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DRINKING WATER FLUORIDATION IN LONDON

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Drinking Water Fluoridation is Genotoxic and Teratogenic

Drinking water fluoridation has been implicated as a cause of gene damage leading to birth defects and cancer. Government regulators evaluate scientific studies based one of two regulatory philosophies. One philosophy demands absolute and unequivocal evidence of harm (a body count) while the other approach is called the precautionary principle which argues that some evidence of harm should cause stoppage of the use of an offending drug or environmental pollutant until the item is proven safe by further experiments. In Europe the precautionary principle is written into law while in Canada and the United States regulators depend on elevated body counts to act on a particular cause of harm. There is a current and growing body of peer reviewed scientific publications showing that fluoridated water causes gene damage leading to birth defects and cancer and that humans are genetically different in their sensitivity to levels of fluoride in their drinking water.

Some current peer reviewed scientific publications that have not yet been chewed over by the proponents of drinking water fluoridation are described below. These studies should have been sufficient to trigger eliminate of the addition of fluoride to the drinking water supply.

Resistance to fluoride toxicity: People are not genetically uniform clones they are highly polymorphic in their response to drugs and environmental toxins. According to a recent report fluorides mediate their actions through the MAPK gene signaling pathway and can lead to changes in gene expression, cell stress, and cell death. Different strains of inbred mice demonstrate differential physiological responses to ingested fluoride. Genetic studies in mice are capable of identifying and characterizing fluoride-responsive genetic variations. Ultimately, this can lead to the identification of at-risk human populations who are susceptible to the unwanted or potentially adverse effects of fluoride action (1).The MAPK pathway is a chain of proteins in the cell that communicates a signal from a receptor on the surface of the cell to the DNA in the nucleus of the cell. The signal starts when a growth factor binds to the receptor on the cell surface and ends when the DNA in the nucleus expresses a protein and produces some change in the cell, such as cell division.

The fluoride exposure of pregnant women living in Poznan (Poland)was determined by examination of fluoride levels in blood plasma. The values suggest that apart from drinking water, there were other important sources of fluoride in the examined sample. The results indicate that a reliable assessment of fluoride exposure in a given population cannot be based solely on the concentration of fluoride in drinking water. Relatively high levels of fluoride in blood plasma of examined women suggest that there is no need for fluoride supplementation in this group (2). Indeed, the genotoxicity of fluoride suggests that pregnant people should avoid drinking fluoridated water. Fluoride (F) bone injury in humans was

found to be modulated by polymorphism in the calcitonin receptor gene . The interactive effect of F burden and calcitonin receptor polymorphism increased the F bone injury in the study group (3).

Genetic factors underlie the susceptibility and the resistance to dental fluorosis (DF). The A/J (DF susceptible) and 129P3/J (DF resistant) mouse strains have been used to detect quantitative trait loci (QTLs) associated with DF on chromosome (Chr) 2 and Chr 11(4). QTLs have been found to drive F resistance in animals including , presumably, humans and it is essential to identify those in the population who are being poisoned by fluoridated drinking water. It has been found that among all cellular organisms there are widespread genetic switches and resistance proteins for F (5).

Fluoride damages genes and chromosomes: There is a large body of publications showing that were published prior to 2004. These studies showed that F caused chromosome aberrations in human and great ape chromosomes but not in rodents. Current studies continue to provide evidence of gene damage. In rat hippocampal neurons F caused DNA damage during DNA synthesis and caused cell cycle arrest during S-phase and enhances expression of NF-kappaB a gene product related to stress response (6). In human embryo hepatocytes F caused DNA damage, apoptosis (cell suicide) and p53 cancer gene expression (7).

Fluoride suppresses male reproduction: Exposure to F containing drinking water in male rats caused impaired sperm hyper activation which is essential for fertilization. The inhibition was achieved through impairment of a calcium signaling pathways (8). Female rats exposed to fluorinated drinking water gave birth to males suppressed in sperm production(9). Male rats exposed to F in drinking water had adversely effected fertility and reproduction (10).Fluoride in drinking water caused oxidative damage to the sperm of treated rats (11). Fluoride in drinking water hampered the reproduction of male rabbits through reduced sperm count and defective sperm (12). Rats exposed to F in drinking water expressed the death receptor Fas causing apoptosis (cell suicide) (13). Male mice exposed to F produced sperm in global gene expression profiling, 34 up-regulated and 63 down-regulated genes, which are involved in several sperm biological processes including signal transduction, oxidative stress, apoptosis, electron transport, glycolysis, chemotaxis, spermatogenesis and sperm capacitation, were significantly differentially expressed. As well apoptosis was induced in sperm (14).

Fluoride causes birth defects: F has been associated with fetal malformations since the 1940s A few recent publications will illustrate the state of knowledge in that area. F in water caused malformations in frog embryos in tadpoles F caused dysfunction of the neuro muscular system (15). F caused lipid peroxidation, DNA damage and apoptosis in human embryo hepatocytes (16). Rats exposed to F during early gestation showed changes in gene expression in genes associated with Tourettes'syndrome, obsessive compulsive disorders, Huntington's disease and schizophrenia in humans (17). Chronic F ingestion in drinking water caused a marked multigenerational destruction in lung tissue of rats (18).

To conclude: The well documented effects of F in drinking water include gene damage, birth defects, suppression of male reproduction and cancer. The entrenched proponents of F addition to drinking water justify that drug on the basis that the addition is a public health benefit and that the adverse impacts are not observed at the low level of F in drinking water. However, as reported in a number of

publications various sources provide adequate F to maintain dental health so that F addition to drinking water is neither necessary nor effective. Furthermore , animal studies are frequently rejected on the basis of the claim that the drinking water levels of F in the animal experiments are higher than that in municipal drinking water. However, there is a disturbing similarity in the concentration of F in many animal experiments and the level of F in drinking water. As well, a conversion of animal dose to human equivalent dose based on body surface area is essential in drug or pollutant safety evaluation. For example , the F concentration in drinking water in a mouse experiment must be multiplied by 0.08 for comparison with the F in human drinking water. The 10 ppm F used in a mouse experiment , for example , is equivalent to 0.8 ppm in human drinking water(19). The failure to heed the surface area conversion factor caused human injury when the concentration of drug in a mouse experiment was directly applied to healthy humans in clinical trials. Failure to heed the conversion factor seems to have been ignored in evaluating f in drinking water animal studies.

Removal of F from London's drinking water does not only save tax dollars it saves the health of F sensitive London residents.

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