Toxicological risks from novel forms of fluoride in drinking water and in the environment.

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The source of novel forms of fluoride.

The production of novel forms of fluorine compounds by the chemical industry has expanded enormously in recent years, and there are many substances now in large-scale production that have no analogues in the natural world. Arguably the most irrational examples are the fluorosilicates, a waste product of the extractive industries producing phosphate fertiliser, aluminium and hydrogen fluoride, and which are now commonly diluted in municipal drinking water for supply to the general public.

Fluorosilicates are highly toxic, and are classed by many environmental protection administrations as hazardous wastes, yet their extensive use in the fluoridation of municipal water supplies constitutes a very significant and virtually uncontrolled release of entirely novel fluorinated effluents, whose toxicological properties are almost unexplored. The purpose of this note is to draw attention to the potential risks associated with the administration of consumable products containing these chemicals to entire human populations, and the discharge of very significant volumes of fresh water containing them to the natural environment.

A water provider planning to start to fluoridate its product needs to be fully aware not only of the direct toxicological implications for its customers, but also of the ecotoxicological hazards associated with this practice. Before deciding to manufacture and supply this product, water companies should make sure that they are fully aware of the fate of the 99% of their water that is not drunk by customers but that is eventually discharged to rivers from water treatment works that, in many cases, they themselves operate. Both the medical toxicological and the ecotoxicological consequences are very much more extensive than all recent discussions appear to recognise.

Evidence of the toxicity of low-solubility natural fluoride to human populations.

The interminably repeated dismissal by proponents of water fluoridation of legitimate concern over the toxicity of fluoride with the claim that ‘fluoride is naturally present in all waters’ is deliberately misleading. Using modern extremely sensitive analytical equipment all elements can be detected down to almost vanishingly small concentrations. This spurious argument is used to suggest that fluoride is therefore a harmless substance, and that extremely low concentrations are entirely harmless, or even beneficial.

In fact, fluoride has no essential biochemical function in the human body, and its effects are purely pathological. Where it is present in a bioavailable form, it must be regarded as a potentially hazardous source of chemical contamination, and capable of contributing to a lifetime accumulation of fluoride within human and other animal bodies.

The calcium fluoride contained in natural waters has far lower solubility than the new forms used by the water industry, but even so it is capable of exerting a directly toxic effect on human populations. It is repeatedly asserted that drinking water containing 1 mgF/litre of ‘natural’ fluoride has no adverse effects on human health, and that where dental fluorosis does occur it is rare and of mild appearance. It is also claimed that the more serious levels of fluorosis are mainly caused by the ingestion of fluoride from other sources, especially highly fluorinated dental products such as toothpaste. There is clear evidence that this is nonsense.

The adverse impacts that water containing even the ‘recommended optimal concentration’ of fluoride can have on human populations otherwise unexposed to any other significant source of fluoride is well demonstrated by evidence from the palaeopathology of the Dilmun Culture in the Persian Gulf.

For seven thousand years the inhabitants of what is now Bahrain have been drinking water from wells and springs fed from an aquifer containing around 1.3mgF/l. This is slightly less than the maximum permissible concentration of fluoride (1.5mgF/l) established in the European drinking water quality legislation. The main additional dietary sources of fluoride intake were tea and fish, but no fluoridated dental products were available at

that time. Calcium fluoride absorption from food is low [1, 2], and amongst the agrarian communities alternative sources of dietary fluoride were infrequent and of little signficance. The fluoride contained in their drinking water was the only effectively bioavailable source of fluoride for the Dilmun people.

Yet archaeological evidence reveals that even the agrarian population of the Dilmun Culture had an extremely high incidence of dental caries. During the Islamic Period up to 80% of adult teeth were carious, whilst amongst children aged 3 to 6 years, 17% of deciduous teeth were carious [3]. At the same time, the prevalence of dental fluorosis was around 50%, with up to 20% of the population experiencing moderate or severe fluorosis. Even severe skeletal fluorosis was fairly common, with manual labourers being particularly prone to this debilitating condition.[4]

Since the prevention of caries in children is the principle objective of water fluoridation, the evidence of the high prevalence of dental fluorosis in children in the Bahraini population confirms the significant medical risks that this practice presents to both children and adults. At the same time, for these communities the supposed 'optimal concentration' of fluoride in the drinking water was still manifestly incapable of preventing the very high prevalence of dental caries in the population.

