environment, the law, ethics and science the practice of mandatory water fluoridation can no longer be justified.

It is alarmingly that as far back as 1991 the U.S Public Health Service recommended further research be undertaken including conducting analytical epidemiological studies to determine the relationship, if any, among fluoride intake, fluoride bone levels, diet, body levels of nutrients such as calcium, research on bone fractures as well as studies on the reproductive toxicity of fluoride using various dose levels including the minimally toxic maternal dose and further studies to investigate whether or not fluoride is genotoxic. It is disturbing that similar recommendations were made in the York Review (2001), the NRC review (2006) and the SCHER review (2010). While the policy of water fluoridation continues in Ireland it is politically, morally and ethically unacceptable that the public and ultimate consumers of fluoridated water still await the commencement of comprehensive epidemiology, toxicology, clinical medicine and environmental exposure assessments.

It is clear, based on the information contained herein, that the Health Service Executive, Food Safety Authority, the Environmental Protection Agency, Inland Fisheries and other State Agencies and Departments must adopt a precautionary approach to risk prevention and in doing so align Ireland with every other European country by ending the legislative policy of water fluoridation immediately in accordance with the principles of European and international law.

It is apparent that by ending the policy of water fluoridation (in line with all European countries) it would save the Exchequer much-needed tax revenue that could be spent on critical healthcare, education, infrastructure or community social programmes. It is also apparent, given the alarming number of potential and known health risks and diseases caused by exposure to fluoride, that the single biggest contribution and potential cost saving exercise that the current government could make, to support the Health Service in Ireland while benefiting the health and welfare of its citizens could be achieved by following the precautionary approach and ending the policy of water fluoridation forthwith.

To conclude in the words of the Taoiseach, Enda Kenny T.D., in his first State of the Nation address:[1217]

"We will also do all we can to protect the most vulnerable in our communities – our children, the sick, and the elderly" And *"Ireland is an island nation – we cannot operate in isolation. We are part of the European Union."*

Ireland remains the only European country with statutory legislation for water fluoridation. It is clear that the continued implementation of such an ineffective, unsustainable and environmentally harmful policy puts children, the sick and the elderly in harm's way.

It is clearly time that Irish Citizens and the natural environment in Ireland had the same rights and protection as provided to our fellow European member states.

It is time for the practice of mandatory fluoridation of public water supplies to end. There is little doubt that failure to do so, based on the evidence presented in this report, will result in legal action against the State for negligent breach of duty in continuing to allow an untested product that is known to be harmful to humans to be added to public drinking water supplies.

Good governance should respect the rights of citizens and parents to safeguard their personal health and protect their children's wellbeing. Good governance should enhance human rights as provided in the Charter for Fundamental Rights of the European Union, the EU Treaty of Europe and United Nations Treaties mandating that all signatory states, which include the Government of Ireland, comply with the 'Precautionary Principle'.

Failure to do so, in light of the findings in this report, would represent a gross failure of responsibilty and political leadership.

[1217] State of the Nation Address by Taoiseach Enda Kenny 4TH December 2011

Appendix 1:

Peer-Reviewed Journals Reporting Findings on Fluoride Toxicity or related research supporting these findings.

Acta Odontologica Scandinavica Journal	Biochemica. Biophysica. Acta: Molecular and Cell Biology of Lipids
Advanced Dental Research	Biochemical and Biophysical Research
Advanced Dental Research	Biochemistry Journal
Age and Ageing	Biochimica et Biophysica Acta
American Chemistry Society	Biological Trace Element Research
American Heart Journal	Biology of the Neonate Journal
American Journal of Clinical Nutrition	Biomedical &Environmental Sciences
American Journal of Epidemiology	Biostatistics
American Journal of Epidemiology	Blood
American Journal of Geriatric Psychiatry	BMC Cancer Journal
American Journal of Medical Genetics	Bone
American Journal of Medicine	Bone and Mineral Research
American Journal of Obstetric Gynecology	Brain Research
American Journal of Physical Anthropology	Brazialian Journal oF Medical and biological Research
American Journal of Physiology	British Journal of Cancer
American Journal of Public Health	British Journal of Haematology
American Journal of Respiratory Critical Care Medicine	British Journal of Pharmacology
Anales Espanoles de Pediatria	British Journal of Radiology
Annals of Clinical Laboratory Science	British Medical Journal
Annals of Internal Medicine	Bulletin de 1'Academie Nationale Medecine
Annals of New York Academy of Sciences	Bulletin of Environmental Contamination and Toxicology
Annals of Periodontology	Cad. Saúde Pública
Annals of the New York Academy of Sciences	Calcified Tissue International
Annual Review of Physiology	Canadian Journal of Cardiology
Annual Review of Psychology	Canadian Journal of Genetics and Cytology
Applied Statistics	Canadian Journal of Physiology and Pharmacology
Aquatic Insects	Cancer Treatment and Research
Archives of Environmental Contamination and. Toxicology	Caries Research
Archives of Environmental Health	Chemico-Biological Interactions
Archives of Industrial Health	Chemosphere
Archives of Neurology	Circulation
Archives of Toxicology	Clinical Chemistry
Arteriosclerosis Thrombosis and	Clinical Infectious Diseases

Vascular Biology Journal	
Arzneimittel-Forschung.	Clinical Oral Investigations
Australian and New Zealand Journal of Public Health	Clinical Research
Australian Dental Journal	Folia Medica Cracoviensia
Community Dental Oral Epidemiology	Geochemical et Cosmochimica Acta
Community Dentistry and Oral Epidemiology	Gigiena I Sanitariia
Comparative Biochemistry and Physiology	Human and Experimental Toxicology
Critical Reviews in Oral Biology & Medicine	International Journal of Experimental Biology
Current Vascular Pharmacology	Industrial & Engineering Chemistry
Dementia	Institute of Evolutionary Physiology and Biochemistry, Russian Academy of Sciences.
Dementia and Geriatric Cognitive Disorders	International Archives of Allergy & Immunology
Diabetes	International Clinical Nutrition Review
Diabetologia	Human and Experimental Toxicology
Endocrine	International Journal of Experimental Biology
Environmental Pollution	International Journal Clinical Pharmacology Therapy and Toxicology
Environmental Health Perspectives	International Journal of Cancer
Environmental Research	International Journal of Clinical and Experimental Medicine
Environmental Science & Technology	International Journal of Environmental Research
Environmental Toxicology	International Journal of Geriatric Psychiatry
Environmental Toxicology and Chemistry	International Journal of Human and Experimental. Toxicology
Environmental Toxicology and Pharmacology	Irish Journal of Medical Science
Epidemiology	Journal of Fish Research Board Canada
Estuarine Coastal Shelf Science	Japanese Journal of Cancer Research
European Food and Food Law Review	Journal Applied Chemistry
European Journal Clinical Investigation	Journal of American Dental Association
European Journal of Clinical Pharmacology	Journal of American Medical Association
European Journal of Dentistry	Journal of Anatomy
European Journal of Human Genetics	Journal of Animal Science
European Journal of Neurology	Journal of Applied Genetics
European Journal of Oral Science	Journal of Applied Physiology
European Neurology	Journal of Applied Physiology
Experimental Biology and Medicine	Journal of Biological Chemistry

Experimental Cell Research	Journal of Bone Mineral Research
Fluoride	Journal of Clinical Investigation
Journal of Bone and Mineral Research	Journal of Clinical Pediatric Dentistry
Journal of Clinical Investigation	Journal of Dental Medicine
Journal of Clinical Pediatric Dentistry	Journal of Dental Research
Journal of Dental Medicine	Neurobiology of Aging
Journal of Dentistry	Neurologia
Journal of Enzyme Inhibition	Neuron
Journal of Food and Chemical Toxicology	Neurotoxicology
Journal of Food Science	Neurotoxicology and Teratology
Journal of Gastroenterology	New England Journal of Medicine
Journal of Human Lactation	North American Journal of Fish Management
Journal of Industrial Hygiene and Toxicology	Nuclear Medicine Communications
Journal of Industrial Microbiology and Biotechnology	Oral Epidemiology
Journal of Inherited Metabolic Disease	Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology
Journal of International Academy of Periodontology	Osteoporosis International
Journal of Inorganic Chemistry	Paediatric Child Health
Journal of Medical Case Reports	Pathophysiology
Journal of Medical Chemistry 1993	Pediatric Dentistry
Journal of Medicinal Chemistry	Pediatric Research
Journal of National Cancer Institute	Pediatric Surgery International
Journal of Neurology	PEDIATRICS
Journal of Neuropathology	Pharmacological Reviews
Journal of Neuroscience	Physiological Research
Journal of Nutrition	Proceedings of Finnish Dental Society
Journal of Pediatric Nursing	Progress in Neuropsychopharmacol Biological Psychiatry Journal
Journal of Periodontology	Psychoneuroendocrinology.
Journal of Public Health Dentistry	Royal College of Surgeons in Ireland Student Medical Journal
Journal of The Spanish Neurology Society	Science
Journal of Toxicology and Environmental Health	Science of the Total Environment
Journal of Water Pollution Control Federation	Sarcoidosis Vasculitis And Diffuse Lung Diseases Journal
Journal of Wildlife Diseases	Pharmacological Reviews
Lancet	Physiological Research
Medical Science Monitor	Proceedings of Finnish Dental Society
Medicina	Talanta: Pure and Applied Chemistry
Medicine	Teratology
Medscape	The American Journal of Clinical Nutrition

Metabolism	The Biochemical Journal
Molecular and Cellular Biochemistry	The European Food Safety Authority Journal
Molecular Neurobiology	The FASEB Journal
Mutation Research	The International Journal of Angiology
Nature	The Journal of Biological Chemistry
Nature Neuroscience	The Journal of Neuroscience
Nature Reviews Neuroscience	The International Journal of Pure and Applied Analytical Chemistry
The Journal of Nutrition	World Applied Sciences Journal
The Journal of Pedodontics	
The Journal of the Alzheimer's Association	
The official publication of the American Federation for Clinical Research	
Toxicological Sciences	
Toxicology	
Toxicology In Vitro	
Trends BioChem Sci	
Urological Research	
Vascular Medicine	
Water	
Water Research	
Water Science Technology	
World Applied Sciences Journal	
World Journal of Gastroenterology	

Appendix 2:

Letter from Professor Trevor A. Sheldon, Chair of the NHS York Advisory Group for the Systematic Review on the Effects of Water Fluoridation

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3/1/2001

In my capacity of chair of the Advisory Group for the systematic review on the effects of water fluoridation recently conducted by the NHS Centre for Reviews and Dissemination the University of York and as its founding director, I am concerned that the results of the review have been widely misrepresented. The review was exceptional in this field in that it was conducted by an independent group to the highest international scientific standards and a summary has been published in the British Medical Journal. It is particularly worrying then that statements which mislead the public about the review's findings have been made in press releases and briefings by the British Dental Association, the British Medical Association, the National Alliance for Equity in Dental Health and the British Fluoridation Society. I should like to correct some of these errors.

