The Methionine Cycle and Glutathione

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Mercury has a high affinity for thiol (sulfhydryl (-SH)) groups. The thiol-containing antioxidant, glutathione (GSH), provides the major intracellular defense against mercury-induced neurotoxicity. (1,2,3,4)

Glutathione plays a critical role in a large number of cell reactions, particularly protecting cells and the energy producing units in the cell known as mitochondria. The reactions mitochondria use to utilize oxygen and produce the energy containing molecules of ATP also produce free radicals. The presence of toxins such as heavy metals leads to an increase in the production of free radicals which can damage the mitochondria and surrounding cell structures.

Normal production of glutathione depends on the function of a biochemical cycle called the remethylation of methionine. When this cycle is compromised, the production of the many products associated with the cycle slow down.

The importance of the methionine cycle has been shown in research on autistic children. (5) Several biological markers pointed out that interruption of this cycle leads to decreased production of glutathione. It appears that decreased availability of methylcobalamin is part of this defect as injections of methylcobalamin help restore the methionine cycle in the situation. In a twist of biochemical irony, glutathione appears to be needed to maintain the production of methylcobalamin and the function of the methionine cycle.

The methionine cycle produces the biochemicals that produce critical components of the methylation reaction. Methylation is involved with a wide range of functions including protein production, DNA regulation and neurotransmitter production. Methylation is required for the production of melatonin.

Other articles have shown that a critical enzyme in this cycle, methionine synthetase, is inhibited by the

presence of heavy metals such as mercury, lead and other toxins.(6)

(1) Neurotoxicology.2005 Jan;26(1):1-8.
(2) Free Radic Biol Med. 1995 Feb;18(2):321-36.
(3) Curr Top Med Chem. 2001 Dec;1(6):529-39.
(4) Curr Med Chem. 2005;12(10):1161-208.
(5) James SJ, et al Metabolic biomarkers of increased oxidative stress and impaired methylation capacity in children with autism Am J Clin Nutr 2004;80:1611–7.
(6) Mol Psychiatry. 2004 Apr;9(4):358-70.