

# Protecting Yourself from Mercury Toxicity

*When Jove sent blessings to all men that are  
And Mercury conveyed them in a jar,  
That friend of tricksters introduced by stealth  
Disease for the apothecary's health,  
Whose gratitude impelled him to proclaim:  
"My deadliest drug shall bear my patron's name!"*

Ambrose Bierce (1842 - 1914)  
The Devil's Dictionary

Mercury, used by many cultures for thousands of years, is the one of most toxic non-radioactive heavy metals known. A systemic assassin so poisonous it can injure or kill any cell with which it comes into contact, its vapor passes easily through the skin and the oral mucosa where the metal is quickly absorbed into the blood and transported to every cell in the body.

Dissolved mercury penetrates the blood-brain barrier<sup>1</sup> and accumulates in the motor regions of the brain and central nervous system (CNS)<sup>2,3,4</sup> where the metal damages nerve cells<sup>5</sup> and generates high levels of reactive oxygen species (free radicals).<sup>6,7</sup> A potent neurotoxin that inhibits the production of neurotransmitters,<sup>8,9</sup> mercury creates serious imbalances in the development of the brain.<sup>10</sup> The poison also disrupts the endocrine system<sup>11,12,13</sup> and causes serious kidney damage by selectively destroying cells in the main filtering structures of the kidney).<sup>14</sup> Mercury is also a powerful reproductive and developmental toxin that can impair the transport of oxygen and nutrients to the fetus.<sup>15,16</sup> Exposure to maternal mercury in the developing fetus and nursing child can lead to severe neurological and learning impairments, including autism.<sup>17,18</sup> The effect of mercury toxicity on children can be extensive and involve the entire cortex and frontal region of the brain, resulting in a reduction in brain mass and functionality.<sup>19</sup> Chronic exposure in adults has been shown to cause cardiovascular disease and increase the risk of heart attack.<sup>20,21</sup> It also increases the prevalence of opportunistic bacterial, viral, and fungal infections. Damage to the immune system from mercury toxicity results in several autoimmune disorders, leading some researchers to suggest that mercury toxicity should be classified as an autoimmune disease.<sup>22,23,24</sup>

## Sources of Exposure

Mercury is released into the environment by both natural and man-made activities. Volcanic activity, degassing of the earth's crust, and evaporation from bodies of water are the major natural sources of mercury release. Industrial emissions, particularly from coal-fired power plants, add considerably to this burden with the total global atmospheric release of mercury exceeding 3,300 tons per year (1990). The mining of mercury worldwide releases thousands of additional tons of the metal each year through atmospheric discharge and the dumping of mine tailings.<sup>25</sup> Other major sources of mercury include the combustion of hydrocarbon fuels, smelting of metal ores, refining of gold and production of cement, refuse incineration, and the industrial applications of metals.

In all, there are over 3000 industrial uses for mercury and its various compounds. From pulp and paper to floor waxes and vaccine preservatives, use of this powerful environmental poison is widespread. Ironically, despite grave concern about its toxicity, the greatest source of human exposure to mercury is from mercury-amalgam fillings that are still used for the vast majority of tooth restorations. Each year in the United States, over 100 million mercury amalgams are placed into the mouths of Americans—without a scintilla of evidence of safety.

## Relative Human Exposures

The regulatory agencies that monitor occupational exposures to toxins know that mercury, in any form, is extremely toxic. The World Health Organization (WHO) recognizes a time-weighted average for occupational exposure to mercury vapor of 25  $\mu\text{g}/\text{m}^3$  per eight-hour shift. The current permissible limit of exposure set by the Occupational Safety and Health Administration (OSHA) is 50  $\mu\text{g}/\text{m}^3$  of air, based on an eight-hour work-related exposure for a 40-hour week. OSHA has also set a ceiling of 100  $\mu\text{g}/\text{m}^3$  of air that *cannot* be exceeded. The limit of exposure established by the National Institute for Occupational Safety and Health (NIOSH) is 50 ( $\mu\text{g}/\text{m}^3$ ) of air for a ten-hour workday and a 40-hour workweek,<sup>26</sup> with an additional stipulation: at no time shall a worker's exposure to mercury vapor exceed 100  $\mu\text{g}/\text{m}^3$ . Of all the regulatory bodies, the Agency for Toxic Substances and Disease Registry (ATSDR) takes the most prudent approach to mercury exposure. The agency has established a level for chronic exposure of only 0.2  $\mu\text{g}/\text{m}^3$  of air over a 24 hour period. According to the agency, this is the upper limit to which a person can be *continuously* exposed without exhibiting any observable effects.<sup>27</sup>

In 1990, the World Health Organization (WHO) concluded that mercury released from amalgam fillings was the primary source of human exposure to the toxin. Dr. Charles

Williamson of the Toxic Studies Institute, Boca Raton, Florida, contends that the greatest majority of the body burden of mercury—up to 87 percent—comes from the continuous release of mercury vapor from dental amalgams.<sup>28</sup> Individual exposures to mercury amongst those who have mercury-amalgam fillings range from 3.8 to 21 micrograms ( $\mu\text{g}$ ) per day—up to 7 times the intake from fish or other food sources and up to 500 times the level of intake from other environmental sources. Considering the many avenues for environmental and occupational exposure to mercury, people with amalgam fillings are at significant risk for chronic mercury poisoning.

<b><i>Estimated Daily Intake &amp; Retention of Mercury</i></b>			
<b>Source</b>	<b>Type</b>	<b>Av. Daily Intake <math>\mu\text{g}/\text{day}</math></b>	<b>Av. Daily Retention <math>\mu\text{g}/\text{day}</math></b>
Amalgam fillings	Elemental mercury	3.8 – 21	3 – 17
Fish	Methyl/inorganic mercury	3.0	2.3
Other food	Inorganic mercury	3.6	0.25
Air	All types	0.04	0.031
Water	All types	0.05	0.0035
<b>TOTAL</b>		<b>10.6-27.7 <math>\mu\text{g}/\text{day}</math></b>	<b>5.58-19.6 <math>\mu\text{g}/\text{day}</math></b>

Adapted from, *Environmental Health Criteria 101: Methylmercury (WHO, 1990)*<sup>29</sup>

And *A Mouth Full of Poison* (Wentz, 2004)<sup>30</sup>

As long as amalgam fillings are in your mouth they are slowly and inexorably releasing mercury vapor. Intra-oral mercury levels in dental patients have been recorded that are 30 to 100 times greater than the maximum allowable limit for air quality set by the U.S. Environmental Protection Agency<sup>31,32,33,34</sup> and 10 to 50 times greater than that considered safe by the U.S. ATSDR and Health Canada.<sup>35,36</sup> By NIOSH standards, at no time should a worker be exposed to more than 100  $\mu\text{g}/\text{m}^3$  of mercury vapor. However, work by Malmström in the late 1980s demonstrated that the amount of mercury released into the oral cavity by amalgam fillings can range from 36 to 4200  $\mu\text{g}/\text{m}^3$ , depending on the degree of stimulation.<sup>37</sup> According to Mamström's data, anyone with a mouthful of metal fillings—and that includes a significant portion of the North American population—exceeds the “safe” levels of exposure solely from what is released from their amalgams.

Unfortunately, when oral mercury release exceeds safe levels of exposure, people with amalgam fillings can't simply leave the contaminated area. The *only* way to reduce your exposure to mercury vapor from amalgams is to have your fillings removed—then you need to deal with the mercury that has accumulated in your body.