1. Whilst there is evidence that water fluoridation is effective at reducing caries, the quality of the studies was generally moderate and the size of the estimated benefit, only of the order of 15%, is far from "massive".
2. The review found water fluoridation to be significantly associated with high levels of dental fluorosis which was not characterised as "just a cosmetic issue".
3. The review did not show water fluoridation to be safe. The quality of the research was too poor to establish with confidence whether or not there are potentially important adverse effects in addition to the high levels of fluorosis. The report recommended that more research was needed.
4. There was little evidence to show that water fluoridation has reduced social inequalities in dental health.
5. The review could come to no conclusion as to the cost-effectiveness of water fluoridation or whether there are different effects between natural or artificial fluoridation.
6. Probably because of the rigour with which this review was conducted, these findings are more cautious and less conclusive than in most previous reviews.
7. The review team was surprised that in spite of the large number of studies carried out over several decades there is a dearth of reliable evidence with which to inform policy. Until high quality studies are undertaken providing more definite evidence, there will continue to be legitimate scientific controversy over the likely effects and costs of water fluoridation.

(Signed) T.A. Sheldon,

Professor Trevor Sheldon, MSc, MSc, DSc, FMedSci.

Appendix 3:

My Views on the Fluoridation of Water, Professor Robert L. Isaacson, Member, National Research Council NRC Committee for the evaluation of possible hazards of fluoride in drinking water.

Professor Robert L. Isaacson, **Emeritus, Distinguished Professor of Psychology** Ph.D., Binghamton University, New York. Former Director, Center for Neurobehavioral Sciences, Past President, International Behavioral Neuroscience Society (IBNS), Fellow: American Psychological Society, Fellow: IBNS, Member, American Physiologic Society and Visiting Lecturer for Minority Institution, Member Editorial Boards: Brain Research . Past service on several NIH and NIMH Review Panels and Committees, Chairman and member of several committees for the Society for Neuroscience. Member, National Research Council NRC Committee for the evaluation of possible hazards of fluoride in drinking water.

My Views on the Fluoridation of Water

Robert L. Isaacson
Distinguished Professor of Psychology
Binghamton University

A note on terminology**: Fluorine** is an element in the halogen group as are chlorine and iodine. Of all the known elements, fluorine is the most chemically reactive, most powerful oxidizing, and most electronegative element. It is a stronger oxidizing element than ozone. It reacts with many compounds at room temperature. It is never found in its pure form in nature.

Fluoride: Any combination of fluorine with another element or chemical group of elements. Thus, the addition of fluorides to the drinking water can indicate the addition of a large number of chemical agents. The most commonly used fluorides for this purpose are sodium fluoride, NaF, and compounds that contain both fluorine and silicon. Such agents are collectively called "Fluorosilicictes." They include fluorosilicic acid, fluorosilicicte, hydrofluosilicic acid, and hexafluorosilicic acid.

In 2003 when I accepted an invitation to join the National Research Council's Committee formed to evaluate the EPA standards for the amount of fluoride that should be allowed in our drinking water, I had no fixed opinion on whether or not fluoride should be added to drinking water. Probably I was asked to serve on the committee because I had organized a series of experiments published between 1993 and 1998 using rats to study the effects of chronic administration of aluminum fluoride in their drinking water. My primary interest was in the effects of aluminum on the brain and behavior. Aluminum fluoride was used because fluoride facilitates the passage of aluminum into the brain. At the time, aluminum was considered by a number of scientists to be an important factor in Alzheimer's disease. Scientists are still actively investigating this possibility. Our studies had to include the investigation of the effects of the fluoride since the aluminum and the fluoride readily become associated after ingestion. In essence we wanted to know the effects of the aluminum, fluoride, and the aluminum fluoride complex[1].

In my more than three years working on the National Research Council Committee I learned about the many influences fluoride has on the nervous system and the brain. I also learned about the variety of ways in which people become exposed to it and the work that had been done in trying to determine if fluorides were a hazard to human health and well being. The results and recommendations of this Committee were published late in 2006.[2]

Slowly, I came to the conclusion that there were strong experimental and clinical indications that fluorides present health hazards to people in many ways. The more I learned, the more I became convinced that the addition of fluorides to drinking water was, and is, a mistake. Accordingly, I decided to share some of my conclusions with any who might wish to know them.

Fluorine-containing compounds can affect every living animal and person. Exposure to fluorides can come from the air, the water, and the foods we eat. Fluoride compounds were long used as insecticides. They were especially effective for ants and roaches. Their containers were always boldly marked as a poison and there were warnings on the label to keep them well away from children. This is mentioned only to note that for many years fluorides have been considered to be major health hazards.

In regard to health the total accumulation of fluorine in the body is important. Only about half of the amount of fluorides taken in by a person is excreted. The rest stays in the body.

Toxic effects are determined by the amount of fluoride stored in the body, current exposure level, and age at the time of exposure. In addition each person has his or her own tolerance level for fluorides. Once this level is exceeded however, dysfunctions of body and/or brain will occur. How these dysfunctions will be expressed depends on the genetic makeup and past experiences of the person. Another factor that helps determine a person's sensitivity to fluoride is their age. Both the
very young and the very old are most likely to be adversely affected after exposure to fluorides.

As noted, different people exhibit a wide range of toxic reactions to fluorides. Some people affected by fluorides complain of general weakness and chronic fatigue, others complain of cramp-like pains in the abdomen, or nausea. Still others express toxin-induced effects by diminished vision, headaches, migraine attacks, or pains in muscles and joints. These fluoride effects have been described in books by Leo Spira (1950, 1959) [3] and George Waldbott and his associates (1978).[4] It is difficult to determine whether or not a given set of symptoms is a consequence of fluoride intake. It is first necessary to rule out the presence of other diseases that could produce the observed symptoms. A correct diagnosis is best shown by repeated observations of an individual when drinking pure water or water contaminated with a fluoride. These exposures must last for periods of a week or two under conditions in which the patient doesn't know which type of water is being consumed. If the symptoms disappear when the person is drinking pure water and return with the resumption of drinking the fluoridetreated water, this is evidence that the problems arise from the fluoride. Leo Spira and George Waldbott and his associates used this type of experimental approach in their research.

Since people vary so much in their sensitivities to fluorides and also in the nature of their symptoms caused by this toxin, determination of a uniform "safe" level of exposure for everyone is impossible. In a way, fluorides are like ozone: there is no really "safe" level that would protect everyone. The Congressional Safe Drinking Water Act instructed that the level of fluoride in drinking water should be set so as to be safe for *everyone* regardless of age or overall health.

Increasing the problems that can be induced by fluorine in its different forms is its ability to enhance the effects of other toxins to which we are exposed. For example, fluorides in the drinking water accelerate the absorption of lead, aluminum, and silicon into the body and brain. The toxic effects of lead have been known for hundreds of years. In recent years the focus of attention has been on the learning deficits lead produces in children. The mechanisms proposed for the induction of this effect are not known entirely but there is evidence that many of the most important neurotransmitters of the brain are being affected. These include alterations in dopaminergic, cholinergic, and glutaminergic systems as well as in the "supportive" glia cells of the brain. There is also evidence that lead toxicity may go beyond impairments of intelligence. Indeed, lead toxicity may produce behavioral changes that include loss of impulse control and a related increase in the frequency of violent acts.

The health hazards associated with enhanced incorporation of lead are not induced by all fluorides but primarily, and maybe only, by the addition of a silicofluoride to our drinking water. The fluoride most often added to our drinking water is hexafluorosilicic acid. This fluorosilicicte dissociates when it enters the body. One component contains silicon and another fluorides. As a consequence when silicofluorides are added to our drinking water there are really two toxic hazards: one coming from the fluoride and another from the silicon. Silicon can produce its own toxic effects including the formation of solids (silica and silicates) that can lodge anywhere in the body. In addition the silicon portion also can generate destructive hydroxyl ions in many organs including the brain. The brain damage caused by the production of these free radicals has been related to anti-social behavioral actions and violence.[6] Recently data from 327 towns and cities,

some having fluoridated water and others not, have been compared in terms of crime rates. All the communities with fluoridated water had substantially higher rates than did those with non-fluoridated water. This indicates that fluorides can act to enhance the damage being done by other toxins.

The impairment of intelligence from lead toxicity is now well established. It is possible that fluorides can produce negative effects on measured intelligence also. The country devoting the greatest attention to this possibility is China. As of February 2007, several groups of Chinese investigators had published over 20 scientific papers on this topic. Scientists from many different areas of China participated in these investigations. The children studied in these reports ranged in age from 4 to 14. All were tested by the same or very similar standardized I.Q. tests. Overall the results came from children tested at different places, at different ages, and tested by different investigators. All the results from China have found that communities with high levels of fluoride in their drinking water have fewer children scoring at the "bright" end of the intelligence spectrum than communities with low or no level of fluoride. Since China does not fluoridate their drinking water, the Chinese studies compare the I.Q. scores of children from towns and school areas that differ in the amount of fluoride naturally present in their water supplies. While not all of Chinese studies were perfectly designed, the large number of studies showing the same pattern of results calls for our attention. A negative effect of fluoride on intelligence seems to be a possibility. Other studies in China have indicated that fluoride exposure in the drinking water of mothers during the 6th to 8th months of pregnancy can produce anatomical changes in the fetal brains. There are also reports of impaired responsiveness to visual and auditory stimuli in babies in the first three days after birth induced by the intake of fluoridated water by young mothers during gestation.[7]

The ingestion of fluoride tends to increase the uptake of aluminum by the brain. In the studies done in my laboratory the increase in aluminum in the brains of rats was *not* a function of the amount of aluminum fluoride given the animals in their drinking water. The smallest dose of aluminum fluoride produced about the same amount of aluminum in the brain as a dose 10 or even a 100 times larger. A small amount of fluoride seems capable of opening aluminum pathways to a maximal degree. It is of great interest that the relative risk of having Alzheimer's disease is increased when individuals had high amounts of aluminum in the brain coupled with low amounts of fluoride.[8] Another observation of interest is that aluminum by itself may not exert toxic affects on the nervous system. It may only become a toxin after joined together with a fluoride to become an aluminum fluoride. 9
The chronic administration of fluorides in rats produces changes in the microscopic structure of the brain. There were significant losses of cells in areas of the hippocampus and the neocortex. Many apparently dead or dying cells were found in areas analogous to locations in which similar dying cells are found in the brains of Alzheimer's patients.

A common and, perhaps universal, characteristic of dementia is a reduction of aerobic metabolism in the brain. The blood supply reaching the brain is the primary supplier of oxygen and nutrients. Reductions in this sole source of brain energy can be due to a number of physical or chemical changes. When the brains of animals chronically exposed to aluminum fluoride were examined histologically, deposits of aluminum-based crystals were found along the walls of both large and small blood vessels in the brain. Similar deposits were also found in the center of many vessels suspended by collagen fibers. These deposits decreased the normal transfer of oxygen from the red blood cells to the brain since they must have created turbulence in its blood flow. It is of historical interest that Alois Alzheimer, the man for whom a type of dementia was named, noted that most patients with this disorder suffered from atherosclerosis in addition to other brain anomalies. This condition is one in which there are deposits formed on the sides and in the center of arteries in the brain. The deposits disrupt the flow of blood to the brain often cause severe brain damage.