Dangers of relying on toxicological data on simple ionic fluorides when dealing with water fluoridation.

The water quality criteria relating to fluoride are based almost exclusively on toxicological data on the effects of exposure of subjects (both human and animal) to purified solutions of sodium fluoride or a related highly soluble simple ionic fluoride. Very few comparable investigations of the toxicity of the fluorosilicates that are actually employed in fluoridation have been made, and it is simply assumed that fluorosilicates dissociate entirely into fluoride ions and harmless hydration compounds of silicon.

In fact, there is no foundation on which such assumptions can safely be based. 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, fluorosilicates dissociate in highly complex fashion in water, with an astonishing range of complex derivatives forming at different pH values, none of whose toxicological properties has been investigated in sufficient detail to provide confidence that they have no potentially adverse medical implications.

The changing environmental threat from fluoride.

Historically, the fluoride found in natural water supplies has been derived from solution of relatively insoluble calcium fluoride. This is why it is generally present only at very low concentrations, normally of a few parts per million. But for commercial use it is too difficult to dissolve calcium fluoride at municipal water treatment works, so very much more soluble sources of fluoride have been introduced. These are sodium fluoride and fluorosilicic acid (or its sodium salt).

Fundamental questions are raised by the practice of water fluoridation, since the practice introduces new forms of fluorinated substances that are not found in nature and that have different chemical and physical properties to naturally-occurring fluoride. Unfortunately, the implications of the rise in exposure to complex fluorosilicates, and even to a highly soluble form of a simple ionic fluoride such as sodium fluoride, have been either dismissed or ignored by fluoridation proponents, who appear to have little understanding of even the basic principles of medical and ecological toxicology.

The contamination of surface waters by fluoride is not solely the result of discharges of fluoridated effluents. Even before treated potable water reaches its consumers a large proportion of it may escape to groundwater,

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1 Afseth J, Ekstrand J, Hagelid P, 1987. Dissolution of calcium fluoride tablets *in vitro* and bioavailability in man. Scand. J. Dent. Res. 95, 191-192.

2 Calcium fluoride as a source of fluoride added for nutritional purposes to food supplements. Scientific Opinion of the Panel on Food Additives and Nutrient Sources added to Food (Question No EFSA-Q-2005-088) The EFSA Journal (2008) 882, 8-15

3 Frohlich B and Littleton J. An Analysis of dental pathology and diet on historic Bahrain**.** Paléorient 15(2):59-75 (1989) http://www.persee.fr/web/revues/home/prescript/article/paleo_0153-9345_1989_num_15_2_4509

4 Littleton J. Paleopathology of skeletal fluorosis. American Journal of Physical Paleopathology 109:465-483 (1999) http://www.clas.ufl.edu/users/krigbaum/4468/Littleton_AJPA_1999_fluorosis.pdf

through leakage. In the UK, up to 30% of the total local production may escape from older network infrastructures into groundwater aquifers. The question of what effects this inadvertent addition of novel forms of fluoride to sub-surface aquifers may have in the environment is almost never addressed in debate.

The question also arises in agricultural regions, since fluoride is taken up by roots and may accumulate in vegetable produce fed to humans and livestock, as well as endemic wildlife. In already acidic soils, and in soils and waters affected by acid rain, a range of ecotoxicological effects occurs that can have profound environmental and economic repercussions for the farming and fishing communities.

The differing chemistry of fluoridation chemicals in water

There is a considerable difference in the chemical behaviour of fluoridated effluents once they are discharged to rivers and the sea, depending on both the original substance that was used to fluoridate the water supply and on the physico-chemical characteristics of the receiving water itself. So it is important to be aware of the very different chemical dynamics of simple fluorides and of the fluorosilicates used widely in water fluoridation.

Simple fluorides. One source of the 'fluoride' used in water fluoridation is sodium fluoride. This is an ionic substance - its component atoms are present as monovalent ions (elements or substances that have a single electrical charge) that are free to move totally independently in the water. Although they are present in a fixed ratio - in this case, one sodium ion to one fluoride ion - other sources of sodium ions may also be present, derived from other dissolved salts. The sodium ions have no effect on the toxicity of the fluoride ions themselves.

