

Mercury from Amalgam Fillings is a Common Cause of MS, ALS, PD,
SLE, RA, MCS, AD, etc.

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1.1 Introduction

Proper functioning of the human body and mind depends on interactions of the brain and CNS using neuronal signaling mechanisms with elaborate metabolic and enzymatic processes and respiration that occurs at the cellular level in the various organs and parts of the body, as controlled by low levels of hormones from the endocrine system. It will be shown that toxic substances, such as mercury that the body is chronically exposed to, accumulate in the brain, pituitary gland, CNS, liver, kidneys, etc. and can damage, inhibit, and cause imbalances at virtually any stage of these various processes at very low levels of exposure, which can have major neurological, immunological, and metabolic effects on an individual. Multiple Sclerosis (MS) is caused by the erosion of myelin, a substance which helps the brain send messages to the body. Metal particles entering the body can bind to this myelin. For those who are hypersensitive, this myelin-metal bond comes under attack from the immune system. This is called *autoimmunity*¹. In such cases, the progression of MS can be halted by removing the source of the metal (369, 303b, 35).

Mercury is known to be one of the most toxic substances commonly encountered and to be along with lead and arsenic the toxic substances adversely affecting the largest numbers of people (276). Dental amalgam is documented by medical studies and medical lab tests to be the *largest source of both inorganic and methyl mercury*² in most people who have several mercury amalgam fillings (599). Bacteria, yeasts, and Vitamin B12 methylate inorganic mercury to methyl mercury in the mouth and intestines (599, 510) and mercury inhibits functional methylation in the body, a necessary process (509).

¹**Internet:** “<http://www.flcv.com/autoimmD.html>”.

²**Informativo:** “Dental Amalgam Mercury Solutions”.

The main factors determining whether chronic conditions are induced by metals appear to be exposure and genetic *susceptibility*³, which determines individuals immune sensitivity and ability to detoxify metals (405). Very low levels of exposure have been found to seriously affect relatively large groups of individuals who are immune sensitive to toxic metals, or have an inability to detoxify metals due to such as deficient sulfoxidation or metallothionein function or other inhibited enzymatic processes related to detoxification or excretion of metals.

A large epidemiological study of 35,000 Americans by the National Institute of Health, the nation's principal health statistics agency, found that there was a significant correlation between having a greater than average number of dental amalgam surfaces and having the a chronic condition such as epilepsy, MS, or migraine headaches. Fewer of those with this condition have zero fillings than those of the general population while significantly more of those with the condition have 17 or more surfaces than in the general population (543). MS clusters in areas with high metals emissions from facilities such as metal smelters have been documented (184).

As far back as 1996 it was shown that the lesions produced in the myelin sheath of axons in cases of multiple sclerosis were related to excitatory receptors on the primary cells involved called oligodendroglia. The loss of myelin sheath on the nerve fibers characteristic of the disease are due to the death of these oligodendroglial cells at the site of the lesions (called plaques). Further, these studies have shown that the death of these important cells is as a result of excessive exposure to excitotoxins at the site of the lesions (576, 598). Most of these excitotoxins are secreted from microglial immune cells in the central nervous system. This not only destroys these myelin-producing cells it also breaks down the blood-brain barrier (BBB), allowing excitotoxins in the blood stream to enter the site of damage. Some common exposures that cause such proliferation of such excitotoxins resulting in MS are mercury and aspartame, with additional effects from MSG and methanol. Mercury and other toxic metals inhibit astrocyte function in the brain and CNS (119), causing increased glutamate and calcium related neurotoxicity (119, 333, 416, 496) which are factors in neural degeneration in MS and ALS. There is evidence that astrocyte damage/malfunction is a major factor in MS (544). Mercury and increased glutamate activate free radical forming processes like xanthine oxidase which produce oxygen radicals and oxidative neurological damage (142, 13). Nitric oxide related toxicity caused by peroxynitrite formed by the reaction of NO with superoxide anions, which results in nitration of tyrosine residues in neurofilaments and manganese Superoxide Dimustase (SOD) has been found to cause inhibition of the mitochondrial respiratory chain, inhibition of the glutamate transporter, and glutamate-induced neurotoxicity involved in ALS (524, 521).

It is now known the cause for the destruction of the myelin in the lesions is overactivation of the microglia in the region of the myelin (598). An enzyme that converts glutamine to glutamate called glutaminase increases tremendously, thereby greatly increasing excitotoxicity. Any dietary excitotoxin can activate the microglia, thereby greatly aggravating the injury. This includes the aspartate in aspartame and MSG which is in many processed foods. The methanol in diet drinks adds to this toxicity as well. Now, the secret to treatment appears to be calming down inflammation of the microglia.