Brain functions are entirely dependent on the availability of oxygen. The brain itself consumes 20% of all the oxygen used by the entire body. The brain area most affected by the reduction in oxygen availability is the forebrain. The lower centers of the brain, namely the midbrain and hindbrain, are more resistant to oxygen deprivation. This is why the higher functions of the brain are the first to be affected, as well as the most affected, by oxygen deprivation. Basic motor and visceral functions are often spared even in patients with profound interruptions of normal blood supplies to the brain. One of the best-known chemical alterations produced by fluorides is a reduction in cholinesterases, including acetylcholinesterase. Fluorides also directly affect the actions of many of other important neurotransmitters in the brain. Fluorides seem to have a special attraction to acetylcholine. Nerve cells that synthesize this transmitter have numerous projections to many forebrain areas, including the neocortex and deeper areas of the brain that provide information to the neocortex. Not only do fluorides change the amount of the acetylcholine in the brain, they selectively block certain receptors that respond to this transmitter. Fluoride reduces the number of one type of "nicotinic receptors" for acetylcholine. Some other nicotinic subtypes are not affected.[10] Added to all of the other alterations in structure and function of the brain caused by fluorides, the opportunity for mental and behavioral changes are almost limitless.

While the cholinergic system of the brain has been most studied in regard to the effects of fluoride, it is not the only neural transmitter affected. It is likely that all neural transmitter systems are affected by fluoride intake, directly or indirectly. Other anomalies related to fluoride intake are found in many other chemical systems of the brain.

During the period from 1956 to 1963, the endocrinologist, Ionel Rapaport, presented evidence of a link between fluoride exposure and the numbers of babies born with Down's syndrome, (Trisomy 21). For a number of years the only follow up to his work was in the form of epidemiological comparisons between the number of births of such children both to mothers living in fluoridated drinking water vs. the number of such born to mothers births in or non-fluoridated drinking water areas. The demographics of the two or more areas being compared were not fully taken into account in most of the studies. Maternal ages were also not taken into consideration. Overall, the "follow up" studies to Rapaport's report were not decisive but none of them failed to rule out his original findings.

Furthermore, a determination of fluoride effects using standard epidemiological procedures cannot provide convincing information. This is because it is impossible to find populations virtually the same in all regards except for the amount of fluoride in their drinking water. Another problem arises from the difficulty in accurately determining the number of Down's syndrome children born. Some investigators use the number of birth certificates on which the attending physician notes that the baby had Down's syndrome. Other investigators use only closed hospital records made sometime later. Still other investigators use both. Neither method is perfect.

The use of entries on hospital records would seem to be the most accurate method since physicians seldom enter the nature of possible deformities like Down's syndrome on birth certificates after delivery. Indeed because of the possibility of making a mistake from delivery, the diagnosis is not often made until a determination can be made by laboratory results.

Probably the best collection of relevant data comes from a study of births of children born in two areas of Atlanta, Georgia, as reported by Erickson et al. in 1976. Two different estimates of the number of Down's children and normal children were presented. One estimate of Down's syndrome births was made by the examination of copies of birth certificates and the other was based on hospital records. A re-examination of Erickson's

data by Burgstahler11 showed an overall enhancement of Down's syndrome births to mothers from the fluoridated area. Later, in 1998 Takahashi did a fine grain analysis of data from a number of sources that included the corrected numbers from the 1966 Erickson report.[12]

In the Takahashi report a clear-cut relationship between fluoride exposure and the number of affected children was found in mothers 30 years of age and younger. Recently, Juan C. Molino13 and I using only data from hospital records found the same age-fluoride-Down's syndrome birth effect.

In his report Takahashi extended the analysis of his data through the use of a regression analysis. He wanted to determine if there could be any dose that would not increase the likelihood of having a Down's syndrome child. According to his calculations there was no such dose. All doses of fluoride caused some enhancement of the likelihood of a woman having such a child. There are other data supporting the idea that fluorides can induce genetic alterations. Evidence indicating biochemical interactions of fluoride with the genetic mechanisms of cell division are presented in the NRC report on Fluoride in the Drinking Water. (See Endnote 2)

When the possible benefits and possible dangers of fluoride are considered there really is no comparison. Consider the following: There is no known benefit of adding any form of fluoride to our drinking water.

- Who would want to increase chances of having a less than perfect child? Who would wish to take a chance on a possible reduction of their own mental capacity?
- Who would want to have their personality altered by fluoride induced alterations in their brain chemistry?
- Who would want to increase their odds of developing Alzheimer's disease?

Eliminating the addition of fluoride to our drinking water would remove these possibilities. The cost of doing this is zero. In fact it would enrich the communities now adding fluorides to their drinking water.

Endnotes

1. Varner, J. A., Huie, C. W., Horvath, , W. J., Jensen, K. F., and Isaacson, R. L. (1993) Chronic AlF3 administration: II. Selected histological observations. Neurosci. Res. Comm. 13:99-104.Varner, J. A., Jensen, K. F., Horvath, W. J. and Isaacson R. L. (1998) Chronic administration of aluminum fluoride or sodium fluoride to rats in drinking water: alterations in neuronal and cerebrovascular integrity. Brain Res., 784: 284-298.Varner, J. A., Horvath, W. J., Huie, C. W., Naslund, H. R., and Isaacson, R. L. (1994) Chronic aluminum fluoride administration. Behav. Neural Biol., 61: 233-241.Isaacson, R. L., Varner, J. A., and Jensen, K. F. Toxin-induced blood vessel inclusions caused by the chronic administrations of aluminum and sodium fluoride. Ann. NY Acad. Sci.,825:152.166.
2. The final report of the committee was published by the National Academies Press in December 2006, entitled "Fluoride in drinking water." It can by obtained from the National Academies Press and by special order from any bookstore.
3. Spira, L. The drama of fluorine, archenemy, of mankind. Milwaukee: The Lee Foundation for Nutritional Research, 1950, 1959.
4. Waldbott, G. L. Fluoridation the great dilemma, Lawrence, KA: Coronado Press, 1978.
5. Masters R. D., Coplan, M.J. Association of silicofluoride treated water lead with elevated blood lead. Neurotoxicology, 2000. 21:1091-1100. Masters, R. D., Coplan M. J. A dynamic, multifactoral model of alcohol, drug abuse and crime: Linking neuroscience and behavior to toxicology. Soc. Sci. Information, 1999, 38: 591-624.
6. Seavy, J., (2005) Water fluoridation and crime in America. Fluoride, 38:11-22.
7. Du Li. (1992) The effect of fluorine on developing human brain. Chinese Journal of Pathology, 21:218-20. Li Jing, Yao L., Shao, Q-L, and Wu, C-Y. (2004) Effects of high fluoride

level on neonatal neurobehavioral development. Chinese Journal of Endocrinology, 23: No.5.
8. Belovjovic, G., Jakovlevic, B. (1999) Aluminum and Alzheimer's disease. Spr. ArArh. Celok 126: 283-289.
9. Strunecka, A.(1999)Aluminum plus Fluoride: a new deadly duo. Dement. 1:2-3.
10. Long, Y-G, Wang, Y-N, Chen, J., Jiang, S-F, Nordberg, A., and Guan, Z-Z. (2002) Chronic fluoride toxicity decreases the number of acetylcholine receptors in the rat brain. Neurotox. Terat, 23: 751-757.
11. Burgstahler, A. W. (1966) Fluoridated water and Down's syndrome. Long abstract of a report of the 21st Conference of the International Society for Brain Research, Budapest.
12. Takahashi, K. (1998) Fluoride-linked Down syndrome births and their estimated occurrence due to water fluoridation. Fluoride, 31: 61-73.
13. Juan Carlos Molina is the Director of the Ferryra Research Institute at the University of Cordoba, Argentina, as well as holding his distinguished professor position there. He also is a visiting research professor at Binghamton University.

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Appendix 4:

UK EXPERT GROUP ON VITAMINS AND MINERALS

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The Expert Group on Vitamins and Minerals (EVM) is an independent expert advisory committee which was asked to advise on safe levels of intakes of vitamins and minerals in food supplements and fortified foods. In Great Britain, products that are not regulated as medicines may be regarded as foods and, as such, are subject to the general provisions of the Food Safety Act 1990, the Food Labelling Regulations, 1996 (as amended) and the Trade Descriptions Act 1968. Northern Ireland has similar but separate legislation. Food products containing added vitamins and minerals can be divided into two categories: fortified foods, such as margarine and breakfast cereals, and food supplements. EVM Members were drawn from the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT), the Committee on Safety of Medicines (CSM), the Committee on the Medical Aspects of Food and Nutrition Policy (COMA) and the Food Advisory Committee (FAC). The membership represented a range of expertise in toxicology, pharmacology, epidemiology, medicine and nutrition as well as a non-specialist Member to represent consumer interests.

Including:

- Prof Michael J Langman (CHAIRMAN) Professor of Medicine, University of Birmingham
- Prof Peter Aggett Head Postgraduate School of Medicine and Health, University of Central Lancashire
- Prof Donald S Davies Head of Clinical Pharmacology, Director of DH Toxicology Unit, Faculty of Medicine, Imperial College, London
- Prof Alan A Jackson Professor of Human Nutrition, University of Southampton
- Prof Brian J Kirby Professor of Medicine University of Exeter Consultant Physician Royal Devon and Exeter Hospitals
- Prof André McLean Professor of Toxicology, University College, London
- Prof Andrew G Renwick Professor of Biochemical Pharmacology, University of Southampton
- Dr Lesley Rushton Head of Epidemiology, Institute forEnvironmental Health, University of Leicester
- Ms Clare E ShawChief Dietitian, Royal Marsden NHS Trust
- Dr Anita Thomas Consultant Physician and Associate Medical Director (Education), Plymouth Hospital NHS Trust, Clinical Subdean Peninsula Medical School
- Dr Anthony F Williams, Senior Lecturer and Consultant in Neonatal Paediatrics, St George's Hospital, London

In their final report[1218] on Vitamins and Minerals the EVM concluded that it was inappropriate to comment on fluoride with regard to food fortification since this is carried out as a public health measure.

[1218] Safe Upper Levels for Vitamins and Minerals, May 2003, Expert Group on Vitamins and Minerals, Final Report.