<b><i>Stimulation of Amalgam Fillings</i></b>	
Type of Stimulation	Mercury Released ( $\mu\text{g}/\text{m}^3$ )
None	36
Chewing Food	68
Eating Sweets	70
Tooth Brushing	272
Polishing after a Dental Cleaning**	504
Wet Polishing of Filling**	597
Dry Polishing of Filling**	4295

\*\* Stimulation initiated at a dental office.

From Malmström C, Nylander M. Silver amalgam: An unstable material. *Danish Dental Journal*. Tidsskr. f. Tandlaeger. October 1989. Swedish paper translated by Mats Hansson Ph.D., in Bio-Probe Newsletter, Vol 9(1):5-6, Jan.1993.

## No Safe Level

The U.S. Centers for Disease Control acknowledge that, practically speaking, there is *no safe level* of mercury exposure.<sup>38,39,40</sup> Even the smallest amounts mercury, once inside the body, can kick-start a kaleidoscope of adverse reactions in sensitive individuals. Mercury vapor that is absorbed by the body is soon deposited in its tissues and organs, particularly in the kidneys and the brain.<sup>41,42,43,44,45</sup> Once past the blood-brain barrier, mercury becomes trapped within the central nervous system; it is here that the slow accumulation of the poison will eventually display its most profound effects.

It has been shown that the level of mercury in the blood spikes dramatically when amalgam fillings are stimulated and remains elevated during the course of chewing, declining slowly over 90 minutes following cessation of stimulation.<sup>46</sup> The transient release of mercury vapor from dental amalgams—much of which is absorbed by the oral mucosa—can exceed 30  $\mu\text{g}/\text{m}^3$  of air. This is well above the level where harmful effects in sensitive individuals have been demonstrated.

The effects of mercury release at 25 to 80 µg/m<sup>3</sup> of air are generally seen only in sensitive individuals and may include defects in psycho-motor performance, detectable tremors, fatigue, irritability, and loss of appetite. Exposure to mercury vapor exceeding 80 µg/m<sup>3</sup> of air, carries with it a high probability of developing classical neuropathological symptoms, including tremors, proteinuria (excessive protein in the urine) and erythema (excessive irritability and sensitivity to stimuli). At daily intakes of 10 to 30 µg of mercury, symptoms include adverse changes in thyroid uptake, degradation of liver and cardiovascular functions, alterations in adrenal gland activity, and depressed immunologic responses.

Several studies show a strong association between the number of amalgam fillings and the resident mercury content in the brain and kidneys. Using kidney and brain tissue from autopsies, Swedish researchers found that the more fillings you have in your mouth, the higher is the concentration of mercury in the kidneys and the occipital cortex of the brain. The mercury in the kidneys of amalgam carriers was almost 10-fold higher than the amount of mercury in amalgam-free individuals.<sup>47</sup> In a post-mortem study, dental staff who had been occupationally exposed to mercury vapor exhibited an exceedingly high level of the toxin in their pituitary glands that was more than thirty-five times the level of non-occupational controls.<sup>48</sup> Mercury in the grey and white matter of brain tissue of amalgam users that was two to three times that of amalgam-free individuals provides further substantiating evidence that mercury released from amalgams indisputably contributes to the mercury burden in the brain.<sup>49,50</sup>

### ***The Placenta and Fetus***

Even more worrisome is the exposure of pregnant and nursing women to mercury released from amalgams. Using radiometric tracing techniques, researchers have been able to trace the path of mercury released during gestation. Using animal models, researchers confirmed that within two days following placement of radioactive mercury in the teeth of pregnant ewes the poison appeared in both the maternal and fetal blood, and the amniotic fluid. Mercury accumulation in the fetal liver and pituitary gland was exceptionally high—a finding made more ominous by the discovery that the placenta appears to progressively concentrate mercury with the advance of gestation.<sup>51</sup>

These findings have been corroborated in an elegant Canadian study where the mercury excreted in maternal breast milk and urine was found to correlate with the number of amalgams and mercury vapor concentrations in the oral cavities of mothers with aged fillings.<sup>52</sup> The authors conclude that mercury originating from maternal amalgam fillings transfers across the placenta to the fetus. It also enters the breast milk ingested by the newborn and concentrates in the infant's body tissues.

The implications of these findings have great relevance for women of child-bearing years. According to the U.S. Centers for Disease Control (CDC), heavy metals such as mercury, arsenic and lead top the list of global environmental threats to children. The CDC has recently estimated that seven million women throughout the United States have so much mercury in their systems that pregnancy would pose a serious threat to the developing fetus.<sup>53</sup> According to the Toxic Element Research Foundation, the cumulative effect of mercury amalgam poisoning makes it one of the most serious health hazards facing Americans today — particularly our children.

## **Benefits of Removing your Amalgam Fillings**

Studies investigating the effects of amalgam removal confirm that a transient increase in blood levels of mercury are followed by a consequent and rapid reduction in the body burden.<sup>54</sup> In one study, blood and urinary mercury levels were reduced within twelve months of removal to 50 percent and 25 percent, respectively, of the levels prior to removal.<sup>55</sup> Similar findings have been confirmed in several other studies.<sup>56,57</sup>

Swiss dentist Paul Engel<sup>58</sup> has verified significant improvement of symptoms related to mercury toxicity following amalgam removal. In follow-up evaluations of seventy-five patients, each with one or more health challenges, 80 percent of the patients experienced abatement or complete elimination of a wide variety of symptoms, including chronic headaches and migraines, gastro-intestinal problems, neck tension, dizziness, allergies, vision disturbances, chronic back pain, psychological disorders, and joint pain.

A 1998 study, conducted at Sweden's Karolinska Institute, evaluated 12 patients who underwent amalgam removal. A decline in blood and urine mercury levels that began within days of amalgam removal showed a 40 percent decrease by sixty days. Three years later the mercury levels approached those of subjects without any dental amalgams.<sup>59</sup> The findings confirm that, subsequent to amalgam removal, the body flushes mercury from its system until body-fluid levels eventually reach those of individuals who have had no previous amalgam exposure.<sup>60</sup> These dramatic reductions in blood and urine mercury, however, do not necessarily reflect residual mercury levels in body tissues—particularly in the brain where the metal, with a biological half-life of up to 20 years, stubbornly resists removal.

The weight of scientific evidence legitimizes the case for amalgam removal. Over 1,000 peer-reviewed and government studies compiled by the Dental Amalgam Mercury Syndrome group (DAMS), a U.S.-based patient support group that provides information to mercury-toxic individuals, confirm that mercury is either a cause or major contributing factor in over 40 chronic health conditions. The findings, which document 60,000 cases of amalgam

replacement as reported by qualified health practitioners,<sup>61</sup> provide unequivocal evidence of cure or improvement in a diverse array of chronic conditions.

## **How Mercury Poisons You**

Known as the Great Masquerader, mercury is a poison whose chameleon-like affects are frustratingly unpredictable. The pervasive nature of its toxicity explains why those affected do not react in the same manner or develop the same symptoms of disease. The metal's solubility in fats confers a special affinity for those tissues and organs that have a high lipid content, including the kidneys; brain and CNS; the liver; and the glandular tissues of the pancreas, testes, ovaries and prostate. Only through understanding the mechanisms of toxicity within these target organs can we have a better understanding of the confusing kaleidoscope of symptoms associated with mercury toxicity. It also helps to clarify why patients with seemingly disparate neurological, immunological and systemic disorders (including rheumatoid arthritis, fibromyalgia, eczema, chronic fatigue syndrome (CFS), multiple sclerosis (MS), lupus, amyotrophic lateral sclerosis (ALS), thyroiditis, glomerulonephritis, and emotional/mood disorders), often improve significantly or recover completely after mercury is removed from the body.<sup>62</sup>

Mercury affects the body through three principal means: systemic, immunological, and neurological.

