

- [5] Lazarow A, Liambies J, Tausch AJ. Protection against diabetes with nicotinamide. *J Lab Clin Med* 1950;36: 249–58.
- [6] (a) El Ridi MS, Abdel Kader MM, Habib A. The role of the liver and kidney in the metabolism of nicotinamide in pellagrins and nonpellagrins. *Acta Physiol Acad Sci Hung* 1960;17:429–41;
- (b) Pasquariello G. Tryptophan-nicotinic acid pathway in diabetes mellitus. *Acta Vitaminol* 1964;18(5): 225–34;
- (c) Matsuura T, Miyata I, Kurata H. Nicotinamide coenzyme levels in the livers of alloxan-diabetic rats. *Bitamin* 1968;37:423–7;

(d) Thomas MC, Tikellis CB, Burns WC. Reduced tubular cation transport in diabetes: prevented by ACE inhibition. *Kidney Internat* 2003;63(6):2152–61.

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Thimerosal, micromercurialism and chronic fatigue syndrome

Dear Sir

“The heightened susceptibility of the developing nervous system to mercury is well established, but little is known about factors that modulate sensitivity to repeated low-dose exposures delivered i.m. (and) or restricted to postnatal life” [1], therefore, the following hypotheses are presented in hopes of generating significant thought and discussion supported by sound scientific method.

Chronic fatigue syndrome (CFS) and related illnesses are potentially caused by subacute mercury poisoning or “micromercurialism” [2]. It is also hypothesized that symptoms identified in both CFS and “micromercurialism” are present in a subgroup of adults of Anglo-descent who have received thimerosal-containing vaccines. Specifically, Westphel et al. [3] have reported that a “Thimerosal allergic” exhibits “homozygous gene deletions of the glutathione-S-transferases M1 and T1”. Further as recently published, “immune response genes may be linked to heritable factors mediating toxin-induced CNS damage” [1].

If subacute mercury poisoning or “micromercurialism” is the cause of CFS, FMS and GWS then a drug compound containing one or more sulfhydryl (–SH) groups, known for their exceptional ability to chelate mercury, should promote improvement in the symptoms of patients suffering from the above-mentioned illnesses. The recovery rate reported for CFS patients is only 12%.

In an ongoing study involving the administration of glutathione, a tripeptide necessary for the body’s general wellness, CFS ($n = 478$) patients were treated with glutathione and a glutathione · ATP (GSH · ATP) com-

plex. Results of the study showed that patients receiving 200 mg GSH · ATP weekly exhibited an increase in natural killer (NK) cell count and relief from the chronic low grade temperature usually experienced by CFS patients. Patients receiving 300 mg GSH · ATP weekly reported an 84% improvement in symptoms [4].

Glutathione is required to transport heavy metals out of cells and/or across the blood–brain barrier into the extracellular fluid. The chelate, however, undergoes multiple hydrolysis steps as it makes its way through the liver, gall bladder and into the bowel. The hydrolyzed chelate is then reabsorbed as a L-cysteine complex. A steady-state enterohepatic circulation mechanism explains the aforementioned study results and further suggests that a secondary chelant is needed to shift the steady-state mechanism toward excretion. This hypothesis is supported in part by a recent paper presented at the seventh international AACFS conference [5].

A study of heritable factors in CFS patients is planned as well as a controlled study involving sulfhydryl-based chelants in concert with GSH · ATP therapy. The results will be presented early next year.

Sincerely,
Richard F. Miller, Sc.D.

References

- [1] Hornig M, Chian D, Lipkin WI. Neurotoxic effects of postnatal thimerosal are mouse strain dependent. *Mol Psychiatry* 2004;(June 8):1–13.
- [2] Clarkson TW. The three faces of mercury. *Environ Health Persp* 2002;110(Suppl 1):11–23.
- [3] Westphel GA, Schnuch A, Sculz TG, Reich K, Aberer W, Breach J et al. Homozygous gene deletions of the glutathione-S-