Appendix 5:

U.S. EPA Memorandum 1990, U.S. EPA Headquarters Union Statement 1999, U.S. EPA Unions Statement Of Concern On Fluoridation 2003

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON. D.C. 20460

OFFICE OF WATER

MEMORANDUM

DATE: May 1, 1990

SUBJECT: Fluoride Conference to Review the NTP Draft Fluoride Report

FROM: Wm L. Marcus, Ph.D., Senior Science Advisor, Criteria & Standards Division, ODW (WH-550D)

TO: Alan B. Hais, Acting Director, Criteria & Standards Division, ODW (WH-550D)

The conference was held in RTP at the NIEHS headquarters on April 26, 1990. The subject of the conference was a peer review of the NTP draft report on the toxicology and carcinogenesis studies of Sodium Fluoride in F344/N Rats and B6C3F Mice (Drinking Water Studies) NTP Report Number 393. Dr. Robert Scala was to chair this meeting but was unable to attend because of ill health. Dr. Michael Gallo appointed acting Chairperson. One of the attenders seated with the panel members was David Rall, Ph.D., M.D., Director of NIEHS. Dr. Rall took an extremely active interest in the proceedings and remained seated for the entire proceedings with only two minor interruptions.

The most disturbing part of the report was the continual reference to the historical controls as having the same or higher cancers as the test groups. On pages 89 - 90 of the report starting with the last paragraph the authors State the following:

An important consideration which limits the usefulness of the historical control data base in the current studies is that the diet used in all other NTP studies had not been closely monitored for fluoride content. Fluoride concentrations in typical batches of NHI-07 diet range between 28 and 47 ppm (.7 and 1.2 mg/kg/day)(Rao and Knapka1, 1987). ***Assuming*** *a minimum bioavailability of 60% (Tests show 66% absorption page I-18), the historical database animals actually constitute a group receiving sufficient fluoride to place them* ***between*** *the low- and mid-concentration group in the current (the studies reviewed at RTP at the conference). The fact that this fluoride is available for absorption from the standard diet is supported by the levels of fluoride found in the* ***bones*** *of animals maintained on this diet in the six months studies (Appendix I). (The levels in the bones of the rats on the standard NHI chow was ten [10] times the levels of those fed the semisynthetic diet and deionized water, 0.922 vs 0.0901). If the fluoride [is] in fact influencing the "spontaneous " or background incidence of osteosarcoma in male rats, comparisons with those in the historical database maybe misleading. This forces an even greater reliance on the within-study comparisons, ie., the incidences of the dosed groups compared with the concurrent control, in the interpretation of the results of the sodium fluoride studies.* [italics & emphasis in memo]

When I plotted a bar graph of osteosarcoma in male rats and placed the historical controls on the graph 0.6% is just where expected. This helps demonstrate a relationship between osteosarcoma and fluoride. The purpose of such graphs is to predict occurrence. Since the historical controls comprise some 6,000 animals, this data point is extremely significant compared to the other three. Osteosarcoma is an extremely rare animal tumor and may be the result of the variable high fluoride content in the feed. In order to demonstrate this, all that need be done is require that the fluoride content of animal chow be lowered dramatically and that fluoride be removed from the water given to the animals under study.

The dose of fluoride to which the concurrent controls were exposed is 0.2 mg/kg/day. A 70 kg man who drinks 2 liters daily is exposed to 0.03 mg/kg/day. The "control" animals were exposed to an amount of fluoride six to seven (6-7 X) greater. Lois Gold, Ph.D. of the review panel concluded that, "this group of animals therefore, can hardly be termed a control group. It can best be described as a lowest dosed group." This is an important consideration because as the document reports on page 9, the levels of fluoride in bone are linearly dependent upon dose and length of exposure ("depend upon total intake") in people. The level of fluoride in ashed samples of bone of 20-30 year old people is 200 - 800 mg/kg compared to 70 to 80 year old people of 1,000 - 2.500 mg/kg. In the document, the authors cited Zipkin2 who reported on bone fluoride concentrations in four groups of individuals with average ages of 56 to 76 who lived in areas with fluoride concentrations in drinking water of 0. 1, 1, 2.6, or 4 ppm The relationship to bone fluoride concentrations and water fluoride content was linear; bone fluoride ranged from about 800 to 7,000 ppm ash with increasing water fluoride."

In the animal studies the levels of fluoride (Appendix I) found in the bones of the animals were the same as or lower than those found in people. The highest dosed level of rats had lower levels of fluoride in their bones (5,470 ppm) compared to people (7,000 ppm) at the MCL of 4 ppm. This can be interpreted as people who ingest drinking water at the MCL have 1.3 times more fluoride in their bones than male rats who get osteosarcoma. This is the first time in my memory that animals have lower concentrations of the carcinogen at the sight of adverse effect than do humans. An important toxicological consideration is that a toxic substance stores at the same place it exerts it toxic activity. This is true of benzene and now for fluoride. Fluoride however, is at twice the concentration in human bones compared to benzene which is 10 to 100 [times] greater in animal marrow. This portends a very serious problem. One would expect to be able to discern a carcinogenic effect in the exposed population when compared to the unexposed population especially if data exist on the populations before fluoridation.

Yiamouyiannis and Burk published epidemiology studies that have since been revised twice3, by Burk (former head of the Cytochemistry section at NIH). In these extensively peer-reviewed papers; the authors found that about 10,000 deaths a year are attributable to fluoride water treatment. The U.S. Public Health Service (U.S.PHS) criticized the original studies by erroneously asserting that the results reported by the authors were a result of changes in the age, race and sex composition of the sample. The U.S.PHS made mathematical errors and did not

include 90% of the data. U.S.PHS method of analysis when applied to the database, confirmed that 10,000 excess cancer deaths yearly were linked to fluoridation of water supplies. This evidence has been tested most recently in the Pennsylvania Courts and round scientifically sound after careful scrutiny.

There were three different short term in vitro tests performed on fluoride and all these tests proved fluoride to be mutagenic. An Ames test was performed and reported to be negative. Bruce Ames, in a letter to Arthur Upton introduced in the Congressional Record, stated that his test system was inappropriate for fluoride testing based on a number of technical considerations. EPA's own guidelines require that in vitro tests be taken into consideration when found positive. **In this case, the mutagenicity of fluoride supports the conclusion that fluoride is a probable human carcinogen**. [Emphasis added]

Melvin Reuber, M.D, a board certified pathologist and former consultant to EPA and part time EPA employee, reviewed some of pathology slides and the Battelle report. Dr. Reuber has had his pathologic diagnoses questioned several times in the past. When an independent board together with Dr. Reuber went over the Slides his opinion was always upheld. He first published the work that identified hepatocholangiocarcinoma as a pathologic entity. The report changed Battelle's board certified veterinary pathologists diagnoses from hepatocholangiocarcinoma to hepatoblastoma and finally to hepatocarcinoma. Dr. Reuber reviewed the pathology slides and stated that these lesions are indeed hepatocholangiocarcinoma. Because Dr. Reuber first identified and published his findings on this tumor, I trust his opinion in this matter. These tumors are extremely rare. Dr. Reuber's diagnoses would make the liver cancers significant because of their rarity. **This changes the equivocal finding of the board to at least some evidence or clear evidence of carcinogenicity**.

In addition, the oral changes in the report were down-graded from dysplasia and metaplasia to degeneration. Dr. Reuber said that this. change should also be reviewed. The report also down-graded adrenal pheochromocytomas and tumors to hyperplasia. This needs to be reviewed by an independent board. The other liver carcinomas were down-graded to foci by artificially defining a need for 75% compression in the tumor before it was no longer a foci. Using this changed definition carcinomas were down-graded to adenomas and adenomas downgraded to eosinophilic foci. In almost all instances, the Battelle board certified pathologists' findings were down-graded.

It is my suggestion that a board independent of NIEHS should be assembled by ODW consisting of human pathologists (for their experience in diagnosing osteosarcoma), the Battelle pathologist (to defend his original diagnoses), Dr. Melvin Reuber, Dr. Thomas Squires and two other well known independent board-certified animal pathologists. The charge to this board is to meet as a body, review the slides, agree on a pathologic diagnoses and prepare a report to be submitted to ODW for incorporation in our docket for the fluoride regulation. [emphasis added]

The report talks about the efficacy of fluoride and tooth decay. Since the studies were performed to determine the carcinogenicity of fluoride this should not have

been addressed. There appear to be at least four different publications from the U.S., Canada, and New Zealand that have reported similar or lower tooth decay rates in nonfluoridated areas as compared to fluoridated areas4,5,6,7 Therefore, the entire question of the efficacy of fluoridation based on extensive and multiple studies has been called into question. Our job is to set safe levels for fluoride in drinking water based on the scientific evidence.

The problem with this meeting was the inability of independent reviewers to get to see the slides prior to the meeting. We must perform our own scientific review of the slides and write our conclusions for use in the development of the revised fluoride regulation.

(1) Roa, G.N., and Knappa, J.J. 1987. Contaminant and nutrient concentrations of natural ingredient rat and mouse diet used in chemical toxicology studies. Fundam. Appl. Toxicol. 9, 329-338.

(2) Zipkin, L., McClure, F.J., Leone, H.C., and Lee, W.A. 1958. Fluoride deposition in human bones after prolonged ingestion of fluoride in drinking water. Public Health Rep. 73, 732-740.

(3) Graham, J.R., Burk, O., and Morin, P. 1987. A current restatement and continuing reappraisal concerning demographic variables in American time-trend studies an water fluoridation and human cancer. Proc Pennsylvania Academy of Sci. 61:138-146.

4) Colquhoun, J. 1987. Comm. Health Studies. 11:85.

(5) Gray, a. 1987. J. Canadian Dental Assoc. 53:763.

(6) Hildebolt, C.F. et al. 1989. Amer J, Physiol. Anthropol. 78:79-92. (7) Diesendorf, M. 1986. Nature. 321:125.

May 1, 1999
WHY EPA'S HEADQUARTERS UNION OF SCIENTISTS OPPOSES FLUORIDATION

The following documents why our union, formerly National Federation of Federal Employees Local 2050 and since April 1998 Chapter 280 of the National Treasury Employees Union, took the stand it did opposing fluoridation of drinking water supplies. Our union is comprised of and represents the approximately 1500 scientists, lawyers, engineers and other professional employees at EPA Headquarters here in Washington, D.C.

The union first became interested in this issue rather by accident. Like most Americans, including many physicians and dentists, most of our members had thought that fluoride's only effects were beneficial - reductions in tooth decay, etc. We too believed assurances of safety and effectiveness of water fluoridation.

Then, as EPA was engaged in revising its drinking water standard for fluoride in 1985, an employee came to the union with a complaint: he said he was being forced to write into the regulation a Statement to the effect that EPA thought it was alright for children to have "funky" teeth. It was OK, EPA said, because it considered that condition to be only a *cosmetic* effect, not an adverse *health* effect. The reason for this EPA position was that it was under political pressure to set its health-based standard for fluoride at 4 mg/liter. At that level, EPA knew that a significant number of children develop moderate to severe dental fluorosis, but since it had deemed the effect as only cosmetic, EPA didn't have to set its health-based standard at a lower level to prevent it.

