Admin, LACO

Subject: FW: submission regarding Inquiry into the role of diet in type 2 diabetes prevention and management

From:
Sent: Sunday, 2 September 2018 5:57 PM
To: Committee, Education & Health Standing <laehsc@parliament.wa.gov.au>
Subject: submission regarding Inquiry into the role of diet in type 2 diabetes prevention and management

Honourable WA Parliament,

Please accept this email as my submission regarding Inquiry into the role of diet in type 2 diabetes prevention and management.
Diabetes is a major killer causing huge suffering along the way. It can cause Blindness, shuts down your Kidneys and leads to continuous infection and amputations.

Please consider following points and take provided scientific research seriously:

The cost of type 2 diabetes to the community is quite considerable and could be reduced.

Prevention and intervention programs are not adequate.

Restrictive diets to eliminate the need for type 2 diabetes medication are available, proven to help and not advocated enough.

Healthy eating including *consumption of food and beverages (non-alcoholic and alcoholic)* should be encouraged worldwide, taught at schools, workplaces and universities.

Regulatory measures for the food and beverage industries are essential.

Social and cultural factors as well as behavioural aspects need to be considered, as they affect healthy eating, in particular for:

- at-risk adults
- children and adolescents
- Aboriginal communities
- ethnic groups at greater risk of developing diabetes
- people in rural and regional areas
- low income earners

Culprit number 1: Sugar. Apart from widely organized information sessions for the population, it is most necessary to regulate the food industry. They should be controlled and limited to much smaller amounts of sugar they are allowed to add to their products with proper labelling.

Culprit number 2: Fluoride is known to damage the Pancreas, reducing Insulin production, as well as interfering with Glucose metabolism, leading to Diabetes and Obesity. Importantly it is recognized as a Low-Dose Endocrine Disruptor.
"... One of the most significant findings against Fluoride is the discovery that hydroxyapatite enhances the mitogenesis of mammary cells, amplifying the malignant process and resulting in accelerated tumor growth [Wilson 2014]. Recently Fluoride, delivered by mandated fluoridation, has been linked to Hypothyroidism [Peckham 2015], Diabetes and Obesity [Vandenberg 2012, Pain 2015b, Pre-term Birth and Impaired Neurodevelopment [McArthur 2015] and Attention Deficit Hyperactivity Disorder (ADHD) [Malin 2015]...."

Exert:
The focus of this brief review and bibliography is the
effect of Fluoride on insulin production, sensitivity and resultant Diabetes. No distinction was made in searching the literature between Diabetes Insipidus, Type 1 Diabetes Mellitus caused by the pancreas not producing adequate amounts of insulin, and Type 2 Diabetes Mellitus caused by the body's cells becoming less responsive to insulin that is produced.
Previous studies have emphasized the adverse impact of Fluoride on diabetic patients because they typically consume much larger quantities of water than average humans and have impaired kidney function leading to higher risk from the diverse toxic effects of Fluoride [see for example Prystupa 2011, NRC 2006, Marier 1977]. It has also been shown that Fluoride toxicity is greater in diabetics [Banu Priya et al. 1997].
Diabetics suffer impaired glucose tolerance (IGT), hypertension, hyperlipoproteinemia and coronary disease. They have a higher risk of death from breast cancer [Youlden et al. 2009], pancreatic cancer [Michaud 2004], uterine cancer [Purdie 2001] and colorectal cancer [Youlden 2008]. Diabetics also suffer reduced bone mass and strength through Fluoride exposure [Dunipace et al. 1996].
Diabetics have a higher incidence of chronic kidney disease which leads to impaired renal clearance of fluoride [Hanhijarvi 1974], the "vicious cycle". The Australian Institute of Health and Welfare has forecast the proportion of diabetics undergoing transplants or dialysis would rise to 64 per cent in 2020 from 45 per cent in 2009 [Henderson 2012].
The total number of Australians being treated for end-stage kidney disease is forecast to rise by up to 80 per cent to about 4300 in the coming decade.
Diabetic women have a higher risk of premature birth and low birth weight children [Patel 1975].
The immense scale of the Diabetes problem is summarized by the World Health Organization [Bergman et al. 2013] as follows: "The number of diabetics in the world is expected to increase from 194 million in 2003 to 330 million in 2030 with three of four affected individuals living in developing countries. The global health expenditure on diabetes alone is expected to rise to US$ 490 billion in 2030 – 12% of all per capita health-care expenditures [Zhang et al. 2010]. The burden of premature death from diabetes in developing countries is similar to that of HIV/AIDS, yet the problem is largely unrecognised in these areas. "
There has been an explosion in the incidence of diabetes in the developed countries over the last 40 years [Bergman et al 2013] which matches the timescale of deliberate fluoridation of public water supplies in those countries.
There is strong evidence that Fluoride causes Diabetes.
Fluoride directly reduces insulin synthesis in rats [Lin et al. 1976]. Microcirculatory defects, increased capillary permeability and altered protein biosynthesis in the pancreas is associated with Fluoride exposure. Because human hormones interact with each other, the known adverse effect of Fluoride on melatonin production and the knock-on effect on insulin should also be considered [Rasmussen et al. 1999]. The fact that Fluoride causes hypothyroidism also exacerbates the damage to diabetics through reduction of peripheral glucose metabolism [Cettour-Rose 2005].
A genetically inherited condition demonstrates an association between pineal gland hyperplasia and insulin resistance [West et al. 1980].
Blood fluoride level of just 234 ppb after a single acute exposure caused significant impairment in glucose metabolism, as evident by sharp rises in blood glucose and decreases in insulin [Whitford 1987]. Similar results have been measured in rats and human volunteers [Rigalli et al. 1990, Suketa 1985].
Short-term acute exposures to high levels of fluoride generated by metabolism of the fluorinated anesthetic methoxylflurane impairs the kidney's ability to concentrate urine and produces a diabetes insipidus-like condition marked by excessive urination [Mazze 1977].
Pancreas pathological morphometry analysis via β cells [Hu et al. 2012] of rats exposed to Fluoride showed increased islet size. The same rats exhibited increased alkaline phosphatise and osteocalcin, increase of serum insulin level and a general decrease of glucagon level.
Rats with Fluoride induced diabetes that were encouraged to exercise demonstrated accelerated skeletal fluorosis [Lombarte 2013]. Diabetic rats also show enhanced contractile responses of arteries to sodium fluoride which directly stimulates GTP-binding proteins (G-proteins) [Weber 1996].