Mercury and cadmium inhibit magnesium and zinc levels as well as inhibiting glucose transfer are other mechanisms by which mercury and toxic metals are factors in metabolic syndrome and insulin resistance/diabetes (43, 198, 338, 597). Reduced levels of magnesium and zinc are related to metabolic syndrome, insulin resistance, and brain inflammation and are protective against these conditions (595, 43).

According to neurologist Dr. RL Blaylock (598), the good news is that there are supplements and nutrients that calm the microglia-the most potent are: silymarin, curcumin and ibuprophen. Phosphatidylcholine helps re-myelinate the nerve sheaths that are damaged, as does B12, B6, B1,

³**Informative:** "Susceptibility Factors in Mercury Toxicity: Immune Reactivity, Detoxification System Function, Enzymatic Blockages, Synergistic Exposures".

vitamin D, folate, vitamin C, natural vitamin E (mixed tocopherols) and L-carnitine (576). A study demonstrated protective effects of methylcobalamin, a vitamin B12 analog, against glutamate-induced neurotoxicity (508), and similarly for iron in those who are iron deficient DHA plays a major role in repairing the myelin sheath. Vitamin D may even prevent MS, but it acts as an immune modulator, preventing further damage - the dose is 2000 IU a day. Magnesium, as magnesium malate, is needed in a dose of 500 mg 2X a day. They must avoid all excitotoxins, even natural ones in foods-such as soy, red meats, nuts, mushrooms and tomatoes. Avoid all fluoride and especially all vaccinations since these either inhibit antioxidant enzymes or triggers harmful immune reactions. It has also been found that the antibiotic minocycline powerfully shuts down the microglia. Dr. Blaylock tried this treatment on a patient who just came down with fulminant MS. He was confined to a wheelchair. He was placed on minocycline and now, just a few weeks later, he is walking.

The various neurological, immune, and metabolic related diseases discussed together here are diagnosed and labeled clinically based primarily on symptoms, along with tests for some underlying conditions found common in each disease. But each individual will be seen to have their own unique combination of neurological, endocrine, and enzymatic imbalances along with autoimmunities that result in the functional problems that lead to symptoms that are diagnosed as multiple sclerosis (MS) or Amyotrophic Lateral Sclerosis (ALS) or Alzheimer's Disease (AD), or Parkinson's Disease (PD), or Systemic Lupus Erythematosus (SLE), rheumatoid arthritis (RA), chronic fatigue syndrome (CFS), or oral lichen planus (OLP), etc.(100) However, a lot of commonality among these factors has been documented, both within specific diseases and among the various diseases discussed here. In MS, an autoimmune T-cell attack on CNS myelin sheath results in demyelinated plaques (405, etc.). Activated T-cells, plasma cells, and macrophages have been found in the demyelinated areas. ALS is a systemic motor neuron disease that affects the corticospinal and corticobulbar tracts, ventral horn motor neurons, and motor cranial nerve nuclei (405, etc.). Approximately 10 percent of ALS cases are of the familial type that has been linked to a mutation of the copper/zinc super oxide dismutase gene (Cu/Zn SOD). The majority of ALS cases are of the sporadic type. There are many toxic substances as well as some common drugs (336) that have been found to be major factors in producing the functional conditions that result in these diseases. However mercury appears to be the most commonly implicated of these, and in particular mercury from amalgam fillings - as will be documented here. For the majority of cases there are now tests to identify the various factors involved in these types of diseases; and once an individual's underlying causative factors have been identified, high success rates at cure or significant improvement are being achieved.

Toxic metals such as mercury, lead, cadmium, etc. have been documented to be neurotoxic, immunotoxic, reproductive/developmental toxins that according to U.S. Government agencies cause adverse health effects and learning disabilities to millions in the U.S. each year, especially children and the elderly (2, 125, 441, 505, 601, 600, 503). Exposure of humans and animals to toxic metals such as mercury, cadmium, lead, copper, aluminum, arsenic, chromium, manganese, etc. is widespread and in many areas increasing. The U.S. Center for Disease Control (276) ranks toxic metals as the number one environmental health threat to children. According to an EPA/ATSDR assessment, the toxic metals mercury, lead, and arsenic are the top 3 toxics having the most adverse health effects on the public based on toxicity and current exposure levels in the U.S., with cadmium, nickel and chromium also highly listed.