We tried to settle this ethics issue quietly, within the family, but EPA was unable or unwilling to resist external political pressure, and we took the fight public with a union *amicus curiae* brief in a lawsuit filed against EPA by a public interest group. The union has published on this initial involvement period in detail.1

Since then our opposition to drinking water fluoridation has grown, based on the scientific literature documenting the increasingly out-of-control exposures to fluoride, the lack of benefit to dental health from ingestion of fluoride and the hazards to human health from such ingestion. These hazards include acute toxic hazard, such as to people with impaired kidney function, as well as chronic toxic hazards of gene mutations, cancer, reproductive effects, neurotoxicity, bone pathology and dental fluorosis. First, a review of recent neurotoxicity research results.

In 1995, Mullenix and co-workers2 showed that rats given fluoride in drinking water at levels that give rise to plasma fluoride concentrations in the range seen in humans suffer neurotoxic effects that vary according to when the rats were given the fluoride - as adult animals, as young animals, or through the placenta before birth. Those exposed before birth were born hyperactive and remained so throughout their lives. Those exposed as young or adult animals displayed depressed activity. Then in 1998, Guan and co-workers3 gave doses similar to those used by the Mullenix research group to try to understand the mechanism(s) underlying the effects seen by the Mullenix group. Guan's group found that several key chemicals in the brain - those that form the membrane of brain cells - were substantially depleted in rats given fluoride, as compared to those who did not get fluoride.

Another 1998 publication by Varner, Jensen and others4 reported on the brain- and

kidney damaging effects in rats that were given fluoride in drinking water at the same level deemed "optimal" by pro-fluoridation groups, namely 1 part per million (1 ppm). Even more pronounced damage was seen in animals that got the fluoride in conjunction with aluminum. These results are especially disturbing because of the low dose level of fluoride that shows the toxic effect in rats - rats are more resistant to fluoride than humans. This latter Statement is based on Mullenix's finding that it takes substantially more fluoride in the drinking water of rats than of humans to reach the same fluoride level in plasma. It is the level in plasma that determines how much fluoride is "seen" by particular tissues in the body. So when rats get 1 ppm in drinking water, their brains and kidneys are exposed to much less fluoride than humans getting 1 ppm, yet they are experiencing toxic effects. Thus we are compelled to consider the likelihood that humans are experiencing damage to their brains and kidneys at the "optimal" level of 1 ppm.

In support of this concern are results from two epidemiology studies from China5, 6 that show decreases in I.Q. in children who get more fluoride than the control groups of children in each study. These decreases are about 5 to 10 I.Q. points in children aged 8 to 13 years.

Another troubling brain effect has recently surfaced: fluoride's interference with the function of the brain's pineal gland. The pineal gland produces melatonin which, among other roles, mediates the body's internal clock, doing such things as governing the onset of puberty. Jennifer Luke7 has shown that fluoride accumulates in the pineal gland and inhibits its production of melatonin. She showed in test animals that this inhibition causes an earlier onset of sexual maturity, an effect reported in humans as well in 1956, as part of the Kingston/Newburgh study, which is discussed below. In fluoridated Newburgh, young girls experienced earlier onset of menstruation (on average, by six months) than girls in non-fluoridated Kingston.8

From a risk assessment perspective, all these brain effect data are particularly compelling and disturbing because they are convergent.

We looked at the cancer data with alarm as well. There are epidemiology studies that are convergent with whole-animal and single-cell studies (dealing with the cancer hazard), just as the neurotoxicity research just mentioned all points in the same direction. EPA fired the Office of Drinking Water's chief toxicologist, Dr. William Marcus, who also was our local union's treasurer at the time, for refusing to remain silent on the cancer risk issue.9 The judge who heard the lawsuit he brought against EPA over the firing made that finding - that EPA fired him over his fluoride work and not for the phony reason put forward by EPA management at his dismissal. Dr. Marcus won his lawsuit and is again at work at EPA. Documentation is available on request.

The type of cancer of particular concern with fluoride, although not the only type, is osteosarcoma, especially in males. The National Toxicology Program conducted a two-year study10 in which rats and mice were given sodium fluoride in drinking water. The positive result of that study (in which malignancies in tissues other than bone were also observed), particularly in male rats, is convergent with a host of data from tests showing fluoride's ability to cause mutations (a principal "trigger" mechanism for inducing a cell to become cancerous) e.g.11a, b, c, d and data showing increases in osteosarcomas in young men in New Jersey12 , Washington and Iowa13 based on their drinking fluoridated water. It was his analysis, repeated Statements about all these and other incriminating cancer data, and his requests for an independent, unbiased evaluation of them that got Dr. Marcus fired. Bone pathology other than cancer is a concern as well. An excellent review of this issue was published by Diesendorf et al. in 1997.14 Five epidemiology studies have shown a higher rate of hip fractures in fluoridated vs. non-fluoridated communities.15a, b, c, d, e. Crippling skeletal fluorosis was the endpoint used by EPA to

set its primary drinking water standard in 1986, and the ethical deficiencies in that standard setting process prompted our union to join the Natural Resources Defense Council in opposing the standard in court, as mentioned above.

Regarding the effectiveness of fluoride in reducing dental cavities, there has not been any double-blind study of fluoride's effectiveness as a caries preventative. There have been many, many small scale, selective publications on this issue that proponents cite to justify fluoridation, but the largest and most comprehensive study, one done by dentists trained by the National Institute of Dental Research, on over 39,000 school children aged 5-17 years, shows no significant differences (in terms of decayed, missing and filled teeth) among caries incidences in fluoridated, non-fluoridated and partially fluoridated communities.16 The latest publication17 on the fifty-year fluoridation experiment in two New York cities, Newburgh and Kingston, shows the same thing. The only significant difference in dental health between the two communities as a whole is that fluoridated Newburgh, N.Y. shows about twice the incidence of dental fluorosis (the first, visible sign of fluoride chronic toxicity) as seen in non-fluoridated Kingston.

John Colquhoun's publication on this point of efficacy is especially important.18 Dr. Colquhoun was Principal Dental Officer for Auckland, the largest city in New Zealand, and a staunch supporter of fluoridation - until he was given the task of looking at the world-wide data on fluoridation's effectiveness in preventing cavities. The paper is titled, "Why I changed My Mind About Water Fluoridation." In it Colquhoun provides details on how data were manipulated to support fluoridation in English speaking countries, especially the U.S. and New Zealand. This paper explains why an ethical public health professional was compelled to do a 180 degree turn on fluoridation.

Further on the point of the tide turning against drinking water fluoridation, Statements are now coming from other dentists in the pro-fluoride camp who are starting to warn that topical fluoride (e.g. fluoride in tooth paste) is the only significantly beneficial way in which that substance affects dental health.19, 20, 21 However, if the concentrations of fluoride in the oral cavity are sufficient to inhibit bacterial enzymes and cause other bacteriostatic effects, then those concentrations are also capable of producing adverse effects in mammalian tissue, which likewise relies on enzyme systems. This Statement is based not only on common sense, but also on results of mutation studies which show that fluoride can cause gene mutations in mammalian and lower order tissues at fluoride concentrations estimated to be present in the mouth from fluoridated tooth paste.22 Further, there were tumors of the oral cavity seen in the NTP cancer study mentioned above, further strengthening concern over the toxicity of topically applied fluoride.

In any event, a person can choose whether to use fluoridated tooth paste or not (although finding non-fluoridated kinds is getting harder and harder), but one cannot avoid fluoride when it is put into the public water supplies.

So, in addition to our concern over the toxicity of fluoride, we note the uncontrolled - and apparently uncontrollable - exposures to fluoride that are occurring nationwide via drinking water, processed foods, fluoride pesticide residues and dental care products. A recent report in the lay media23 that, according to the Centers for Disease Control, at least 22 % of America's children now have dental fluorosis, is just one indication of this uncontrolled, excess exposure. The finding of nearly 12 % incidence of dental fluorosis among children in un-fluoridated Kingston New York17 is another. For governmental and other organizations to continue to push for *more* exposure in the face of current levels of over-exposure coupled with an increasing crescendo of adverse toxicity findings is irrational and irresponsible at best.

Thus, we took the stand that a policy which makes the public water supply a vehicle for

disseminating this toxic and prophylactically useless (via ingestion, at any rate) substance is wrong.

We have also taken a direct step to protect the employees we represent from the risks of drinking fluoridated water. We applied EPA's risk control methodology, the Reference Dose, to the recent neurotoxicity data. The Reference Dose is the daily dose, expressed in milligrams of chemical per kilogram of body weight, that a person can receive over the long term with reasonable assurance of safety from adverse effects. Application of this methodology to the Varner et al.4 data leads to a Reference Dose for fluoride of 0.000007 mg/kg-day. Persons who drink about one quart of fluoridated water from the public drinking water supply of the District of Columbia while at work receive about 0.01mg/kg-day from that source alone. This amount of fluoride is more than 100 times the Reference Dose. On the basis of these results the union filed a grievance, asking that EPA provide un-fluoridated drinking water to its employees.

The implication for the general public of these calculations is clear. Recent, peer-reviewed toxicity data, when applied to EPA's standard method for controlling risks from toxic chemicals, require an immediate halt to the use of the nation's drinking water reservoirs as disposal sites for the toxic waste of the phosphate fertilizer industry.24 This document was prepared on behalf of the National Treasury Employees Union Chapter 280 by Chapter Senior Vice-President J. William Hirzy, Ph.D.

END NOTE LITERATURE CITATIONS

1. Applying the NAEP code of ethics to the Environmental Protection Agency and the fluoride in drinking water standard. Carton, R.J. and Hirzy, J.W. Proceedings of the 23rd Ann. Conf. of the National Association of Environmental Professionals. 20-24 June, 1998. GEN 51-61.
2. Neurotoxicity of sodium fluoride in rats. Mullenix, P.J., Denbesten, P.K., Schunior, A. and Kernan, W.J. Neurotoxicol. Teratol. 17 169-177 (1995)
3. Influence of chronic fluorosis on membrane lipids in rat brain. Z.Z. Guan, Y.N. Wang, K.Q. Xiao, D.Y. Dai, Y.H. Chen, J.L. Liu, P. Sindelar and G. Dallner, Neurotoxicology and Teratology 20 537-542 (1998).
4. Chronic administration of aluminum- fluoride or sodium-fluoride to rats in drinking water: alterations in neuronal and cerebrovascular integrity. Varner, J.A., Jensen, K.F., Horvath, W. And Isaacson, R.L. Brain Research 784 284-298 (1998).
5. Effect of high fluoride water supply on children's intelligence. Zhao, L.B., Liang, G.H., Zhang, D.N., and Wu, X.R. Fluoride 29 190-192 (1996)
6. Effect of fluoride exposure on intelligence in children. Li, X.S., Zhi, J.L., and Gao, R.O. Fluoride 28 (1995).
7. Effect of fluoride on the physiology of the pineal gland. Luke, J.A. Caries Research 28 204 (1994).
8. Newburgh-Kingston caries-fluorine study XIII. Pediatric findings after ten years. Schlesinger, E.R., Overton, D.E., Chase, H.C., and Cantwell, K.T. JADA 52 296-306 (1956).
9. Memorandum dated May 1, 1990. Subject: Fluoride Conference to Review the NTP Draft Fluoride Report; From: Wm. L. Marcus, Senior Science Advisor ODW; To: Alan B. Hais, Acting Director Criteria & Standards Division ODW.
10. Toxicology and carcinogenesis studies of sodium fluoride in F344/N rats and B6C3F1 mice. NTP Report No. 393 (1991).
11a. Chromosome aberrations, sister chromatid exchanges, unscheduled DNA synthesis and morphological neoplastic transformation in Syrian hamster embryo cells. Tsutsui et al. Cancer Research 44 938-941 (1984).
11b. Cytotoxicity, chromosome aberrations and unscheduled DNA synthesis in cultured human diploid fibroblasts. Tsutsui et al. Mutation Research 139 193-198 (1984).