### ***Systemic Disruptions***

By binding with proteins on the surfaces of cell membranes, mercury disrupts the transport of materials in and out of cells. This is particularly damaging to the cells of the blood-brain barrier as it facilitates the penetration of other toxic substances into the brain and CNS,<sup>63</sup> thereby enhancing the overall toxic challenge. The poison even finds its way to the very heart of the life process, where it interferes with cellular respiration by disrupting mitochondrial production of adenosine triphosphate (ATP), the energy currency of the cell. By disabling certain enzymes controlling the respiratory process, including those of the Krebs Cycle and the cytochrome oxidase system, mercury deprives the cell of its ability to replenish vital ATP stores—a systemic interference with energy production that may explain the profound fatigue often experienced with mercury poisoning.

### **Blood and Circulation**

Mercury's ability to block the active sites of hemoglobin, the oxygen-carrying pigment of red blood cells, reduces the ability of the circulatory system to deliver oxygen to the tissues and

further compromises the body's energy demands. The poison also destroys red blood cell membranes and damages the lining of blood vessels. Its accumulation in the heart damages the heart muscle and the valves which regulate blood flow.

Consequently, it is no surprise that mercury poisoning is well associated with irregularities in blood hemoglobin, chest pains and tachycardia (abnormally rapid heart beat). Mercury levels in the heart tissues of individuals who have died from idiopathic dilated cardiomyopathy (IDCM) have been found to contain mercury levels 22,000 times those of individuals who died from other forms of heart disease.<sup>64</sup> Many of the victims were well-conditioned athletes who dropped dead during sporting events.

## **Protein Damage**

The ability of mercury to bind to the sulfhydryl groups (-SH) of proteins results in the inactivation of sulfur-containing enzymes and the production of sulfur metabolites in the body. Disruption of sulfur metabolism may, in turn, be related to the release of inflammatory cytokines, common in many degenerative processes, including Parkinson's disease (PD), ALS, lupus, rheumatoid arthritis and Alzheimer's disease (AD).<sup>65, 66, 67</sup> According to noted toxicologist, Boyd Haley, "Mercury should be considered as a causal contributor [to Alzheimer's disease] since the mercury can produce two pathological hallmarks of the disease and inhibits the same thiol-sensitive enzymes that are dramatically inhibited in the AD brain."<sup>68</sup>

## **Oxidative Stress**

Researchers at the National Center for Toxicological Research found that superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) are both impaired by exposure to mercury. A significant loss of SOD and GSH-Px antioxidant activity, and a consequent increase in free radical formation in the cerebellum and brain stem, follows exposure to mercury. The findings led the authors to conclude that oxidative stress related to mercury poisoning is a major contributor to the development of neurodegenerative disorders.<sup>69</sup> Similar findings also reveal impairment of other antioxidant enzymes in the blood caused by chronic mercury exposure.<sup>70</sup> Add to this the fact that mercury also depletes several minerals essential for proper enzyme function and it is not a stretch to see how mercury effectively antagonizes and diminishes the body's antioxidant and detoxification mechanisms.<sup>71,72</sup>

Mercury's ability to generate free radicals is prodigious, and its presence in the body also results in an increase in the production of advanced glycation end products (AGEs). These troublesome protein/sugar complexes accelerate aging and cause certain nerve cells in the brain to pump out damaging superoxide free radicals. Unquenched oxidation, in turn,

increases the formation of AGEs and creates a vicious cycle which leads to cell death. The substantia nigra, a region of the brain which manufactures the neurotransmitter dopamine, is susceptible to AGE-related oxidative damage induced by mercury's presence. Loss of dopamine production in the hippocampus, a region of the brain responsible for converting short-term memory to more permanent memory and for recalling spatial relationships, results in a profound degradation of voluntary control. Damage to the hippocampus has been shown to be the likely cause of PD.

## **Immunological Reactions**

Even at very low levels, mercury can depress the number of T lymphocytes (T cells) in the blood. One of the body's frontline defenses against infection, T cells are white blood cells that attack other cells which the body recognizes as invaders. A low T cell count, indicative of a compromised immune system, diminishes the ability of the body to meet even the slightest challenge.

When amalgam is placed in a person's mouth the number of T cells in their blood declines markedly.<sup>73</sup> Alteration of the ratio of T cells in the blood as a consequence of insertion of amalgam fillings<sup>74</sup> is now believed to play a central role in the development of several diseases related to immune suppression, including lupus, inflammatory bowel syndrome, anemia, MS and eczema.

Mercury also impairs the function of neutrophils, another type of white blood cell important in the immune response. Individuals exposed to moderate levels of mercury exhibit an impaired resistance to infections such as *Candida albicans*, a recurring yeast infection that can spread throughout the body.<sup>75</sup> *Candida*, in turn, causes a conversion of mercury into an extremely toxic organic form, methylmercury, which can cross the blood brain barrier and accumulate in the CNS. According to noted immunologist, Myron Wentz, through depression of the immune system and its promotion of *Candida* infections, mercury exposure from amalgam appears to be a major contributor to the development of chronic fatigue syndrome.<sup>76</sup>

Mercury poisoning also impairs phagocytic immune cells (B lymphocytes and macrophages), and interferes with the production of cytokines, specialized proteins that prompt the white blood cells to swing into action, thereby crippling the immune system's early warning defense against viral infection.<sup>77, 78</sup> By reducing the ability of cells to manufacture antibodies and the anti-viral protein, Interferon, mercury exposes the individual to opportunistic infections.<sup>79</sup> From common challenges, such as *Herpes simplex*, a virus which causes fever

blisters and cold sores, to *Chlamidia trachomatis* and other sexually transmitted diseases, the individual burdened with a heavy accumulation of mercury becomes an easy target.

## **Autoimmune Responses**

It has been estimated that up to 35 percent of those individuals who have amalgam fillings may be sensitized to mercury exposure. In such individuals chronic exposure can lead to autoimmune disease, a severe allergic response which causes the body to attack its own tissues.<sup>80</sup>

Glomerulonephritis, an acute inflammation of the kidneys, is an autoimmune response to mercury exposure and is, perhaps, the most insidious effect of early mercury toxicity. By attaching to the cells of the glomeruli, the microscopic filtering structures in the kidneys, mercury “fools” the body into thinking the kidney cells are foreign pathogens. The body consequently manufactures antibodies that attack and destroy the tissue—and with it the ability of the kidneys to function.

Mercury also triggers the release of Interleukin I (IL-1) and Tumor Necrosis Factor (TNF) from white blood cells. These inflammatory cytokines cause inflammation in joint tissue<sup>81</sup> and chronic stimulation can cause cartilage damage, bone destruction and contribute to cardiovascular disease. Similar autoimmune damage, induced by mercury poisoning, occurs in the protective myelin sheaths of nerve cells. Destruction of the myelin sheath leads to severe neurological damage and motor nerve dysfunction, as seen in MS.

Mercury’s facile ability to incite autoimmune responses is a consequence of the toxin’s capacity to bind with sulfur-containing enzymes and proteins, thereby changing their structure and functionality. The body, no longer recognizing the proteins, activates T cells to destroy them,<sup>82</sup> resulting in an autoimmune response. Dramatic evidence of this is provided in a 1994 Swedish study, in which mercury amalgam was implanted in the peritoneal cavities of mice. The results reveal an unsettling litany of immunosuppressive effects as well as evidence of stimulatory autoimmune responses.<sup>83</sup> Many researchers now argue that tests for mercury toxicity should become a standard practice in the diagnosis of autoimmune disease.<sup>84</sup>

## **Neurological**

Mercury is a potent neurotoxin, consequently neurological problems are among the most common and most serious effects of chronic exposure. Adverse behavioral effects of

exposure to mercury vapor include the inability to make trivial decisions, resolve doubts, resist temptations and perform intellectual tasks. Because the effects are so unpredictable, mercury poisoning can be expressed as dementia, schizophrenia, neurosis, psychosis—or simply dismissed as nervous “jitters.”