Fluorosilicates. However, when fluorosilicic acid (or its sodium salt) is used to fluoridate water supplies, its chemistry in the receiving system is far more complex. Fluorosilicates are not simple substances like sodium fluoride, and they behave in entirely different fashion, depending on the acidity of the water into which they are introduced.

When fluorosilicates are added to water they dissociate to form fluorosilicate ions $[SiF_6]^{2-}$ with two negative electrical charges, accompanied by either two individual ions of hydrogen H^+ (from fluorosilicic acid) or of sodium Na^+ (from sodium fluorosilicate). The individual elements, silicon (Si) and fluorine (F) in the fluorosilicate ion cannot move independently - at neutral pH they act as the complex substance fluorosilicate.

Fluorosilicates are therefore emphatically **not** identical to 'fluorides'. In fact, fluorosilicates should not be referred to as 'silicofluoride', because this improperly implies that they are fluorides and have similar properties. This spurious argument is often used to mislead audiences into believing that fluorosilicates are chemically interchangeable with true fluorides, and that adding fluorosilicate to drinking water is merely a 'topping up' process to augment fluoride concentrations below the 'optimal' level for preventing tooth decay.

The role of acidity in fluoride chemistry.

At neutral acidity, water has a 'pH value' of 7. Alkaline water has a higher value, whilst acidic water has a lower value. The manner in which fluorides and fluorosilicates react to changes in pH is extremely relevant to discussions on the form in which 'fluoride'is given to the public. The acidity of the human stomach - around pH 2 to 3 - governs the chemical form in which fluoridation chemicals are present and exert their toxic effects on humans..

The effect of dissolving any form of fluoride or fluorosilicate in drinking water at the restricted permissible pH range for drinking water - generally between 6 and 9 - has no effective toxicological significance. **It is the toxicology of complex fluorinated substances at very low pH that governs whether or not fluoridation presents a hazard to human well-being and to the environment.**

Fluoride and fluorosilicate dissociation in acidic conditions.

When simple fluorides are dissolved in water, they are in the ionic form, F^-. Provided that no aluminium is present, they remain so at all relevant pH levels, whether in pure water or in the acidity of the stomach. Fluoride in the industrial sodium fluoride sometimes used in fluoridation is more bioavailable than that in calcium fluoride, whilst fluoride from water containing fluorosilicates is even more completely absorbed.

At around the normal pH of 7, approximately 97% of the fluorine in fluorosilicate 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 fluorosilicate is pre-

sent 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.

These reactions cannot occur in the case of simple fluorides in drinking water, and it is for this reason that research data on the toxicity of fluorides to animals (and occasionally humans) that have been obtained using these forms of fluorides cannot be relied on for assessing the toxicity of fluorosilicate-dosed water.

The role of alumino-fluoride complexes in aluminium toxicology

Concern over the resultant enhanced bioavailability of fluoride from these novel sources is compounded by the parallel practice of using aluminium sulphate as a flocculant to clarify raw waters, before they are treated for municipal supplies. Until recent times the bioavailability of both fluoride and aluminium was extremely low, but this has changed very significantly in recent decades, as discharges of the effluents from waste water treatment works, containing significant concentrations of highly mobile fluoride, have increasingly contaminated the same freshwater sources on which other municipal water supplies depend.

The increased bioavailability of aluminium in the presence of traces of fluoride has been reported by many researchers, in both the human body and the environment in general. In such circumstances, the toxicity of aluminium is significantly increased, and this is almost entirely attributable to the formation of alumino-fluoride complexes in acidic conditions.

In waters containing even a trace of aluminium, the medical and ecological significance of the change from insoluble to highly soluble (and therefore much more bioavailable) forms of fluoride has gone almost unnoticed. Both fluoride and aluminium are recognised neurotoxins, and in the environment - including the 'environment' of the human stomach, their individual toxicities are augmented by the formation of aluminium-fluoride complexes.

These have been found to promote the adsorption of these neurotoxins into the blood stream, and their translocation from the blood into the brain. The reconfiguration in the presence of aluminium of the prion protein alpha-synuclein in brain cells, and the subsequent development of the precursors of beta-amyloid plaque at such locations, has been reported widely in recent literature, and aluminium has been repeatedly implicated in the development of a number of synucleopathies that result in the development of dementia (for example, Alzheimer's Disease, some forms of Parkinsonism and Multiple Systems Atrophy) [5].