11c. Positive mouse lymphoma assay with and without S-9 activation; positive sister chromatid exchange in Chinese hamster ovary cells with and without S-9 activation; positive chromosome aberration without S-9 activation. Toxicology and carcinogenesis studies of sodium fluoride in F344/N rats and B6C3F1 mice. NTP Report No. 393 (1991).
11d. An increase in the number of Down's syndrome babies born to younger mothers in cities following fluoridation. Science and Public Policy 12 36-46 (1985).
12. A brief report on the association of drinking water fluoridation and the incidence of osteosarcoma among young males. Cohn, P.D. New Jersey Department of Health (1992).
13. Surveillance, epidemiology and end results (SEER) program. National Cancer Institute in Review of fluoride benefits and risks. Department of Health and Human Services. F1-F7 (1991).
14. New evidence on fluoridation. Diesendorf, M., Colquhoun, J., Spittle, B.J., Everingham, D.N., and Clutterbuck, F.W. Australian and New Zealand J. Public Health. 21 187-190 (1997).
15a. Regional variation in the incidence of hip fracture: U.S. white women aged 65 years and older. Jacobsen, S.J., Goldberg, J., Miles, ,T.P. et al. JAMA 264 500-502 (1990)
15b. Hip fracture and fluoridation in Utah's elderly population. Danielson, C., Lyon, J.L., Egger, M., and Goodenough, G.K. JAMA 268 746-748 (1992).
15c. The association between water fluoridation and hip fracture among white women and men aged 65 years and older: a national ecological study. Jacobsen, S.J., Goldberg, J., Cooper, C. and Lockwood, S.A. Ann. Epidemiol.2 617-626 (1992).
15d. Fluorine concentration is drinking water and fractures in the elderly [letter]. Jacqmin-Gadda, H., Commenges, D. and Dartigues, J.F. JAMA 273 775-776 (1995).
15e. Water fluoridation and hip fracture [letter]. Cooper, C., Wickham, C.A.C., Barker, D.J.R. and Jacobson, S.J. JAMA 266 513-514 (1991).
16. Water fluoridation and tooth decay: Results from the 1986-1987 national survey of U.S. school children. Yiamouyiannis, J. Fluoride 23 55-67 (1990).
17. Recommendations for fluoride use in children. Kumar, J.V. and Green, E.L. New York State Dent. J. (1998) 40-47.
18. Why I changed my mind about water fluoridation. Colquhoun, J. Perspectives in Biol. And Medicine 41 29-44 (1997).
19. A re-examination of the pre-eruptive and post-eruptive mechanism of the anti-caries effects of fluoride: is there any anti-caries benefit from swallowing fluoride? Limeback, H. Community Dent. Oral Epidemiol. 27 62-71 (1999).
20. Fluoride supplements for young children: an analysis of the literature focussing on benefits and risks. Riordan, P.J. Community Dent. Oral Epidemiol. 27 72-83 (1999).
21. Prevention and reversal of dental caries: role of low level fluoride. Featherstone, J.D. Community Dent. Oral Epidemiol. 27 31-40 (1999).
22. Appendix H. Review of fluoride benefits and risks. Department of Health and Human Services. H1-H6 (1991).
23. Some young children get too much fluoride. Parker-Pope, T. Wall Street Journal Dec. 21, 1998.
24. Letter from Rebecca Hanmer, Deputy Assistant Administrator for Water, to Leslie Russell re: EPA view on use of by-product fluosilicic (sic) acid as low cost source of fluoride to water authorities. March 30, 1983.

OTHER CITATIONS (This short list does not include the entire literature on fluoride effects)

a. Exposure to high fluoride concentrations in drinking water is associated with decreased birth rates. Freni, S.C. J. Toxicol. Environ. Health 42 109-121 (1994)

b. Ameliorative effects of reduced food-borne fluoride on reproduction in silver foxes. Eckerlin, R.H., Maylin, G.A., Krook, L., and Carmichael, D.T. Cornell Vet. 78 75-91 (1988).

c. Milk production of cows fed fluoride contaminated commercial feed. Eckerlin, R.H., Maylin, G.A., and Krook, L. Cornell Vet. 76 403-404 (1986).

d. Maternal-fetal transfer of fluoride in pregnant women. Calders, R., Chavine, J., Fermanian, J., Tortrat, D., and Laurent, A.M. Biol. Neonate 54 263-269 (1988).

e. Effects of fluoride on screech owl reproduction: teratological evaluation, growth, and blood chemistry in hatchlings. Hoffman, D.J., Pattee, O.H., and Wiemeyer, S.N. Toxicol. Lett. 26 19-24 (1985).

f. Fluoride intoxication in dairy calves. Maylin, G.A., Eckerlin, R.H., and Krook, L. Cornell Vet. 77 84-98 (1987).

g. Fluoride inhibition of protein synthesis. Holland, R.I. Cell Biol. Int. Rep. 3 701-705 (1979).

h. An unexpectedly strong hydrogen bond: ab initio calculations and spectroscopic studies of amide-fluoride systems. Emsley, J., Jones, D.J., Miller, J.M., Overill, R.E. and Waddilove, R.A. J. Am. Chem. Soc. 103 24-28 (1981).

i. The effect of sodium fluoride on the growth and differentiation of human fetal osteoblasts. Song, X.D., Zhang, W.Z., Li, L.Y., Pang, Z.L., and Tan, Y.B. Fluoride 21 149-158 (1988).

j. Modulation of phosphoinositide hydrolysis by NaF and aluminum in rat cortical slices. Jope, R.S. J. Neurochem. 51 1731-1736 (1988).

k. The crystal structure of fluoride-inhibited cytochrome c peroxidase. Edwards, S.L., Poulos, T.L., Kraut, J. J. Biol. Chem. 259 12984-12988 (1984).

l. Intracellular fluoride alters the kinetic properties of calcium currents facilitating the investigation of synaptic events in hippocampal neurons. Kay, A.R., Miles, R., and Wong, R.K.S. J. Neurosci. 6 2915-2920 (1986).

m. Fluoride intoxication: a clinical-hygienic study with a review of the literature and some experimental investigations. Roholm, K. H.K. Lewis Ltd (London) (1937).

n. Toxin-induced blood vessel inclusions caused by the chronic administration of aluminum and sodium fluoride and their implications for dementia. Isaacson, R.L., Varner, J.A., and Jensen, K. F. Ann. N.Y. Acad. Sci. 825 152-166 (1997).

o. Allergy and hypersensitivity to fluoride. Spittle, B. Fluoride 26 267-273 (1993)

Coalition of U.S. Environmental Protection Agency Unions

A STATEMENT OF CONCERN ON FLUORIDATION 2003

Understanding and appreciating the historical reasons for advocating fluoridation, the undersigned professionals now recognize valid concerns about its safety and about its impact on the environment. This Statement serves as a vehicle for expressing these concerns. However, it is not a position Statement on fluoridation, nor does it commit the undersigned to any point of view other than what is stated clearly in this document. A brief summary of recent events, reports, and research underlying our concerns, as well as a list of references, are supplementary to this document.

OUR MAJOR CONCERNS:

I. Environmental Concerns

Silicofluorides: unrefined industrial waste

91% of Americans ingesting artificially fluoridated water are consuming silicofluorides[1]. This is a class of fluoridation chemicals that includes hydrofluosilicic acid and its salt form, sodium fluorosilicicte. These chemicals are collected from the pollution scrubbers of the phosphate fertilizer industry. The scrubber liquors contain contaminants such as arsenic, lead, cadmium, mercury, and radioactive particles[2], are legally regulated as toxic waste, and are prohibited from direct dispersal into the environment. Upon being sold (unrefined) to municipalities as fluoridating agents, these same substances are then considered a "product", allowing them to be dispensed through fluoridated municipal water systems to the very same ecosystems to which they could not be released directly. Sodium fluoride, used in the remaining municipalities, is also an industrial waste product that contains hazardous contaminants.

Scarcity of environmental impact studies

This is of deep concern to us. Studies that do exist indicate damage to salmon and to plant ecosystems.[3a] It is significant that Canada's water quality guideline to protect freshwater life is 0.12 ppm (parts per million). [3b]

99.97% of fluoridated water is released directly into the environment at around 1ppm

This water is NOT used for drinking or cooking.[4]

II. Health Concerns

Absence of safety studies on silicofluorides

When asked by the U.S. House Committee on Science for chronic toxicity test data on sodium fluorosilicicte and hydrofluorosilicic acid, Charles Fox of the EPA answered on June 23, 1999, "EPA was not able to identify chronic toxicity data on these chemicals". [5] Further, EPA's National Risk Management Research Laboratory stated, on April 25, 2002, that the chemistry of silicofluorides is "not well understood" and studies are needed.

EPA health goals ignored

The EPA defines the Maximum Contaminant Level Goal (MCLG) for toxic elements in drinking water thus: "the level below which there are no known or anticipated effects to health." The MCLG for arsenic, lead, and radioactive particles, all contaminants of the scrubber liquors used for fluoridation, is 0.0 ppb (zero parts per billion). Therefore, any addition of fluorine-bearing substances to drinking water that include these contaminants is contrary to the intent of EPA's established health goals.

Increased blood lead levels in children

Two recent studies with a combined sampling of over 400,000 children found significantly increased levels of lead in children's blood when silicofluorides from the phosphate fertilizer industry were used as the fluoridating agent.[6] This shows that there is a significant difference in health effects even between different fluoridation compounds.