## **Inactivation of Neurotransmitters**

Acetylcholine (ACh) is an important neurotransmitter that transports nervous impulses across the synaptic gap, a tiny space separating nerve cells. Exposure to mercury elevates Ach,<sup>85,86,87</sup> which can lead to aggressive, violent and erratic behavior. Conversely, noradrenalin, an excitatory neurotransmitter produced by the adrenal glands, is depressed by exposure to the poison. This is likely a consequence of inhibition of the enzyme responsible for its manufacture. A loss of noradrenalin activity deepens depressive states and enhances mood disorders. Exposure to mercury also suppresses serotonin, another neurotransmitter associated with depression and other mood disorders. A recent study conducted at Colorado State University found that general health problems—particularly those related to mental and emotional health—were 45 percent greater in those patients with mercury amalgam fillings.<sup>88</sup> Unexplained anger, irritability, anxiety and depression subsided or disappeared within one year after subjects had their amalgams removed.

## **Excitotoxicity**

Mercury dramatically diminishes astrocyte activity in the brain,<sup>89</sup> and the loss of these ancillary nerve cells causes a toxic buildup of glutamate, an excitatory neurotransmitter.<sup>90,91</sup> Glutamate-induced toxicity causes cells in certain regions of the brain to become so over-stimulated that they undergo apoptosis, a process of cellular suicide. It appears that the excess glutamate binds with receptors on the surfaces of nerve cells, causing a rapid inflow of calcium, which is lethal to the cell. This leads to a degradation of motor nerve function similar to that observed in ALS and a gradual extinction of brain cells in the hippocampus. Loss of these cells appears to be an underlying cause of AD.<sup>92</sup> Glutamate-induced neurotoxicity and related calcium toxicity may also be principal factors in the neural degeneration observed in MS and PD.<sup>93</sup>

## **Disruption of Microtubules**

Microtubules are microskeletal-like structures that are vital to proper cell structure. Chronic mercury exposure has been shown to disrupt the production of microtubules in brain cells—an insidious effect with grave consequences for the cell.<sup>94</sup> Canadian researchers recently uncovered convincing visual evidence of mercury-induced disintegration of microtubules in brain cells. This, in turn, disrupted cell membrane structure and caused the formation of

neurofibrillary tangles, characteristic of AD.<sup>95</sup> The findings directly implicate mercury as a causative factor in the development of AD. Similar observations are reported in a Swiss study that showed nerve cells exposed to mercury increased their production of amyloid protein. This sticky protein makes up the tangled plaques found in the autopsied brains of Alzheimer's patients.<sup>96</sup> Noted toxicologist, Boyd Haley, comments: "Mercury is the toxicant behind Alzheimer's disease. It may not be the only one, but mercury's role in the development of Alzheimer's disease is clear."<sup>97</sup>

There are myriad ways for mercury to harm us, consequently is no predictable pattern to the symptoms or to the level of a person's sensitivity to the poison. There is, however, one thing of which we *can* be certain: mercury, in any form, is poisonous and if you are exposed to it for long enough you are going to be hurt.

## **Detoxification through Amalgam Removal**

For those who have a dental amalgams the best way to reduce your exposure to mercury is to have your fillings replaced with biologically "friendly" composites—and the sooner the better. Despite the claims made by the American and Canadian Dental Associations that the mercury released from amalgams does not cause harm, the scientific evidence clearly shows otherwise. As long as amalgams are in your mouth, they are leaking mercury vapor which travels immediately through the oral mucosa to the neural pathways and into the brain—an absorption route that effectively delivers more mercury to the brain than eating it.<sup>98</sup>

For those readers interested in having their amalgams removed, you are urged to contact a mercury-free dentist who can advise you on safe-removal protocols. Amalgam removal *without* the use of appropriate safety procedures will expose you to extremely high levels of mercury vapor. For those women considering pregnancy, you should have your amalgams removed at least six months prior to conception. If you are already pregnant or nursing, under no circumstances should you have your amalgams removed at this time. Doing so would expose the fetus and nursing child to dangerously high levels of maternal mercury.

Through their website at [www.iaomt.com](http://www.iaomt.com) the International Academy of Oral Medicine and Toxicology (IAOMT) provides detailed information on safe removal protocols for mercury amalgams. The academy also provides a comprehensive listing of mercury-free practitioners in Canada and the United States. Another excellent source for information on safe removal of amalgams is [mercuryfreenow.com](http://mercuryfreenow.com), a website created by immunologist Myron Wentz. The site is entirely devoted to the issue of mercury amalgam toxicity and provides information on amalgam removal and post-operative detoxification protocols.

## **Detoxification through Supplementation**

For those who have had their amalgams replaced with biological composites there is still the need to remove the mercury that has accumulated in your body tissues. For years this toxin has been slowly accumulating and, while the body will flush much of the residual mercury over time, it is best to facilitate this process through sound nutritional supplementation. This is particularly true for mercury that has accumulated in the brain, which is difficult to remove. The most effective natural means of doing so is by enhancing your diet with nutrients that can irreversibly bind the mercury and move it safely back across the blood-brain barrier and out of the body. Fortunately, many of the nutrients that help protect the body from mercury toxicity are also powerful antioxidants, so they provide additional health benefits.

### ***The Première Detoxicant***

Glutathione (GSH), a small protein composed of three amino acids: glutamic acid, cysteine and glycine, is the most potent heavy-metal detoxicant manufactured by the body. Additionally, GSH is a powerful antioxidant, which helps counter the oxidative damage wrought by mercury and other environmental toxins. It is the chemical nature of the sulfur-containing amino acid, cysteine, which accounts for the antioxidant punch of GSH.

So dependent is the body on the detoxifying power of GSH that its tissue level is a sensitive indicator of cellular health and the ability to resist toxic challenge.<sup>99,100</sup> In fact, depletion of mitochondrial GSH may be the ultimate factor that determines a cell's vulnerability to oxidative attack.<sup>101</sup>

When it comes to mercury, GSH plays three specific roles:<sup>102</sup>

- As a carrier, GSH irreversibly binds with mercury and sacrifices itself to form a conjugated complex, thereby preventing the toxin from damaging structural proteins and inactivating enzymes.
- GSH-mercury complexes, once formed, are removed from tissues and organs and eliminated from the body through the feces and urine.<sup>103</sup>
- As a powerful free radical scavenger, GSH increases the antioxidant status of the cell and defends against hydrogen peroxide and other free radical species produced by mercury.<sup>104,105</sup>

In the liver, GSH is conjugated (chemically joined) with solvents, fat-soluble pesticides, toxic chemicals, and other xenobiotics (materials foreign to the cell), such as mercury. This renders the toxin water-soluble and prepares it for excretion from the body via the kidneys and the bile. The power of glutathione in the conjugation and elimination of toxins is prodigious. As the body's major cellular detoxification mechanism, GSH conjugation accounts for up to 60 percent of all liver metabolites in the bile.<sup>106</sup> Formation of the GSH-mercury complex is, in fact, one of the few known mechanisms by which the body can flush the toxin back across the blood-brain barrier and out of brain tissue.<sup>107</sup>

Once the metal is captured by GSH it cannot be released; therefore the body must expel the good with the bad, resulting in the unavoidable loss of this important nutrient. Cellular GSH stores can only be replenished through cellular manufacture, requiring the consumption of foods rich in the precursors of GSH.