The exact role of aluminium in the development of these devastating conditions remains unclear, but the evidence that it is involved in their pathology is increasingly persuasive. The recent sudden onset of rapidly fatal early dementias in a number of individuals exposed to the 1988 Lowermoor Incident in the UK [6], and the extremely high levels of aluminium in brain tissues at post mortem analysis, suggests that exposure to aluminium and fluoride under severely acidic conditions may be linked to the ability of aluminium to cause changes in brain tissues that are capable of leading to the development of dementia in later life.

In the UK today (3rd February 2010), the Alzheimer's Research Trust has announced that the prevalence of dementia is significantly greater than had been previously supposed. The possibility that the rising prevalence of this form of dementia may be related to the increasing prevalence of non-natural forms of fluoride in water and other consumable products cannot be excluded. Since these dementias are increasing in prevalence, are individually devastating, and of immense social and economic concern, the risk that any ill-considered action that increases the prevalence of such discharges may exacerbate the problem needs to be taken extremely seriously.

Mobility of alumino-fluorides in the human body.

When absorbed into blood serum, fluoride is able to cross the placental barrier during pregnancy, resulting in a deterioration of brain function in fetuses

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[5] For example, see Jellinger KA. Alpha-synuclein pathology in Parkinson's and Alzheimer's disease brain: incidence and topographic distribution--a pilot study. Acta Neuropathol. 2003 Sep;106(3):191-201.

[6] Draft Report, appendices and additional information relating to the Lowermoor incident., http://cot.food.gov.uk/cotwg/lowermoorsub/draftlowermoorreport/

> ***Elevated fluoride content was found in embryonic brain tissues obtained from required abortions in areas where fluorosis was prevalent. These studies showed poor differentiation of brain nerve cells and delayed brain development*** [7]

This is consistent with the recently disclosed Chinese evidence of significant levels of mental impairment in children living in areas in which drinking water contains fluoride.

Both aluminium and fluoride individually are potent neurotoxins, whilst the ecotoxicological significance of aluminium is mainly due to the presence of alumino-fluorides in acidic soils. In animals, including man, at the pH of the stomach and in the presence of the quantity of fluoride that is specified for fluoridated water supplies, all of the residual aluminium present in purified municipal water combines with the fluoride to form one or more complex ionic alumino-fluorides. This is the case for both simple fluorides (including 'natural' fluoride), and for fluorosilicates.

These complexes have enhanced capacity to cross both the gut-blood barrier and the blood-brain barrier [8], and are responsible for increased rates of translocation of both aluminium and fluoride to the human brain.

> ***the simultaneous administration of fluorine and aluminum in drinking water increased plasma concentrations of aluminum in rats. The same authors have reported that fluoride enhances the uptake of aluminum, whereas aluminum suppresses the uptake of fluoride. This effect seems to depend on the high affinity of aluminum to fluoride and the formation of complexes in the gastrointestinal tract associated with the breakdown of polymer forms and rapid uptake of aluminum by cells of the intestinal mucosa.***[9]

This apparent reduction in the neurological threat from fluoride in the presence of aluminium is of less toxicological significance that the enhanced rate of uptake of aluminium. Fluoride in soft tissues is relatively mobile and can be transported to relatively safe permanent storage in bone, whereas aluminium in brain tissues appears to be permanently locked into brain cells, and its accumulation has significant long-term neurological significance in aging people.

The role of aluminium and alumino-fluorides in the aetiology of synucleopathies such as Alzheimer's Disease (AD) has also been highlighted in recent research. Strunecká has suggested that

> ***pathologic changes are not raised by aluminum alone, but also by aluminofluoride complexes. However, aluminofluoride complexes may act as the initial signal that stimulates impairment of homeostasis, degeneration, and death of the cells. By influencing energy metabolism these complexes can accelerate aging and impair the functions of the nervous system. With respect to the etiology of AD, the long-term action of aluminofluoride complexes may represent a serious and powerful risk factor for the development of this devastating disease.*** [10]

Finally, the toxicological significance of the additional silicon present in fluorosilicates has been almost completely ignored. In acid water, fluorosilicates dissociate to release a series of intermediate soluble and colloidal silicon-containing compounds. These are capable of reacting with both aluminium- and fluorine-containing complexes, and the ecotoxicological significance of these various derivatives of fluorosilicates remains unknown.