Insulin resistance in humans caused by chronic Fluoride exposure from drinking water is well known [Bergman et al. 2013, Vandenberg et al. 2012, Chiba et al. 2012, Menoya et al. 2008, Stephen 1994, Trivedi et al. 1993].

In chronic exposures, effects on glucose metabolism occurred when plasma fluoride concentrations exceeded 0.1 mg/L (5 µmol/L) [Rigalli et al. 1992, 1995], or just one 15th the concentration allowed in Australian drinking water. The US National Research Council [2006] stated "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."

Townsville in the state of Queensland, Australia, fluoridated since 1964, suffers 10% higher rate of diabetes than the rest of non-fluoridated Queensland [PHIDU 2005]. Townsville also suffers higher rates of hospital admissions for unspecified dental conditions as well as asthma, congestive heart failure, convulsions and epilepsy, congestive obstructive pulmonary disease, ear nose and throat conditions and pyelonephritis. Townsville also suffers increased death rates due to circulatory system, ischaemic heart disease, cerebrovascular disease – Stroke, chronic lower respiratory disease and cancer of the trachea [Queensland Hospital Data 2005-2006]. Townsville can therefore be considered a randomised control trial centre for Fluoride toxicology.

Workers in the phosphate fertilizer industry are exposed to Fluoride and experience higher incidence of diabetes as well as skeletal fluorosis [Renke 1987].

Workers in the cryolite industry also suffer Chronic Fluoride Intoxication (CFI) and have lower insulin and increased C-peptide serum levels [Tokar 1992]. It was shown that the incidence of diabetes increased with years of exposure. The observed lower serum insulin levels in Fluoride intoxication might be due to associated liver damage [Tokar 1992]. Liver damage has also been observed by Vasant and Narasimhacharya [2013a] who state "Exposure to fluoride through drinking water not only significantly increased plasma glucose and lipid profiles, but also elevated both hepatic and renal lipid peroxidation, hepatic lipid profiles and G-6-Pase activity with a reduction in plasma HDL-C, hepatic glycogen content, hexokinase activity and antioxidant status".

Fluoride induced hyperglycemia has been stated to be mainly due to increased hepatic glycogenolysis [Varadacharyulu et al. 1997]. Rabbits fed 16 mg of Fluoride per day exhibited hyperglycemia as well as reduction of bone strength through fluorosis [Turner 1997].

People exposed to high Fluoride levels in their drinking water suffer a high incidence of skeletal fluorosis. As demonstrated by Xie et al. [2000] they exhibit a higher and longer lasting blood glucose level after an oral glucose tolerance test (OGTT). Those with diagnosed skeletal fluorosis demonstrate high levels of serum insulin.

Diabetics are exposed to an acceleration of their disease due to water fluoridation. They typically drink much larger volumes of water [Prystupa 2011] and accumulate more Fluoride.

The mechanisms by which Fluoride induces diabetes most likely include antagonism to calcium and magnesium centred biochemistry [De Valk 1999]. Insulin secretion (both basal and glucose-stimulated) by isolated islets of Langerhans in vitro is inhibited as a function of fluoride concentrations [Rigalli et al. 1990, 1995].

Diabetics are more susceptible to Fluoride induced arterial contraction [Hattori et al. 2000] increasing risk of cardiovascular disease.

Fluoride induced diabetes will also cause damage to the periodontum and tooth loss [AHMAC 2001].

Diabetics are a "Sensitive Subpopulation" or "Vulnerable Group" and no attempt has been made by Australian health authorities to warn diabetics about Fluoride toxicity or protect them from harmful exposure.

Acknowledgment

I am grateful to Professor Paul Connett and the Fluoride Action Network whose online database facilitated identification of relevant studies. See FluorideAlert.org

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Note: Those marked * were deliberately excluded from the NMRC 2007 Review.

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Yours Sincerely
Dörte Dörte Planert,