Depletion of GHS is dangerous for the cell. Inadequate GSH levels will accelerate oxidative damage, reduce immune functions,<sup>108,109</sup> increase the overall toxic challenge and reduce the body's resistance to infection.<sup>110,111</sup> Depletion of GSH can also lead to the development of several neurological disorders;<sup>112</sup> low levels have been reported in PD<sup>113</sup> and AD patients.<sup>114</sup> It is likely that mercury-induced GSH depletion plays a central role in the manifestation of these neurological diseases.

If the source of mercury contamination is not removed—such as when an individual has mercury amalgam fillings—GSH levels will slowly be degraded, and the ability of the body to remove mercury will be seriously impaired. Even if the rate of absorption of mercury stays constant, less and less of the toxin will be removed. Eventually, the slow but relentless accumulation of mercury in the body will become manifest with the development of chronic toxicity symptoms.

## ***Nutrients that Replenish GSH***

While dietary GSH is efficiently absorbed in the gut, the same may not be the case for supplementation. Oral dosing does appear to boost tissue GSH levels, albeit with great variability between individuals. Such variations raise concern about the efficacy of oral supplementation with GHS, itself. Fortunately, several common nutrients are known to replenish GHS stores as well as reduce mercury levels in the body.

Similar to GSH, vitamin C and the antioxidant  $\alpha$ -lipoic acid (ALA) can also bind to mercury to form an excretable complex, removing the metal from the body through the feces and urine.

<b><i>Nutrients involved in Glutathione Metabolism</i></b>	
<b>Nutrient</b>	<b>Effects on Glutathione Status</b>
Vitamin C	antioxidant, maintains tissue GSH levels
$\beta$ -Carotene	antioxidant, enhances GSH production
Vitamin E	antioxidant, enhances GSH production
Selenium	antioxidant mineral, GSH cofactor
N-Acetyl-Cysteine	GSH precursor; raises GSH levels
$\alpha$ -Lipoic Acid	chelator; enhances cellular and extracellular GSH
SAM-e	raises RBC and liver GSH levels
Riboflavin	facilitates GSH-Px system
Niacin	facilitates GSH-Px system
Cysteine	metabolic precursor; raises GSH levels

High dose vitamin C (ascorbic acid) is now commonly used in detoxifying the body before, during and after amalgam removal. Beyond its ability to chelate heavy metals, the vitamin has also been found to help replenish GSH levels. One double-blind study found that red blood cell GSH levels increased nearly 50 percent when subjects were given 500 mg per day of ascorbic acid.<sup>115</sup> Vitamin C appears to boost GSH levels by helping the body manufacture it.

Alpha-lipoic acid (ALA) is another nutrient important in flushing mercury from the body. ALA is capable of complexing with heavy metals previously bound to proteins and can readily penetrate the blood-brain barrier to reach the mercury trapped within the CNS.<sup>116</sup> More-over, supplementation with ALA also enhances cellular and extra-cellular levels of GSH.<sup>117</sup> In animal studies it has been shown to dramatically increase the release of inorganic mercury by stimulating the release of GSH into the bile.<sup>118</sup> ALA's ability to cross the blood-brain barrier and its high affinity for fatty tissues makes it one of the most important natural chelators of mercury and other heavy metals. While no clinical studies have yet investigated the use of ALA as a heavy metal detoxificant, its use as a safe chelating agent for removal of mercury from the body deserves further investigation.

Cysteine, the metabolic precursor that most severely limits the synthesis of glutathione, is another nutrient that has proven very effective in boosting GSH levels.<sup>119</sup> However, at high levels cysteine has been found to auto-oxidize, raising questions about its safety as a supplement.<sup>120</sup> N-acetyl cysteine (NAC), a precursor of cysteine, avoids the problem of auto-

oxidation attributed to cysteine. NAC converts easily to cysteine in the cell, providing a precursor for the manufacture GSH. NAC has been found to significantly boost GSH levels in deficient subjects.

Oral supplementation with S-adenosyl methionine (SAM-e) has also been found to be effective in raising red blood cell and liver GSH.<sup>121</sup> Moreover, the ability of the nutrient to arrest depressive states can blunt some of the neuropsychological consequences attributed to mercury toxicity.

### ***Nutrients that Detoxify***

Vitamins A, E, β-carotene, coenzyme Q<sub>10</sub> and the mineral selenium—all powerful antioxidants—help reduce mercury toxicity by quenching rampant free radical generation, which is a hallmark of the metal's presence. There is also evidence that the mineral selenium may be a unique chelator of mercury that, while not removing the mercury from the body, renders it non-toxic. Supplementation with other antioxidants, including the plant-based bioflavonoids can further boost the body's antioxidant status, replenish other antioxidants and protect cell structures from oxidative damage caused by this aggressive pro-oxidant.

Zinc is an important trace metal in mercury detoxification because mercury is able to compete with and displace zinc in many critical enzymes. Zinc also has the ability to stimulate the production of metallothioneine, another important heavy metal chelator found in the body.

### ***Nutrients that Replenish the GSH-Px Pathway***

Several nutrients also play a vital role in GSH metabolism through their participation in the glutathione peroxidase pathway, an important antioxidant pathway of the cell. Selenium is one such nutrient that is essential for the activation of the glutathione peroxidase enzyme. In one study, high dose supplementation with selenium, vitamin C and vitamin E raised glutathione peroxidase (GSH-Px) activity five-fold, conferring a marked enhancement of cellular antioxidant status.<sup>122</sup> Because chronic exposure to mercury can knock this critical enzyme system out of commission, it is reasonable that supplementation with high doses of selenium, vitamin C and vitamin E would offer significant benefits to anyone with chronic mercury exposure.

Other nutrients involved in the GSH-Px system are riboflavin (vitamin B<sub>2</sub>) and niacin (vitamin B<sub>3</sub>). Both nutrients are important for their role in the energy transfer reactions that are the driving force of this vital antioxidant enzyme pathway.

## Conclusion

In our modern world we are constantly exposed to mercury through the foods we eat, the places where we work, the products we consume—even from the air we breathe. For those with amalgam fillings—and that includes hundreds of millions worldwide—the risk of developing a mercury-related disease is considerable. Consequently, it is prudent that we take precautions against its toxic effects.

The variance in response to mercury poisoning within a population likely has much to do with the nutritional status of the individual. Mercury exerts its presence as a powerful oxidizing agent and a prodigious inhibitor of enzymes; however, to do damage mercury must first overcome the defenses of several protective antioxidants and natural chelating agents, including the body's première detoxicant, GSH.<sup>123</sup> Together, these nutrients form a formidable barrier that, while not infallible, can provide a substantive defense against this retentive poison.

Individuals with a mouthful of amalgam and those whose inadequate diets and unhealthy lifestyles place them under undue oxidative stress are the most vulnerable to the serious consequences of chronic mercury exposure. Unfortunately, most North Americans—adults and children alike—fall into this category.

On the other hand, those who take preventive action through amalgam removal can dramatically reduce their risk of contracting a mercury-related disease. Additionally, individuals who optimize their nutritional status through the wisdom of a healthy diet and nutritional supplementation will further indemnify themselves against this crafty “friend of tricksters.”

---

<sup>1</sup> Chang LW, Hartmann HA. Blood-brain barrier dysfunction in experimental mercury intoxication. *Acta Neuropathol (Berl)* 1972;21(3):179-84.

