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PARKINSONS AND CHEMICAL EXPOSURE

ENVIRONMENTAL ISSUES

Parkinson's disease may be, in part, due to environmental issues, according to an article, ["Environmental Risk Factors in Parkinson's Disease", Russell, Tanya, Food of Chemical Toxicology, 1992;30(4):343-348.]. Parkinson-like symptoms can be caused by exposure to chemicals like phenothiazines and butyrophenones. In the early 1980's drug abusers who actually injected themselves with the chemical 1-methyl-4-phenyl-1,2,3 6-tetrahydropyridine (MPTP) developed, within a few days, a severe motor disorder resembling the advanced stages of Parkinson's disease. This drug has been used as a model in studying Parkinson's disease. The compound paraquat has been studied also since it has a similar structure to MPP+. There is some anecdotal evidence of pesticide-induced Parkinson's disease, as well as a significant correlation in some cases between rural residents and early onset Parkinson's disease, and between childhood well water drinking and early onset Parkinson's disease, Although the latter are still debatable. In another study there was some relationship between working in a paper mill or an orchard and the correlation with Parkinson's disease. The investigators suggest that the industrial chemicals used in the paper mills and orchards might be related to the cause and development of the disease.

According to an article, [*"Oxidative Stress in Parkinson's Disease," Jenner P, Ann Neurol, 2003;53(Suppl 3):S26-S38.*], oxidative stress may be a factor in Parkinson's disease. There is convincing evidence that nitric oxide reacts with superoxide to produce peroxynitrite and ultimately hydroxyl radical, which can lead to dopamine cell degeneration in Parkinson's disease. Oxidative stress is associated with mitochondrial dysfunction, excitotoxicity, nitric oxide toxicity and inflammation. Oxidative stress can impair ubiquitination and degrade proteins directly. Products of oxidative stress can damage the 26S proteasome. Impairment of proteasomal function leads to free radical generation and oxidative stress. Oxidative stress and the release of products of oxidative stress are a part of dopaminergic cell death. There is little doubt that oxidative stress leads to an increase in oxidative damage in the substantia nigra. Oxidative stress can be an initiator and a component of dopaminergic cell degeneration in Parkinson's disease.

THYROID, THE BRAIN AND THE ENVIRONMENT

An article in *Environmental Health Perspective* (June 2000;108(Suppl 3):433-438) reviewed the importance of the thyroid for brain function and the effect environmental chemicals have on both the thyroid and the nervous system. Proper thyroid function is especially important for brain development in the fetus and during the first two months after birth.

Animal studies have shown that exposure to PCBs and dioxins create abnormal neurologic function and impaired thyroid function. Chemical exposure can enlarge the thyroid, and decrease T4 levels. Many environmental toxins mimic thyroid hormones and bind to proteins used to transport thyroid hormone, competing with thyroid hormone and altering function.

Hypothyroidism can cause high cholesterol and heart disease. Now, research appearing in the *Annals of Internal Medicine* (2000; 132(4):270-8) shows that subclinical hypothyroidism and thyroid autoimmunity can also increase the risk of heart disease. Could it be that your patient who is on cholesterol lowering medication should actually be getting thyroid support? Hypothyroidism can be responsible for miscarriages. If an expectant mother has hypothyroidism and doesn't miscarry, her baby will have a lower IQ than if her thyroid was normal [According to a study published in the New England Journal of Medicine (1999;341:549-555, 601-602),]. Problems losing weight, dry skin and immune system problems may have the thyroid dysfunction at their root.

An article, ["Thyroidal Dysfunction and Environmental Chemicals-Potential Impact on Brain Development," Porterfield SP, Environ Health Perspect, June 2000;108(Suppl 3):433-438] discussed the affect PCBs and dioxin have on the thyroid. Polychlorinated biphenyls (PCBs) and dibenzo-p-dioxins (dioxins, 2,3,7,8-tetrachlorodibenzo-p-dioxin) have been shown to have neurotoxic effects and to alter thyroid function during critical periods of thyroid hormone-dependent brain development. Thyroid hormones are essential for normal brain development during a critical period beginning in utero and extending through the first 2 years postpartum. Thyroid hormones regulate neuronal proliferation, migration and differentiation in specific areas of the brain during definitive time periods. Thyroid hormones are important for normal cytoskeletal assembly and stability. The cytoskeletal system is critically

