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DOES MERCURY AND DENTAL AMALGAM AFFECT THE IMMUNE SYSTEM?

Sam Ziff

There is a growing collection of scientific research indicating the answer to the title question is an unqualified yes. Some of the following papers are considered pertinent.

Dr. Koller in a 1973 paper titled "Immunosuppression produced by lead, cadmium, and mercury" describes an experiment utilizing rabbits to determine the effect that lead, cadmium and mercury had on the humoral antibody response when exposed to a viral agent. Dr. Koller concludes with the statement "The significant aspect of the present study is that chronic exposure to lead, cadmium or mercury produces immunosuppression to a viral agent."

In a follow-up study published in 1975 Dr. Koller describes the results of an experiment where mice were fed methylmercury chloride at doses of 1 or 10 ppm for 84 days. The mice fed methylmercury chloride had significantly higher mortality rates when inoculated with encephalomyocarditis virus (EMCV) than did nonmethlymercury-treated mice. One ppm methlymercury produced a significant increase in the mortality rate of mice inoculated with EMCV. Dr. Koller concludes "The present study illustrated that prolonged exposure to subclinical concentrations (1 ppm) of methylmercury increased susceptibility of a host to a nononcogenic virus, but did not affect an oncogenic virus. Results emphasize that methylmercury not only poses a threat to public health as a toxic agent, but at subclinical concentrations it may augment infectious agents to cause disease."

Some of you, at this point, probably think that discussing methylmercury and dentistry is inappropriate because dentistry is only concerned with mercury vapor. That thought process would have been valid a few years ago but I am afraid it is slightly outdated today.

Today we have documented scientific evidence that mercury is released from amalgams under various conditions: (1) In certain aqueous solutions (Brune D., 1981); (2) Through migration from restorations into the enamel, dentin, pulp tissue and adjacent gingival tissue where it may accumulate or migrate elsewhere in the body (Soremark, 1962); (3) The normal act of chewing can cause a 15 fold increase in the release of mercury vapor (Gay et al., 1979). We also know now that microorganisms from various sources including the human intestine, are capable of methylating mercuric ions both under aerobic and anaerobic conditions.

In April 1983 Heintze and his associates presented evidence that methylmercury could be formed, in vitro, by common oral streptococci. The experiment utilized mercuric chloride and pulverized dental amalgam in distilled water, as the sources of mercury. Using the oral bacteria *Streptococcus mitior*, *S. mutans* and *S. sanguis*, the researchers found methylmercury in the bacterial cells of all three tested strains. The results indicated that organic mercury compounds may be formed in the oral cavity.

In a study published in 1981, D.A. Lawrence investigated the ability of heavy metals to modulate in vitro primary humoral immune responses. The heavy metals which significantly inhibited antibody production included (relative order of inhibitory activity): Hg^{2+} > Cu^{2+} > Cd^{2+} > Co^{2+} ; Cr^{3+} ; Mn^{2+} ; Zn^{2+} ; Sn^{2+} .

The immunosuppressive activities of the heavy metals usually correlated with their toxicity and their inhibition of lymphocyte proliferation. Hg^{2+} inhibited the mixed lymphocyte culture response, was extremely toxic and inhibited all in vitro lymphocyte proliferation. The study concluded with the following statement "Heavy metal effects on the various components of the immune system need to be assessed so that their potential role in the development of immunologically related diseases can be determined."

P. Druet, et al, 1982, discuss some interesting studies that had recently been completed in France. The researchers were investigating immune dysregulation and auto-immunity induced by toxic agents. The mechanisms of how different drugs can induce auto-immune disorders including immunologically mediated nephropathies in humans is poorly understood. The accepted theory was that drugs act as haptens or by modifying auto-antigens. However, the mechanism of action to support the theory has not been established. Recent experiments with rats utilizing mercury to induce auto-immune disease suggest that some of these toxic agents modify the fine balance between subpopulations of lymphocytes leading to immune dysregulation and to production of auto-antibodies.

Some of the data from the experiments with the rats strongly suggest that mercury modifies immune homeostasis resulting in immune dysregulation and the production of auto antibodies some of which are of pathogenic significance. It is postulated that this dysregulation may result from the interaction of Hg with the cell membrane of lymphocytes. Other possibilities are: (1) that a modification of B-cell surface determinants could lead to nonspecific B cell stimulation through an enhancement of T helper function and (2) that Hg decreases suppressor T cell functions.

Sporadic cases of the nephrotic syndrome (massive proteinuria) following exposure to mercury imply a direct pathogenesis than occurs in acute tubular necrosis. Renal biopsies of such patients show a variety of histologic types of glomerulonephritis. "Glomerular changes associated with mercury occur independently of acute proximal tubule damage and in the absence of neurologic manifestations of mercurialism." (Wedeen, 1983)

Wedeen also reproduced the results reported by Druet, et al, utilizing Lewis/Brown-Norway F-1 hybrid rats. "These animals showed granular IgG deposits in glomeruli and proteinuria 2 to 6 months after injection." (mercuric chloride 0.1 mg/100 gm intra-peritoneally three times weekly for four weeks) "The biphasic HgCl_2 -induced immune disease in rat kidneys appears to be under direct effects on B and/or T suppressor cells rather than a result of modification of autologous antigens."

Wedeen concludes his findings with this statement: "Genetically controlled immunologic variability in rats suggest that immunologically mediated glomerulonephritis may occur as a genetically determined "idio-