<sup>2</sup> Arvidson K. Corrosion studies of dental gold alloy in contact with amalgam, *Swed Dent J*. 1984;68:135-139,1984.

<sup>3</sup> Monnet-Tschudi F, et al. Comparison of the developmental effects of 2 mercury compounds on glial cells and neurons in the rat telencephalon. *Brain Research*. 1996;741:52-59.

<sup>4</sup> Huggins HA, Levy TE. Cerebrospinal fluid protein changes in MS after Dental amalgam removal. *Alternative Med Rev*. Aug 1998;3(4):295-300.

<sup>5</sup> Lorscheider FL et al. Mercury exposure from silver tooth fillings: emerging evidence questions a paradigm. *FASEB J*. 1995;9:504-508.

<sup>6</sup> Hussain et al. Mercuric chloride-induced reactive oxygen species and its effect on antioxidant enzymes in different regions of the rat brain. *J Environ Sci Health B*. 1997 May;32(3):395-409.

<sup>7</sup> Bulat P. Activity of Gpx and SOD in workers occupationally exposed to mercury. *Arch Occup Environ Health*. 1998 Sep;71 Suppl:S37-39.

<sup>8</sup> Albers JW, et al. Neurological abnormalities associated with remote occupational elemental mercury exposure. *Ann Neurol*. 1998;24(5):651-659.

- 
- <sup>9</sup> Soleo L, et al. Effects of low exposure to elemental mercury on short term memory. *Br J Ind Med.* 1983;40(4):413-419.
- <sup>10</sup> Ronnback L, et al. Chronic encephalopathies induced by low doses of mercury or lead. *Br J Ind Med.* 1992; 49:233-240.
- <sup>11</sup> Gerhard I, Waibel S, Daniel V, Runnenbaum B. Impact of heavy metals on hormonal and immunological factors in women with repeated miscarriages. *Hum Reprod Update.* 1998 May; 4(3):301-309.
- <sup>12</sup> Veltman C, et al. Alterations of heme, cytochrome P450, and steroid metabolism by mercury in rat adrenal gland. *Arch Biochem Biophys.* 1986; 248(2):467-478
- <sup>13</sup> Kawada J, et al. Effects of inorganic and methyl mercury on thyroidal function. *J Pharmacobiodyn.* 1980;3(3):149-159.
- <sup>14</sup> Nylander M, et al. Mercury concentrations in human brain and kidneys and exposure from amalgam fillings. *Swed Dent J.* 1987;11:179-187.
- <sup>15</sup> Lee IP. Effects of Mercury on Spermatogenesis. *J Pharmacol Exp Ther.* 1975;194(1):171-181.
- <sup>16</sup> Ogura H, Takeuchi T, Morimoto K. A comparison of chromosome aberrations and micronucleus techniques for the assessment of genotoxicity of mercury compounds in human blood lymphocytes. *Mutat Res.* 1996;Jun;340(2-3):175-182.
- <sup>17</sup> Marlowe M, Cossairt A, Moon C, Errera J, MacNeel A, Peak R, Ray J, et al. Main and interaction effects of metallic toxins on classroom behaviour. *J Abnormal Child Psychol.* 1985;13(2):185-198.
- <sup>18</sup> Moon C, Marlowe M, Stellern J, Errera J. Main and interaction effects of metallic pollutants on cognitive functioning. *J Learn Disabil.* 1985 Apr;18(4):217-21. No abstract available.  
PMID: 3989363
- <sup>19</sup> Null, G. Mercury Dental Amalgams – Analyzing the Debate. Dr. Gary Null's webpage on dental Mercury amalgams. URL: <http://www.garynull.com/documents/dental/malgam/Amalgam2.htm>. Accessed May 5, 2004.
- <sup>20</sup> Salonen JT, et al, Intake of mercury from fish and the risk of myocardial infarction and cardiovascular disease in eastern Finnish men. *Circulation.* 1995;91(3):645-55.
- <sup>21</sup> Salonen JT, Seppanen K, Lakka TA, Salonen R, Kaplan GA. Mercury accumulation and accelerated progression of carotid atherosclerosis: a population-based prospective 4-year follow-up study in men in eastern Finland. *Atherosclerosis.* 2000 Feb;148(2):265-73.
- <sup>22</sup> Abraham J, Svare C, et al. The effects of dental amalgam restorations on Blood Mercury levels. *J Dent Res.* 1984;63(1):71-73.
- <sup>23</sup> Snapp KR, Boyer DB, Peterson LC, Svare CW. The contribution of dental amalgam to mercury in blood. *J Dent Res.* 1989 May;68(5):780-5.
- <sup>24</sup> Zamm AF. Removal of dental mercury: often an effective treatment for very sensitive patients. *J Orthomolecular Med.* 1990;5(53):138-142.
- <sup>7</sup> International Programme on Chemical Safety. Environmental Health Criteria 118: Inorganic Mercury. Geneva: World Health Organization (WHO); 1991.
- 26 Ibid.
- 27 Agency for Toxic Substances and Disease Registry. Minimal Risk Levels (MRLs) for Hazardous Substances. URL: <http://www.atsdr.cdc.gov/mrls.html>. Accessed Jun19, 2004.
- <sup>28</sup> O'Brian J. Mercury Amalgam Toxicity: Your next visit to the dentist may not be as innocent as you think. *Life Extension Magazine.* May 2001.
- 29 World Health Organization. Environmental Health Criteria 101: Methylmercury. Geneva: World Health Organization (WHO); 1990. Geneva.
- <sup>30</sup> Wentz M. A Mouth Full of Poison, the Truth about Mercury Amalgam Fillings. Baha, CA: Medicis, SC; 2004:34.
- 31 United States Environmental Protection Agency. Mercury Health Effects Update. Final Report. EPA-600/8-84-019F. 1984.
- 32 Gay DD, et al. Chewing releases mercury from fillings. *Lancet.* 1979;1(8123):985-986.