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important for migration and neuronal outgrowth. Thyroid hormones regulate the development of cholinergic and dopaminergic systems which serve the cerebral cortex and hippocampus. In animal models of exposure to environmental organohalogens, such as PCBs and dioxins, abnormal thyroid function and neurologic impairment was noted. In animal models, most exposures to these chemicals result in thyroid enlargement and reduced serum T4 levels with normal T3 levels. Some of these xenobiotic compounds resemble natural thyroid hormones and have high affinity to thyroid hormone-binding proteins, such as transthyretin. These toxicants may alter thyroid hormone synthesis and secretion, by either acting directly on the thyroid gland or by acting on the pituitary or hypothalamic control of TSH secretion. Additionally, these toxicants could compete with thyroid hormone for serum-binding proteins, potentially impairing tissue hormone delivery. They could also compete with thyroid hormones for membrane carrier systems and inhibit tissue uptake of the hormones. Thyroid toxicants could either be agonists or antagonists for thyroid hormone receptor binding. Lastly, these toxicants could alter the production of proteins, which act as coactivators or corepressors that regulate transcription of thyroid hormone-regulated genes.

CHEMICAL EXPOSURE AND HEALTH PROBLEMS

The incidence of diabetes and obesity may be increased by toxins in the environment. Research appearing in the journal *Diabetes Care* (30:622-628, 2007) indicates that that OC pesticides and nondioxin-like PCBs may be associated with type 2 diabetes risk by increasing insulin resistance, and POPs may interact with obesity to increase the risk of type 2 diabetes. Researchers at the University of New Hampshire found a connection between obesity and environmental pollution.

Research appearing in *The Journal of Clinical Endocrinology & Metabolism* (Vol. 92, No. 1 196-202) observed declining levels of testosterone that does not seem to be attributed to health or lifestyle and the authors concluded that "These results indicate that recent years have seen a substantial, and as yet unrecognized, age-independent population-level decrease in Testosterone in American men, potentially attributable to birth cohort differences or to health or environmental effects not captured in observed data." One possible explanation of the lower testosterone levels is chemicals in the environment. Studies have that found environmental impacts on testosterone levels. For example, testosterone levels were lower in US Air Force veterans exposed to dioxins (*Environmental Health Perspectives,* Nov. 2006, vol. 114, #11). Testosterone levels were also lower in men exposed to phthalates at work (*Environmental Health Perspectives*, Nov. 2006, vol. 114, #11). Infertility in women has also been linked to chemical exposure. Exposure to BPA (biphenol-A) is linked to prostate cancer in men.

Individually we know that many of these chemicals are dangerous. Very little research is done on combinations of chemicals. An article appearing in the May 10, 2006 issue of *Scientific American* does look into the dangers some of these chemicals have in combination. Scientists at the University of California, Berkeley, have tested four herbicides, two fungicides and three insecticides commonly used in American cornfields. Low concentrations (0.1 ppb) of the chemicals did not have much effect on developing tadpoles. When the tadpoles were exposed to all nine chemicals they developed endemic infection. The survivors ended up smaller than their counterparts raised in clean water--despite taking longer to mature into adults. So individually the chemicals did no harm, but in combination they were deadly. When you consider that there are about 100,000 different chemicals that we are exposed to, it may make you wonder what the long-term health effects are.

It is easier to build strong children than to repair broken men.

Frederick Douglass

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CHEMICALS AND DRUG-RESISTANT BACTERIA

A recent report, ["Dental Fillings, Lawn Chemicals May Contribute to Drug Resistance", Rosenthal, Marie, Infectious Disease News, July 1993;4/ *Antimicrobial Agents Chemotherapy*, 1993;37:825-34] noted spraying lawns with chemical fungicides and bacteriocides may contribute to the resistance of some bacteria to antibiotics. Lawns treated with chemical fertilizers have a higher incidence of multiple-resistant bacteria as compared with untreated lawns. Treated lawns are reservoirs for multiple drug resistant bacteria and can reduce the quality and quantity of agricultural products, increase the genetic pool of infectious drug resistance and possibly cause problems in the treatment of life-threatening bacterial infections. In a study published in Antimicrobial Agents and Chemotherapy, researchers cited amalgam dental fillings as a factor in the development of antibiotic-resistant bacteria in the oral and intestinal flora. These researchers examined the oral and intestinal flora of 6 adult monkeys prior to and after filling their teeth with amalgam fillings and found mercury resistance to several bacterial including oral streptococci, Enterobacteriaceae and enterococci. This is the first report of mercury resistance to streptococci. The bacteria were also resistant to 1 or more of the following antibiotics: ampicillin, tetracycline, streptomycin, kanamycin and chloramphenicol. When the amalgam fillings were replaced with glass ionomer fillings, resistance was found for 5 weeks following the replacement. The researchers reported a high correlation between the incidence of mercury resistance with resistance to multiple antibiotics. Mercury released from dental fillings enriched mercury and antibiotic resistant plasmids in normal bacterial flora.