Ingestion of fluoride linked to many health effects

Contrary to assertions that the health effects of fluoride ingestion already have been scientifically proven to be safe and that there is no credible scientific concern, over the last fifteen years the ingestion of fluoride has been linked in scientific peer-reviewed literature to neurotoxicity[7], bone pathology[8], reproductive effects[9], interference with the pineal gland [10], gene mutations[11], thyroid pathology[12], and the increasing incidence and severity of dental fluorosis[13]. This has caused professionals who once championed the uses of fluoride in preventing tooth decay, to reverse their position and call for a halt in further exposures.[14] It is of significance that 14 Nobel Prize winning scientists, including the 2000 Nobel Laureate in Medicine, Arvid Carlsson, have expressed reservations on, or outright opposition to, fluoridation.[15]

FDA has never approved systemic use of fluoride

The U.S. Food and Drug Administration in December 2000 stated to the U.S. House Committee on Science they have never provided any specific approval for safety or effectiveness for any fluoride substance intended to be ingested for the purpose of reducing tooth decay.[16]

Total fluoride exposure of growing concern

Total fluoride exposure from all sources, including food, water, and air, is of growing concern within the scientific community.[17] As evidenced in the U.S. Public Health Service ATSDR 1993 report which was referenced in correspondence between the U.S. House Committee on Science and Charles Fox of the U.S. EPA, large subsets of the population, including the elderly, children, and pregnant women, may be unusually susceptible to the toxic effects of fluoride.[18]

Centers for Disease Control concession

The CDC now concedes that the systemic value of ingesting fluoride is minimal, as fluoride's oral health benefits are predominantly topical[19], and that there has been a generalized increase in dental fluorosis[20].

III. In Consideration of the concerns raised above, we urge fluoridated cities, States with mandatory fluoridation, healthcare professionals, and public health authorities, to review ALL current information available, and use this information to re-evaluate current practices.

IV. Congressional Investigation is Appropriate

This Statement of Concern (same substance, slightly different content and form), along with a significant list of signatures, was unveiled at the May 6, 2003 EPA Science Forum session on fluoridation in support of the National Treasury Employees Union Chapter 280 (EPA union of professionals) renewed call for a Congressional investigation. No authorities from government agencies or non-governmental organizations responded to widespread EPA invitations over a six-week period, to attend this session to explain/defend the practice of fluoridation. In view of this fact, and also that some serious questions of propriety have been posed but not addressed, about the formulation of the EPA's drinking water standards for fluoride[21], as well as the downgrading of cancer bioassay data by the EPA in 1990[22], it now seems especially valid to ask Congress to hold hearings that will compel promoters to answer many unanswered questions.

It is appropriate that the U.S. Congress undertake an in-depth investigation of this public policy that is endorsed by major U.S. government agencies, but has never been adequately reviewed in its long history. Considering that there is an absence of research on silicofluorides, and that the latest scientific research on toxicity of fluorides has never been included in any government policy-making, and considering the many unanswered questions and concerns, we join the USEPA Union of professional employees in calling for a full-scale Congressional investigation into the public policy of fluoridation.

REFERENCES

to Superscript Numerals in Statement of Concern on Fluoridation

(For a more comprehensive list of scientific literature, see Bibliography section at www.SLweb.org)

1 CDC (1993). *Fluoridation Census* 1992.

2 National Sanitation Foundation International. (2000) Letter from Stan Hazan, General Manager, NSF Drinking Water Additives Certification Program, to Ken Calvert, Chairman, Subcommittee on Energy and the Environment, Committee on Science, US House of Representatives. July 7. http://www.keepersofthewell.org/product_pdfs/NSF_response.pdf

3a Damkaer DM, and Dey DB 1989. Evidence for fluoride effects on salmon passage at John Day Dam, Columbia River, 19821986. *N. Am. J. Fish. Manage*.9:154162. Davison A. and Weinstein L. The effects of fluorides on plants. (1998) *Fluorides and the Environment*. Earth Island Institute. www.earthisland.org.

3b Canadian Environmental Quality Guidelines, http://www.ec.gc.ca/ceqgrcqe/ English/Html/GAAG_Fluoride.cfm

4 Personal communication with Dave Paris, Manchester Water Works, NH. (January 2001) Calculation based on estimated twoliters/ person/day used for drinking and cooking.

5 EPA. (1999) Letter from Charles Fox, Assistant Administrator, Office of Water, to Ken Calvert, Chairman, Subcommittee on Energy and the Environment, Comm. on Science, US House of Representatives. June 23, 1999 http://www.keepersofthewell.org/gov_resp_pdfs/EPAresponse1.pdf

6 Masters RD, et al. (2000). Association of silicofluoride treated water with elevated blood lead. *Neurotoxicology*. 21:6, 1091 1099. Masters RD, and Coplan M. (1999). Water treatment with silicofluorides and lead toxicity. *International Journal ofEnvironmental Studies*. September.

7 **Neurotoxicity**

Varner, J.A. et al (1998). Chronic administration of aluminumfluoride and sodiumfluoride to rats in drinking water: Alterations in neuronal and cerebrovascular integrity. *Brain Research*, 784, 284298. Mullenix, P. et al (1995). Neurotoxicity of sodium fluoride in rats. *Neurotoxicology &Teratology*,
Lu, Y. et al (2000). Effect of highfluoride water on intelligence of children. *Fluoride*, 33, 7478. Li, X.S., (1995). Effect of fluoride exposure on intelligence in children. *Fluoride*, 28:4, 189192
Zhao, L.B. et al (1996). Effect of highfluoride water supply on children's intelligence. *Fluoride*, 29, 190192.

8 **Bone Pathology**

Riggs, B.L. et al (1990). Effect of fluoride treatment on the fracture rates in postmenopausal women with osteoporosis. *N. Eng. J. Med*., 322, 802809.
Li Y, et al.(2001). Effect of longterm exposure to fluoride in drinking water on risks of bone fractures. *J Bone Miner Res*.16(5):9329.
Kurttio, P., N. Gustavsson, et al. (1999). Exposure to natural fluoride in well water and hip fracture: A cohort analysis in Finland. *American Journal of Epidemiology* 150(8): 817824.
Jacobsen, S., J. Goldberg, et al. (1992). The association between water fluoridation and hip fracture among white women and men aged 65 years and older; a national ecologic study. *Annals of Epidemiology* 2: 617626.
Danielson, C., J. L. Lyon, et al. (1992). Hip fractures and fluoridation in Utah's elderly population. *Journal of the American Medical Association* 268(6): 746748.
Sowers, M., M. Clark, et al. (1991). A prospective study of bone mineral content and fracture in communities with differential fluoride exposure. *American Journal of Epidemiology* 133: 649660.

It should be noted that there are studies that found no relation between fluoridation and fractures.

9 **Reproductive Effects**:
Freni SC. (1994). Exposure to high fluoride concentrations in drinking water is associated with decreased birthrates. *J Toxicology and Environmental Health* 1994;42:109121.
Susheela AK, Jethanandani P (1996). Circulating testosterone levels in skeletal fluorosis patients. *J Toxicol Clin Toxicol* 34(2):1839.
Kumar A, Susheela AK (1994). Ultrastructural studies of spermiogenesis in rabbit exposed to chronic fluoride toxicity. *Int J Fertil Menopausal Stud* 39(3):16471.

10 **Impact on Pineal Gland**
Luke, J. (1997). *The Effect of Fluoride on the Physiology of the Pineal Gland*. Ph.D. Thesis. University of Surrey.
Luke, J. (2001). Fluoride deposition in the aged human pineal gland. *Caries Res*. 35:125128.

11 **Genetic Damage**
Department of Health & Human Services (1991). Review of fluoride benefits and risks. App. H. H1H6.
Mihashi M, & Tsutsui T. Clastogenic activity of sodium fluoride to rat vertebral bodyderived cells in culture. *Mutation Research* May 368(1):713.
Aardema MJ, et al (1989). Sodium fluorideinduced chromosome aberrations in different stages of the cell cycle: a proposed mechanism. *Mutation Research* 223 191203.
Caspary WJ, et al (1987). Mutagenic activity of fluorides in mouse lymphoma cells. *Mutation Research*.187(3):16580.
Tsutsui T, et al. (1984). Cytotoxicity, chromosome aberrations and unscheduled DNA synthesis in cultured human diploid fibroblasts induced by sodium fluoride. *Mutation Research* 139:193198.

12 **Thyroid Pathology**
Bachinskii PP, et al. (1985). Action of the body fluorine of healthy persons and thyroidopathy patients on the function of hypophysealthyroid system. *Probl Endokrinol (Mosk)* 31(6):259.
Zhao, W. (1998). Longterm effects of various iodine and fluorine doses on the thyroid and fluorosis in mice. *Endocr Regul* 32(2):6370.
Jooste PL, et al. (1999). Endemic goitre in the absence of iodine deficiency in schoolchildren of the Northern Cape Province of South Africa. *Eur J Clin Nutr* 53(1):812.
Lin FaFu; et al (1991). The relationship of a lowiodine and highfluoride environment to subclinical cretinism inXinjiang. *Iodine Deficiency Disorder Newsletter*. Vol. 7. No. 3.
Galletti, P. & Joyet, G. (1958). Effect of fluorine on thyroidal iodine metabolism in hyperthyroidism. *J. Clinical Endocrinology*. 18:11021110

13 **Dental Fluorosis**
Heller KE et al (1997). Dental caries and dental fluorosis at varying water fluoride concentrations. *J Pub HealthDent*, 57;No. 3, 136143.

14 Colquhoun, J. (1997). Why I changed my mind about water fluoridation. *Perspectives in Biology and Medicine* 41 2944 1997.
Limeback, H. (2000). Why I am now officially opposed to adding fluoride to drinking water. Open Letter. http://www.fluoridealert.org/limeback.htm

15 See: Connett, P. (2000). Fluoride: A Statement of Concern. *Waste Not* #414. http://www.fluoridealert.org/fluorideStatement. htm

16 FDA. (2000) Letter from Melinda K. Plaisier, Associate Commissioner for Legislation, to Ken Calvert, Chairman, Subcommittee on Energy and the Environment, Committee on Science, US House of Reps. Dec. 21.http://www.keepersofthewell.org/gov_resp_pdfs/fda_response.pdf

17 Stannard JG. (1991). Fluoride levels and fluoride contamination of fruit juices. *Journal of Clinical Pediatric Dentistry*. 16 (1): 3840.
1991 UNICEF. Fluoride in water: An overview. Accessed Online May 2001. http://www.unicef.org/programme/wes/info/fluor.htm
U.S. Public Health Service, Agency for Toxic Substances & Disease Registry (ATSDR), April, 1993:
Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine. ATSDR/TP91/ 17, 4/93

18 Letter 9/5/2000 of Charles Fox, Asst. Administrator EPA, Office of Water, to Ken Calvert, Chairman, Subcommittee on Energy and the Environment, Committee on Science, US House of Representatives.
http://www.keepersofthewell.org/gov_resp_pdfs/EPA_response2.pdf
U. S. Public Health Service, Agency for Toxic Substances & Disease Registry (ATSDR), April, 1993:
Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine. ATSDR/TP91/17, 4/93, pp. 1556.

19 CDC (1999). Achievements in Public Health, 19001999: Fluoridation of Drinking Water to Prevent Dental Caries.*Mortality and Morbidity Weekly Review (MMWR*), 48(41);933940 October 22, 1999.