- 
- 33 Svare CW, et al. The effect of dental amalgams on mercury levels in expired air. *J Dent Res*. 1981;60(9):1668-1671.
- 34 Moller B. Reaction of the human dental pulp to silver amalgam restorations.... *Swed Dent J*. 1978;2(3):93-97.
- 35 Null G. Mercury Dental Amalgams – Analyzing the Debate. Gary Null's Natural Living Web site. URL: <http://www.garynull.com> . Accessed Jun 6,2004.
- 36 Dental Amalgam Fillings the Number One Source of Mercury in People. Positive Health Magazine web page. URL: <http://www.positivehealth.com/permit/Articles/Dentist/dental.htm>. Accessed Jun 12, 2004.
- <sup>37</sup> Malmström C, et al. Silver amalgam: An unstable material. *Danish Dental Journal. Tidsskr. f. Tandlaeger*. October 1989. Swedish paper translated by Mats Hansson Ph.D., in *Bio-Probe Newsletter*, Vol 9(1):5-6, Jan.1993.
- 38 World Health Organization (WHO). Elemental Mercury and Inorganic Mercury Compounds: Human Health Aspects. URL: [http://www.who.int/pcs/cicad/full\\_text/cicad50.pdf](http://www.who.int/pcs/cicad/full_text/cicad50.pdf). Accessed June 19, 2004
- 39 Windham B. Facts about Mercury and Dental Amalgam. URL: <http://www.eatingalive.com/windham/windhamA.htm>. Accessed Apr 14, 2004.
- 40 National Institute for Occupational Safety and Health (NIOSH). A Recommended Standard for Occupational Exposure to Inorganic Mercury. Published by NTTS PB-222 223, 1973.
- <sup>41</sup> Gay DD, et al. Chewing releases mercury from fillings. *Lancet*. 1979;1(8123):985-986.
- 42 Svare CW, et al. The effect of dental amalgams on mercury levels in expired air. *J Dent Res*. 1981;60(9):1668-1671.
- 43 Abraham JE, et al. The effect of dental amalgam restorations on blood mercury levels. *J Dent Res*. 1984;63(1):71-73.
- 44 Vimy MJ, et al. Estimation of mercury body burden from dental amalgam: computer stimulation of a metabolic compartmental model. *J Dent Res*. 1986;65(12):1415-1419.
- 45 Abraham JE, et al. The effect of dental amalgam restorations on blood mercury levels. *J Dent Res*. 1984;63(1):71-73.
- 46 Vimy MJ, et al. Serial measurements of intra-oral air mercury: estimation of daily dose from dental amalgam. *J Dent Res*. 1985;64(8):1072-1075.
- 47 Nylander M, et al. Mercury concentrations in the human brain and kidneys in relation to exposure from dental amalgam fillings. *Swed Dent J*. 1987;11(5):179-187.
- 48 Nylander M, et al. Mercury accumulation in tissues from dental staff and controls in relation to exposure. *Swed Dent J*. 1989;13(6):235-243.
- 49 Eggleston DW, et al. Correlation of dental amalgam with mercury in brain tissue. *J Prosthet Dent*. 1987;58(6):704-707.
- 50 World Health Organization (WHO). Elemental Mercury and Inorganic Mercury Compounds: Human Health Aspects. URL: [http://www.who.int/pcs/cicad/full\\_text/cicad50.pdf](http://www.who.int/pcs/cicad/full_text/cicad50.pdf). Accessed June 19, 2004.
- 51 Vimy MJ, et al. Maternal-fetal distribution of mercury (203Hg) released from dental amalgam fillings. *Am J Physiol*. 1990;258(4 Pt 2):R939-R945.
- 52 Vimy MJ, et al. Mercury from maternal "silver" tooth fillings in sheep and human breast milk. A source of neonatal exposure. *Biol Trace Elem Res*. 1997;56(2):143-152.
- 53 Centers for Disease Control and Prevention (CDC). Morbidity & Mortality Weekly Report (MMWR). March 02, 2001. 50(08):140-3.
- 54 Snapp KR, et al. The contribution of dental amalgam to mercury in blood. *J Dent Res*. 1989;68(5):780-785.
- 55 Molin M, et al. Mercury, selenium, and glutathione peroxidase before and after amalgam removal in man. *Acta Odontol Scand*. 1990;48(3):189-202.
- 56 Sandborgh-Englund G, et al. Mercury in biological fluids after amalgam removal. *J Dent Res*. 1998;77(4):615-624.
- 57 Bjorkman L, et al. Mercury in saliva and feces after removal of amalgam fillings. *Toxicol Appl Pharmacol*. 1997;144(1):156-162.
- 58 Engel P.[Observations on health before and after amalgam removal] *Schweiz Monatsschr Zahnmed*. 1998;108(8):811-3. German.

- 
- 59 Sandborgh-Englund G, et al. Mercury in biological fluids after amalgam removal. *J Dent Res*. 1998 Apr;77(4):615-24.
- 60 Wentz M. A Mouth Full of Poison, the Truth about Mercury Amalgam Fillings. Baha, CA: Medicis, SC; 2004:65.
- <sup>61</sup> DAMS Inc. The Dental Amalgam Issue. *Mercury Free and Healthy* web page. URL: www.amalgam.org. Accessed July 4, 2004.
- <sup>62</sup> Wentz M. A Mouth Full of Poison, the Truth about Mercury Amalgam Fillings. Baha, CA: Medicis, SC; 2004:84.
- 63 Chang LW, et al. Blood-brain barrier dysfunction in experimental mercury intoxication. *Acta Neuropathol (Ber)* 1972;21(3):179-84.
- 64 Haley BE. *Dr. Boyd E Haley Responds to Robert M. Anderton DDS, President of the ADA*. Washington, DC. Committee on Government Reform. U.S. House of Representatives, 2001.
- 65 Wilkinson LJ, et al. Cysteine dioxygenase: modulation of expression in human cell lines by cytokines and control of sulphate production. *Toxicol In Vitro* 2002 August;16(4):481-3.
- 66 Heafield MT, et al. Plasma cysteine and sulphate levels in patients with motor neurone, Parkinson's and Alzheimer's disease. *Neurosci Lett* 1990 March 2;110(1-2):216-20.
- 67 Pean A, et al. Pathways of cysteine metabolism in MND/ALS. *J Neurol Sci* 1994 July;124 Suppl:59-61.
- 68 Haley BE. *Dr. Boyd E Haley Responds to Robert M. Anderton DDS, President of the ADA*. Washington, DC. Committee on Government Reform. U.S. House of Representatives, 2001.
- 69 Hussain S, et al. Mercuric chloride-induced reactive oxygen species and its effect on antioxidant enzymes in different regions of rat brain. *J Environ Sci Health B* 1997 May;32(3):395-409.
- 70 Perrin-Nadif R, et al. Catalase and superoxide dismutase activities as biomarkers of oxidative stress in workers exposed to mercury vapors. *J Toxicol Environ Health* 1996 June 7;48(2):107-19.
- 71 Quig D. Cysteine metabolism and metal toxicity. *Altern Med Rev* 1998 August;3(4):262-70.
- 72 Zabinski Z, et al. The activity of erythrocyte enzymes and basic indices of peripheral blood erythrocytes from workers chronically exposed to mercury vapours. *Toxicol Ind Health* 2000 February;16(2):58-64.
- 73 Shenker BJ, et al. Immunotoxic effects of mercuric compounds on human lymphocytes and monocytes. II. Alterations in cell viability. *Immunopharmacol Immunotoxicol* 1992;14(3):555-77.
- 74 Eggleston DW. Effect of dental amalgam and nickel alloys on T-lymphocytes: preliminary report. *J Prosthet Dent* 1984 May;51(5):617-23.
- 75 Perlingeiro RC, et al. Polymorphonuclear phagocytosis and killing in workers exposed to inorganic mercury. *Int J Immunopharmacol* 1994 December;16(12):1011-7.
- <sup>76</sup> Wentz M. A Mouth Full of Poison, the Truth about Mercury Amalgam Fillings. Baha, CA: Medicis, SC; 2004:66.
- 77 Christensen MM, et al. Influence of mercuric chloride on resistance to generalized infection with herpes simplex virus type 2 in mice. *Toxicology* 1996 November 15;114(1):57-66.
- 78 Omura Y, et al. Role of mercury (Hg) in resistant infections & effective treatment of Chlamydia trachomatis and Herpes family viral infections.... *Acupunct Electrother Res* 1995 August;20(3-4):195-229.
- 79 Christensen MM, et al. Influence of mercuric chloride on resistance to generalized infection with herpes simplex virus type 2 in mice. *Toxicology*. 1996 Nov 15;114(1):57-66.
- 80 Wentz M. A Mouth Full of Poison, the Truth about Mercury Amalgam Fillings. Baha, CA: Medicis, SC; 2004:18.
- 81 Windham B. Dental Amalgam Fillings Page. URL: <http://www.earthlink.net~berniew1/indexa.html>. Accessed April 14, 2004.
- 82 Shenker BJ, et al. Immunotoxic effects of mercuric compounds on human lymphocytes and monocytes. II. Alterations in cell viability. *Immunopharmacol Immunotoxicol* 1992;14(3):555-77.
- 83 Hultman P, et al. Adverse immunological effects and autoimmunity induced by dental amalgam and alloy in mice. *FASEB J* 1994 November;8(14):1183-90.

