Fluoride's Insidious, Deadly Effects

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Many of you have been following the lists learning process about the DOE's, the energy sector, the strategic metal sector, and the Mil / Ind Network's biggest health effect. Fluorides are causing problems on a massive global scale, and they are being covered up by these interests.

Many abstracts about the issues of fluorides connected to G-protein trigger effects, then the high bone concentrations, bone concentrations rising with age. You're also seeing some of the information on this process triggering the immune systems cytokine processes. Do note that the immune aging process and that of fluoride's is real similar to rise in T-cells, rise in IL-6 and IL-10. And the shut down of the macrophage systems and rise of varied viral's in the body.

Then keep in mind all the illnesses associated with bone meal use. Chickens and farm animals, like cows, are fed bone meal. This bone meal is rich in fluorides that cause them health problems as well. Even farm produce grown with fluoride contaminated phosphate fertilizers and sprayed with fluoride pesticides enter the health damage equation. These are the factors that are driving much of the mad cow and other diseases. These are the factors driving overuse of antibiotics in farm animals and these too entering the food chain.

One can now see the negative effects on mice and some generational dependence. Mice are bottom feeders and have long eaten things killed by poisons and been poisoned directly from rodenticides like fluoride. Experimental mice lines are largely damaged in their immune resistance from generational exposures to fluorides and other chemicals. The recent Wake Forest mouse line with resistance to all cancers and cancer viruses speaks to this.

In the very same process that mouse research has been tainted by immune damaged experiment animals, this same process affects HIV research. Here fluorides are highly connected to setting up the factors leading to high rates of HIV transmission. Human's now circum to the same environmental factors as the generational mice immune damage has in centuries before.

The factors of fluorides in contaminated rural zones in China play strong roles in

the chicken and swine flu's that sweep the globe each season. These same factors are involved in the West Nile Virus and many others soon to come.

Lets not forget the really big fluoride example about Freon breakdown in the upper atmosphere is driven by radiation. The Sun's radiation, that goes to ionize oxygen to make the ozone, is also the very same radiation process that makes the free fluorine and chlorine in the upper atmosphere from Freon [CFC] ionization. The chlorine goes to reduce the ozone filtering of UV-b and the fluoride ionization emissions make UV-b directly. The free Cl and F ions tend to pull to the poles due to the Earth's mag field. The UV-b and ozone hole effects are driven by radiation effects on alleged inert Freon [CFC's]. The rise in UV-b radiation goes to kills ocean surface plankton and damage the sea's food chain.

The atmospheric science types will talk about the Cl component and the short half life of Cl in the atmosphere, and they don't like to talk about the HF component and the 200 year atmospheric half-life of fluorides in the atmosphere. They especially don't like to talk about the plasma ion effects of fluorides in the upper atmosphere. This is what HAARP is messing with.

These environmental and health related problems are the makings for a virtual apocalypse. These are the factors that even the US Govt. itself hides from its own citizens. There are huge overlaps to factors presented in the issues of religious revelations.

These are the factors to keep in mind as many of you study fluoride linked issues. If you can follow the dialog above then you are ready to read this research survey report about how these factors are at the root of illness like CFS, GWS, and the ills around fluoride plants. See report at:
http://members.aol.com/magnu96196/cfs.html

These illnesses are no longer a mystery and the dangers of fluoride's in the environment are well proven.

Some remaining pertinent fluoride abstracts follow:

Aluminum fluoride reveals a phosphoinositide system within the suprachiasmatic region of rat hypothalamus.
http://www.ncbi.nlm.nih.gov/pubmed/2159821

Nadakavukaren JJ, Welsh DK, Reppert SM.

Laboratory of Developmental Chronobiology, Children's Service, Massachusetts General Hospital, Boston 02114.

The phosphoinositide (PI) transduction system has proven to be of major importance in several regions of mammalian brain. In this report, we examined in rats whether a PI system is present in the hypothalamic suprachiasmatic nuclei (SCN), the site of a biological clock that generate circadian rhythms. Autoradiographic localization of phorbol ester binding revealed moderate levels of protein kinase C, a component of the PI system, in the SCN. Hypothalamic explants containing SCN showed substantial incorporation of [3H]myoinositol into lipids. AlF4-, a non-specific activator of G proteins, produced a dose-dependent increase in inositol monophosphate (IP1) levels in the explants in calcium-free medium, with a maximum increase of 216% of control at 50 mM NaF. Medium containing 1.8 mM calcium stimulated a similar increase in IP1 levels, but the stimulatory effects of AlF4- and calcium were not additive, so that the effect of Al4- was obscured in medium containing calcium. AlF4- stimulated accumulation of IP1, as well as inositol bis-, and trisphosphate, over a 40-min time course in the presence and absence of lithium (10 mM LiCl). Lithium, a known inhibitor of phosphatases in the inositol phosphate recycling pathway, raised levels of all 3 inositol phosphates in SCN explants both at baseline (without A1F4-) and after 30 min AlF4- stimulation. The results show the existence of a lithium-sensitive PI system within the suprachiasmatic region of the rat hypothalamus.

PMID: 2159821 [PubMed - indexed for MEDLINE]

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Histopathological changes in rabbit testes during experimental fluorosis.
http://www.ncbi.nlm.nih.gov/pubmed/2341082

Department of Zoology, Punjabi University, Patiala, India.