20 CDC (2001). Recommendations for Using Fluoride to Prevent and Control Dental Caries in the United States.*Mortality and Morbidity Weekly Review*. August 17, 50(RR14):142.

21 Carton, RJ and Hirzy, JW (1998). Applying the NAEP Code of Ethics to the Environmental Protection Agency and the fluoride in drinking water standard. *Proceedings of the 23rd Annual Conference of the NationalAssociation of Environmental Professionals,* 2024
June 1998, San Diego, CA.

22. Hirzy, JW (2000). Statement before Environment and Public Works Subcommittee on Wildlife, Fisheries and Drinking Water. US Senate. June 29, 2000.

Appendix 6

Summary Findings, Conclusion and Recommendations of the Natick Fluoridation Study Committee

Natick Fluoridation Study Committee Report 9/27/97
Norman R. Mancuso, Ph.D. Benedict J.Gallo, Ph. D.Jason Kupperschmidt, B. Alfred J. Murray, M. S. T. Harlee S. Strauss, Ph.D.

Summary Findings, Conclusion, and Recommendations of the Natick Fluoridation Study Committee

The Natick Fluoridation Study Committee conducted a thorough review of the scientific literature and made the following findings regarding the benefits and risks of water fluoridation:

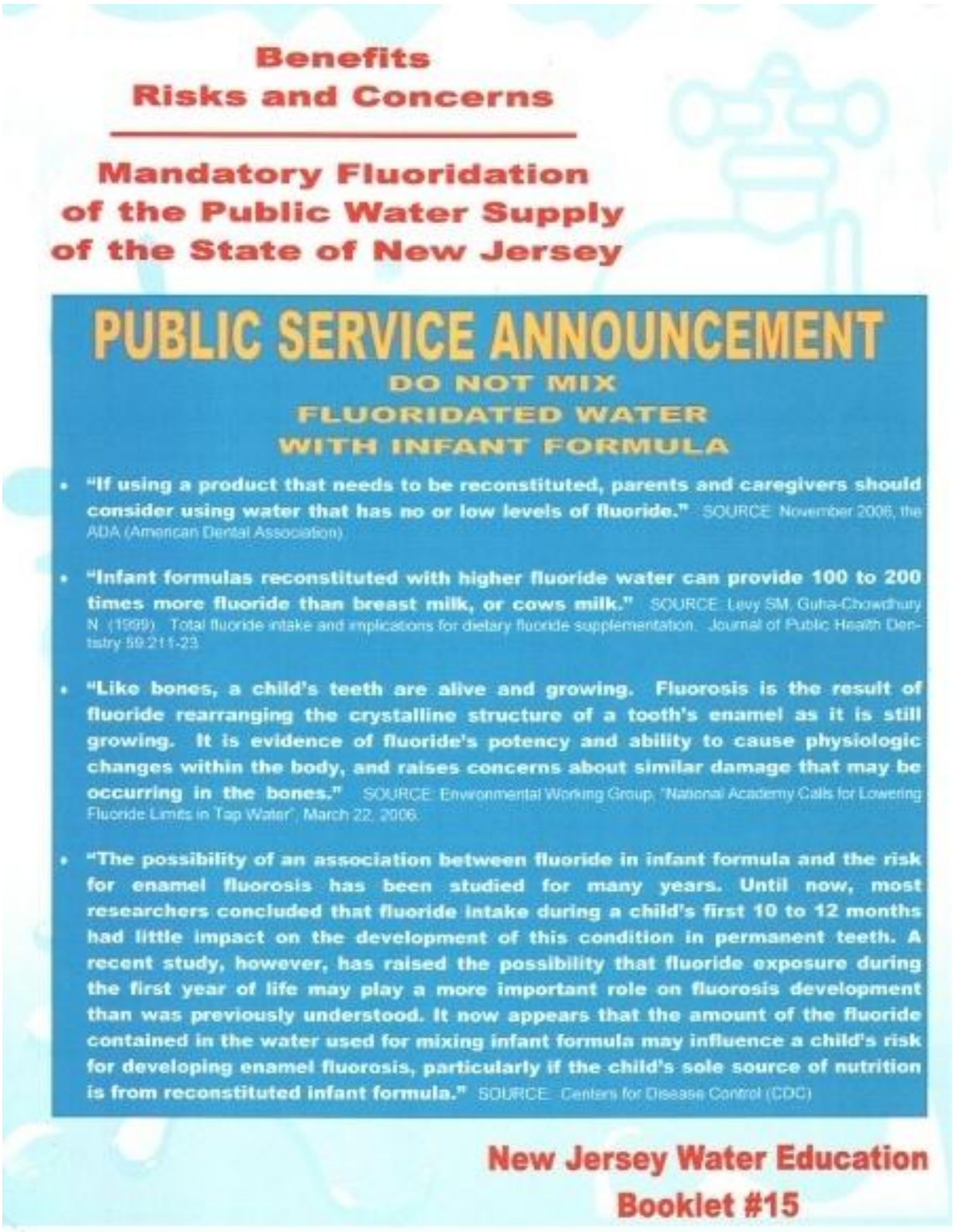
- Recent studies of the incidence of cavities in children show little to no difference between fluoridated and non-fluoridated communities.
- Ten to thirty % (10-30%) of Natick's children will have very mild to mild dental fluorosis if Natick fluoridates its water (up from probably 6% now). Approximately 1% of Natick's children will have moderate or severe dental fluorosis. Dental fluorosis can cause great concern for the affected family and may result in additional dental bills. It should not be dismissed as a "cosmetic" effect.
- Fluoride adversely affects the central nervous system, causing behavioural changes and cognitive deficits. These effects are observed at fluoride doses that some people in the US actually receive.
- There is good evidence that fluoride is a developmental neurotoxicant, meaning that fluoride affects the nervous system of the developing fetus at doses that are not toxic to the mother. The developmental neurotoxicity would be manifest as lower IQ and behavioral changes.
- Water fluoridation shows a positive correlation with increased hip fracture rates in persons 65 years of age and older, based on two recent epidemiology studies.
- Some adults are hypersensitive to even small quantities of fluoride, including that contained in fluoridated water. At least one such person is a Natick resident.
- The impact of fluoride on human reproduction at the levels received from environmental exposures is a serious concern. A recent epidemiology study shows a correlation between decreasing annual fertility rate in humans and increasing levels of fluoride in drinking water.
- Animal bioassays suggest that fluoride is a carcinogen, especially for tissues such as bone (osteosarcoma) and liver. The potential for carcinogenicity is supported by fluoride's genotoxicity and pharmacokinetic properties. Human epidemiology studies to date are inconclusive, but no appropriate major study has been conducted.
- Fluoride inhibits or otherwise alters the actions of a long list of enzymes important to metabolism, growth, and cell regulation.
- Sodium fluorosilicicte and fluorosilicic acid, the two chemicals Natick intends to use to fluoridate the water supply, have been associated with increased concentrations of lead in tap water and increased blood lead levels in children, based on case reports and a new, as-yet-unpublished study.
- If Natick fluoridates its water supply at the proposed level, most children under the age of three will daily receive more fluoride than is recommended for them.

Conclusion
The Committee reached the firm conclusion that the risks of over-exposure to fluoride far outweigh any current benefit of water fluoridation.
Recommendations
The Natick Fluoridation Study Committee unanimously and emphatically recommends that the town of Natick NOT fluoridate the town water supply. The Natick Fluoridation Study Committee unanimously and emphatically recommends that the Board of Selectmen take appropriate action to ensure that fluoridation of the town water supply does not take place.

Appendix 7

Public Health Service Announcement, New Jersey Water Education

Warning not to use Fluoridated Water for Infant Formula


Benefits
Risks and Concerns
Mandatory Fluoridation
of the Public Water Supply
of the State of New Jersey
PUBLIC SERVICE ANNOUNCEMENT
DO NOT MIX
FLUORIDATED WATER
WITH INFANT FORMULA
• "If using a product that needs to be reconstituted, parents and caregivers should consider using water that has no or low levels of fluoride." SOURCE: November 2006, the ADA (American Dental Association)
• "Infant formulas reconstituted with higher fluoride water can provide 100 to 200 times more fluoride than breast milk, or cows milk." SOURCE: Levy SM, Guha-Chowdhury N. (1999). Total fluoride intake and implications for dietary fluoride supplementation. Journal of Public Health Dentistry 59:211-23
• "Like bones, a child's teeth are alive and growing. Fluorosis is the result of fluoride rearranging the crystalline structure of a tooth's enamel as it is still growing. It is evidence of fluoride's potency and ability to cause physiologic changes within the body, and raises concerns about similar damage that may be occurring in the bones." SOURCE: Environmental Working Group, "National Academy Calls for Lowering Fluoride Limits in Tap Water", March 22, 2006.
• "The possibility of an association between fluoride in infant formula and the risk for enamel fluorosis has been studied for many years. Until now, most researchers concluded that fluoride intake during a child's first 10 to 12 months had little impact on the development of this condition in permanent teeth. A recent study, however, has raised the possibility that fluoride exposure during the first year of life may play a more important role on fluorosis development than was previously understood. It now appears that the amount of the fluoride contained in the water used for mixing infant formula may influence a child's risk for developing enamel fluorosis, particularly if the child's sole source of nutrition is from reconstituted infant formula." SOURCE: Centers for Disease Control (CDC)
New Jersey Water Education
Booklet #15

PUBLIC SERVICE ANNOUNCEMENT

PARENTS:

Do Not Mix Fluoridated Water With Infant Formula

While the New Jersey legislature contemplates mandatory fluoridation for the entire State, here is a look at the current recommendation for what a doctor can prescribe and what a fluoridation program will deliver:

INFANTS: 0 to 6 months of age*

*Parents are warned to **NOT** use fluoridated tap water to mix infant formula.*

Mandatory Fluoridation: 250 times the amount found in breast milk

TODDLERS: 6 months to 3 years of age*

Doctors in non-fluoridated communities cannot prescribe more fluoride than the amount found in one cup of fluoridated water per day.

Mandatory Fluoridation: 4 times the amount a doctor can prescribe

CHILDREN: 3 years to 6 years of age*

Doctors in non-fluoridated communities cannot prescribe more fluoride than the amount found in two cups of fluoridated water per day.

Mandatory Fluoridation: 2 times the amount a doctor can prescribe

Predicament

Controlling the amount of fluoride exposure among children across varied age groups. Fluoride cannot be removed by simple filtration. The cost of removal and the purchase of alternative water sources is expensive.

Solution

Read more on the website below, where you can decide for yourself. If you have any questions, please consult with your doctor.

Notice: Information does not constitute health or medical advice. Informational purposes only.

*"American Dental Association and American Academy of Pediatrics recommends brining fluoride supplements and infant formula mixed with fluoridated tap water because of the occurrence of dental fluorosis, the interference with normal development of tooth enamel that results in permanent bright white spots, brown staining, mottling, and fracture prone teeth from fluoride exposures during developmental years.

For free information, visit:

NewJerseyWaterEducation.com