The ecological risk of increasing environmental bioavailability of aluminium.

Thousands of tonnes of highly soluble fluoride and fluorosilicate are released to the environment by the water sector every year. The discharge of these novel chemicals increases the bioavailability of fluoride and related fluorinated substances, but also of both natural and artificial sources of aluminium. The risk that they pose to humans through their supply in drinking water also extends to non-human animals, including domestic pets, live-

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[7] Hatterslay JG (1999) Fluoridation´s defining moment. *J Orthomol Med* **14**: 1-20, 1999.

[8] Varner JA, Jensen KF, Horvath W and Isaacson RL. Chronic administration of aluminium-fluoride or sodium fluoride to rats in drinking water: alterations in neuronal and cerebrovascualr integrity.. Fluoride 31(2), 1998, pp 91-95 (Abstracted from *Brain Research 784* 284-298 1998)

[9] Graczyk A, Dlugaszek M. Biochemical processes and molecular mechanisms of aluminum toxicity. Rocz Panstw Zakl Hig 1993;44:23-42 (in Polish)., cited by Anna Lubkowska A, Zyluk, B, Chlubek D. Interactions between fluorine and aluminium. Fluoride Vol. 35 No. 2 73-77 2002 Editorial Review 73

[10] Strunecká A. Aluminum plus fluoride. a new, deadly duo in AD. The News in Dementia 1999;1:10-1.

stock, fish and other wildlife.

The complex chemical reactions that occur as a result of the dissociation of fluorosilicates in both the human stomach (and of course in other animals) and in acidic environments such as soils and lakes affected by acid rain, are unprecedented in nature. Terrestrial and aquatic wildlife are ultimately all at risk of the same hazards as are humans. In addition, alumino-fluoride complexes are severe inhibitor of plant growth, and have significant ecological and economic implications for primary productivity in both terrestrial and aquatic habitats.

Even in such a well-buffered environment as the sea, fluoride bioaccumulation in some species is remarkably high, and it is not clear whether or not this is a recent phenomenon that may be related to the increased bioavailability of environmental sources of fluoridet For example, the carapace (shell) of the marine euphausid shrimps known as Krill contains 2,000 to 3,000 mg F/kg [11] - 500 to 1000 times its usual concentration in seawater. Krill are a crucial part of the Arctic and Antarctic food chain on which marine fish and mammals depend.

Some cetaceans are known to develop very high bone fluoride concentrations, but skeletal fluorosis does not appear to be prevalent in Odontoceti (toothed) or Mysticeti (baleen) whales [12] . It is possible that whales have developed tolerance to high fluoride concentrations in their food, but whether the increasing discharge of more bioavailable fluoride to, ultimately, the marine environment will have any significant toxicological effect on them in the future is unknown.

There has been almost no investigation into the risks posed by such widespread releases of these neurologically aggressive environmental toxins to the environment. The universal requirement for any large-scale release of potentially polluting substances to be subject to Environmental Assessment appears never to have been enforced in the case of municipal-scale water fluoridation schemes. By default this has permitted the uncontrolled spread of this form of environmental contamination across vast areas of territory, exposing large human populations and connected environments to an increasing background exposure to a toxic, mutagenic and almost certainly carcinogenic substance.

The risks of further threats to the environment and to human health from an increased prevalence of water fluoridation cannot be quantified but are both credible and real. No further fluoridation projects should be permitted to be commenced, and the validity of existing schemes must be subject to critical reappraisal. If they cannot be justified under the Precautionary Principle, then they should be terminated and alternative (and more reliable and precisely targetable) ways to protect children's teeth adopted.

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[11] Adelung D, Buchholz F, Culik B and Keck A. Fluoride in tissues of Krill *Euphausia superba* Dana and *Meganyctiphanes norvegica* M. Sars in relation to the moult cycle**. Polar Biology, 7(1) pp 43-50 (1987)**

[12] Mikaelian I, Qualls CW Jr. De Guise S, Whaley MW and Matineau D. Bone fluoride concentrations in beluga whales from Canada. Wildl Dis. 1999 Apr;35(2):356-60.