The aim of the study was to evaluate relationship between infertility and the histological structure of the testes following the subcutaneous administration of different doses of sodium fluoride (5, 10, 20 and 50 mg/kg/day), for 100 days, to groups of six male albino rabbits; the six control animals were given 1 cc distilled water/kg b.w./day for the same length of time. Deficient maturation and differentiation of the spermatocytes and an increase in the amount of interstitial tissue were found in the experimental animals. In the higher dosage groups, spermatogenesis stopped and the seminiferous tubules became necrotic. The study thus established the existence of a definite relationship between fluorosis and testicular damage.

PMID: 2341082 [PubMed - indexed for MEDLINE

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The effect of fluorine on the developing human brain
http://www.ncbi.nlm.nih.gov/pubmed/1473206

[Article in Chinese]

Du L.

Department of Pathology, Guiyang Medical College.

Fifteen therapeutically aborted fetuses at the 5th-8th gestation month from the endemic fluorosis area were compared with those from the non-endemic area. Stereological study of the brains showed that the numerical density of volume of the neurons and the undifferentiated neuroblasts as well as the nucleus-cytoplasm ratio of the neurons were increased. The mean volume of the neurons was reduced. The numerical density of volume, the volume density and the surface density of the mitochondria were significantly reduced. The results showed that chronic fluorosis in the course of intrauterine fetal life may produce certain harmful effects on the developing brain of the fetus.

Psychopharmacology of fluoride: a review.
http://www.ncbi.nlm.nih.gov/pubmed/8056997

Department of Psychological Medicine, University of Otago Medical School, Dunedin, New Zealand.

Although the blood-brain barrier is relatively impermeable to fluoride, it does not pose an absolute barrier and fluoride has the ability to enter the brain. The literature was examined to assess the quality of the evidence for cerebral impairment occurring due to exposure to fluoride from therapeutic or environmental sources. Several surveys of persons chronically exposed to industrial fluoride pollution reported symptoms related to impaired central nervous system functioning with impaired cognition and memory. Examination of individual case reports showed the evidence for aetiological relationships between symptoms and fluoride exposure to be of variable quality. The evidence was seen as being suggestive of a relationship rather than being definitive. The difficulties with concentration and memory described in relation to exposure to fluoride did not occur in isolation but were accompanied by other symptoms of which general malaise and fatigue were central. Possible mechanisms whereby fluoride could affect brain function include influencing calcium currents, altering enzyme configuration by forming strong hydrogen bonds with amide groups, inhibiting cortical adenylyl cyclase activity and increasing phosphoinositide hydrolysis.

Publication Types:

Review Review, Tutorial PMID: 8056997 [PubMed - indexed for MEDLINE]

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The interrelationship of the thyroid and immune statuses of workers with long-term fluorine exposure
http://www.ncbi.nlm.nih.gov/pubmed/7709355

Balabolkin MI, Mikhailets ND, Lobovskaia RN, Chernousova NV.

Thyroid and immune statuses were studied in workers continuously exposed to fluorine. The examinees with euthyroid condition had immune disorders with an allergic tendency (increased number of B-lymphocytes, immunoglobulins A). In workers with subclinical hypothyrosis (tri-iodothyronine reduced in 51%), the immune alterations were more evident, T-lymphocytes count rose, but their functional activity declined, indicating impaired cooperation of immunocytes as a

result of imperfect control under low concentrations of tri-iodothyronine.

PMID: 7709355 [PubMed - indexed for MEDLINE]

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The phosphoinositide signal transduction system is impaired in bipolar affective disorder brain.
http://www.ncbi.nlm.nih.gov/pubmed/8632163

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The function of the phosphoinositide second messenger system was assessed in occipital, temporal, and frontal cortex obtained postmortem from subjects with bipolar affective disorder and matched controls by measuring the hydrolysis of [3H]phosphatidylinositol ([3H]PI) incubated with membrane preparations and several different stimulatory agents. Phospholipase C activity, measured in the presence of 0.1 mM Ca2+ to stimulate the enzyme, was not different in bipolar and control samples. G proteins coupled to phospholipase C were concentration-dependently activated by guanosine 5'-O-(3-thiotriphosphate) (GTP gamma S) and by NaF. GTP gamma S-stimulated [3H]Pl hydrolysis was markedly lower (50%) at all tested concentrations (0.3-10 microM GTP gamma S) in occipital cortical membranes from bipolar compared with control subjects. Responses to GTP gamma S in temporal and frontal cortical membranes were similar in bipolars and controls, as were responses to NaF in all three regions. Brain lithium concentrations correlated directly with GTP gamma S-stimulated [3H]Pl hydrolysis in bipolar occipital, but not temporal or frontal, cortex. Carbachol, histamine, trans-1-aminocyclopentyl-1,3-dicarboxylic acid, serotonin, and ATP each activated [3H]Pl hydrolysis above that obtained with GTP gamma S alone, and these responses were similar in bipolars and controls except for deficits in the responses to carbachol and serotonin in the occipital cortex, which were equivalent to the deficit detected with GTP gamma S alone. Thus, among the three cortical regions examined there was a selective impairment in G protein-stimulated [3H]Pl hydrolysis in occipital cortical membranes from bipolar compared with control subjects. These results directly demonstrate decreased activity of the phosphoinositide signal transduction system in specific brain regions in bipolar affective disorder.

PMID: 8632163 [PubMed - indexed for MEDLINE]