- 
- 84 Sehnert K, et al. Is Mercury Toxicity an Auto-immune Disorder? URL: <http://www.thorne.com/townsend/oct/mercury.html>. Accessed April 04, 2004.
- 85 Miszta H, et al. Effect of mercury and combined effect of mercury and dimethylsulphoxide (DMSO) on the activity of acetylcholinesterase (AchE-E.C. 3.1.1.7) of rat lymphocytes during in vitro incubation. *Folia Haematol Int Mag Klin Morphol Blutforsch* 1989;116(1):151-5.
- 86 Hastings FL, et al. Methylmercury-cholinesterase interactions in rats. *Environ Health Perspect* 1975 December;12:127-30.
- 87 Devi M, et al. Inhibition of acetylcholinesterase activity in the central nervous system of the red swamp crayfish, Procambarus clarkii, by mercury, cadmium, and lead. *Bull Environ Contam Toxicol* 1995 November;55(5):746-50.
- 88 Silberud R. Report of the International Conference on the Biocompatibility of Materials. Colorado State University, 1998.
- 89 Silberud R. Report of the International Conference on the Biocompatibility of Materials. Colorado State University, 1998.
- 90 Ronnback L, et al. Chronic encephalopathies induced by mercury or lead: aspects of underlying cellular and molecular mechanisms. *Br J Ind Med*. 1992 Apr;49(4):233-40.
- 91 Walum E, et al. Use of primary cultures and continuous cell lines to study effects on astrocytic regulatory functions. *Clin Exp Pharmacol Physiol*. 1995 Apr;22(4):284-7.
- 92 Mader S. *Inquiry into Life* – 10th edition. New York, NY: McGraw-Hill; 2003.
- 93 Clauw DJ. The pathogenesis of chronic pain and fatigue syndromes, with special reference to fibromyalgia. *Med Hypotheses*. 1995 May;44(5):369-78.
- 94 Pendergrass JC, et al. The Toxic Effects of Mercury on CNS Proteins – Similarity to Observations in Alzheimer's Disease. IAOMT Symposium paper. March, 1997
- 95 Leong CC, et al. Retrograde degeneration of neurite membrane structural integrity of nerve growth cones following in vitro exposure to mercury. *Neuroreport*. 2001;12(4):733-737.
- 96 Olivieri G, et al. Mercury induces cell cytotoxicity and oxidative stress and increases beta-amyloid secretion and tau phosphorylation in SHSY5Y neuroblastoma cells. *J Neurochem* 2000 January;74(1):231-6.
- 97 O'Brian J. Mercury Amalgam Toxicity: Your next visit to the dentist may not be as innocent as you think. *Life Extension Magazine* May 2001.
- 98 Galic N, et al. Dental amalgam mercury exposure in rats. *Biometals*. 1999 Sep;12(3):227-31
- 99 Duke RC, et al. Cell suicide in health and disease. *Sci Am* 1996 December;275(6):80-7.
- <sup>100</sup> Slater AF et al. Signalling mechanisms and oxidative stress in apoptosis. *Toxicol Lett* 1995 December;82-83:149-53
- <sup>101</sup> Kidd PM. Glutathione: Systemic Protectant Against Oxidative and Free Radical Damage. 2004. URL: <http://www.thorne.com/altmedrev/fulltext/glut.html>. Accessed February 7, 2004.
- <sup>102</sup> Wentz M. A Mouth Full of Poison, the Truth about Mercury Amalgam Fillings. Baha, CA: Medicis, SC; 2004:95-96.
- <sup>103</sup> Zalups RK. Molecular interactions with mercury in the kidney. *Pharmacol Rev*. 2000;52(1):113-143.
- <sup>104</sup> Kromidas L, et al. The protective effects of glutathione against methylmercury cytotoxicity. *Toxicol Lett*. 1990;51(1):67-80.
- <sup>105</sup> Patrick L. Mercury toxicity and antioxidants: Part I: role of glutathione and alpha-lipoic acid in the treatment of mercury toxicity. *Altern Med Rev*. 2002;7(6):456-471.
- <sup>106</sup> MacWilliam LD. Glutathione. In: *Comparative Guide to Nutritional Supplements*. Vernon, BC. Northern Dimensions Publishing. 2003: 44-47.
- <sup>107</sup> Kerper LE, et al., Ballatori N. Methylmercury efflux from brain capillary endothelial cells is modulated by intracellular glutathione but not ATP. *Toxicol Appl Pharmacol*. 1996;141(2):526-531.
- <sup>108</sup> Droege W, et al. Functions of glutathione and glutathione disulfide in immunology and immunopathology. *FASEB J*. 1994;8(14):1131-1138. .

- 
- <sup>109</sup> Fidelus RK, et al. Glutathione and lymphocyte activation: a function of ageing and auto-immune disease. *Immunology* 1987 August;61(4):503-8.
- <sup>110</sup> Anderson ME. Glutathione and glutathione delivery compounds. *Adv Pharmacol*. 1997;38: 65-78.
- <sup>111</sup> Droege W, et al. Role of cysteine and glutathione in HIV infection and cancer cachexia: therapeutic intervention with N-acetylcysteine. *Adv Pharmacol*. 1997;38: 581-600.
- <sup>112</sup> Lee YW, et al. Role of reactive oxygen species and glutathione in inorganic mercury- induced injury in human glioma cells. *Neurochem Res*. 2001;26(11):1187-1193.
- <sup>113</sup> Lohr JB, et al. Free Radical Involvement in Neuropsychiatric Illnesses. *Psychopharmacol Bull*. 1995;31(1):: 159-165. .
- <sup>114</sup> Jenner P. Oxidative damage in neurodegenerativeneurological disease. *Lancet*. 1994;344(8925): 796-798. .
- <sup>115</sup> Johnston CS, et al. Vitamin C elevates red blood cell glutathione in healthy adults. *Am J Clin Nutr*. 1993;58(1):103-105.
- <sup>116</sup> Patrick L. Mercury toxicity and antioxidants: Part I: role of glutathione and alpha-lipoic acid in the treatment of mercury toxicity. *Altern Med Rev*. 2002;7(6):456-471..
- <sup>117</sup> Han D, et al. Lipoic acid increases de novo synthesis of cellular glutathione by improving cystine utilization. *Biofactors*. 1997;6(3):321-338.
- <sup>118</sup> Gregus Z, et al. Effect of lipoic acid on biliary excretion of glutathione and metals. *Toxicol Appl Pharmacol*. 1992;114(1):88-96.
- <sup>119</sup> Tateishi N, et al. Relative contributions of sulfur atoms of dietary cysteine and methionine to rat liver glutathione and proteins. *J Biochem (Tokyo)*. 1981;90(6):1603-1610.
- <sup>120</sup> MacWilliam LD. Glutathione. In: *Comparative Guide to Nutritional Supplements*. Vernon, BC. Northern Dimensions Publishing. 2003: 44-47.
- <sup>121</sup> Lomaestro BM, et al. Glutathione in health and disease: pharmacotherapeutic issues.." *Ann Pharmacother*. 1995;29(12):1263-1273.
- <sup>122</sup> Horrobin DF. Multiple sclerosis: the rational basis for treatment with colchicine and evening primrose oil. *Med Hypotheses*. 1979;5(3):365-378.
- <sup>123</sup> MacWilliam LD. Glutathione. In: *Comparative Guide to Nutritional Supplements*. Vernon, BC. Northern Dimensions Publishing. 2003: 44-47.