While there is considerable commonality to the health effects commonly caused by these toxic metals, and effects are cumulative and *synergistic*⁴ in many cases, this paper will concentrate on the health effects of elemental mercury from amalgam fillings. The reason is that the public appears to be generally unaware that considerable scientific evidence supports that mercury is the metal causing the most widespread adverse health effects to the public, and amalgam fillings have been well documented to be the number one source of exposure of mercury to most people, with exposure

⁴**Informative:** "Susceptibility Factors in Mercury Toxicity: Immune Reactivity, Detoxification System Function, Enzymatic Blockages, Synergistic Exposures".

levels often exceeding Government health guidelines and levels documented to cause adverse health effects. Much of the direct chronic exposure to toxic metals for persons with the autoimmune diseases discussed here appears to be from use of metals in dental work. The most common dental metals that have been documented to be causing widespread adverse health effects are mercury, nickel, palladium, gold, and copper. Although chronic exposure clearly is affecting a much larger population, nickel has been found to be a major factor in many cases of MS and lupus, with palladium having very similar effects to nickel. Likewise chronic exposures to manganese and copper have been implicated in some cases of Parkinson's disease. Another group of toxic substance substances with widespread exposure that have been demonstrated to generate reactive oxygen species and have positive correlations to some of the diseases discussed here are the organochlorine pesticides. Toxic metals appear to be only one of the factors involved in chronic autoimmune conditions. Pathogens such as viruses, mycoplasma, bacteria and parasites have been found to usually be present and a factor to deal with in treating those with chronic degenerative conditions and weakened immune systems such as MS (448e, 468, 470, 485, 303) and other autoimmune conditions.

1.2 Documentation of High Common Exposures and Accumulation of Mercury in the Brain and Motor Neurons

Amalgam fillings are the largest source of mercury in most people with daily exposures documented to commonly be above government health guidelines (14, 49, 79, 99, 183, 506, 500, 217). This is due to continuous vaporization of mercury from amalgam in the mouth, along with galvanic currents from mixed metals in the mouth that deposit the mercury in the gums and oral cavity (605). Due to the high daily mercury exposure and excretion into home and business sewers of those with amalgam, dental amalgam is also the largest source of the high levels of mercury found in all sewers and sewer sludge, and thus according to government studies a significant source of mercury in rivers, lakes, bays, fish, and crops (603). People also get significant exposure from vaccinations, fish, and dental office vapor (600).

When amalgam was placed into teeth of monkeys and rats, within one year mercury was found to have accumulated in the brain, trigeminal ganglia, spinal ganglia, kidneys, liver, lungs, hormone glands, and lymph glands (22, 303). People also commonly get exposures to mercury and other toxic metals such as lead, arsenic, nickel, and aluminum from food, water, and other sources (601). All of these are highly neurotoxic and are documented to cause neurological damage which can result in chronic neurological conditions over time, as well as ADHD, mood, and behavioral disorders (601, 303).

Mercury is one of the most toxic substances in existence and is known to bioaccumulate in the body of people and animals that have chronic exposure (600). Mercury exposure is cumulative and comes primarily from 4 main sources: silver (mercury) dental fillings, food (mainly fish), vaccinations, and occupational exposure. Whereas mercury exposure from fish is primarily methyl mercury and mercury from vaccinations is thimerosal (ethyl mercury), mercury from occupational exposure and dental fillings is primarily from elemental mercury vapor. Developmental and neurological conditions occur at lower levels of exposure from mercury vapor than from inorganic mercury or methyl mercury (606). Mercury in amalgam fillings, because of its relatively high vapor pressure compared to its PEL safety limit and galvanic action with other metals in the mouth, has been found to be continuously vaporized and released into the body, and has been found to be directly correlated to the number of amalgam surfaces and the largest source of mercury in the majority of people (14, 49, 183, 199, 209, 79, 99, 500), typically between 60 and 90% of the total. The level of daily exposure of those with several amalgam fillings commonly exceeds the U.S. EPA health guideline for daily mercury exposure of 0.1 $\mu\text{g}/\text{kg}$ body weight/day, and the oral mercury level commonly exceeds the mercury MRL of the U.S.ATSDR of 0.2 $\mu\text{g}/$ cubic meter of air (217, 500). When amalgam fillings are replaced, levels

of mercury in the blood, urine, and feces typically rise temporarily but decline between 60 to 85% within 6 to 9 months (79, 600.).

Mercury has been found to accumulate preferentially in the brain, major organs, hormone glands, and primary motor function related areas involved in ALS - such as the brain stem, cerebellum, rhombencephalon, dorsal root ganglia, and anterior horn motor neurons, which enervate the skeletal muscles (22, 14, 99, 163, 291, 327, 329, 442, 48, 604). Mercury, with exposure either to vapor or organic mercury tends to accumulate in the glial cells in a similar pattern, and the pattern of deposition is the same as that seen from morphological changes (327g, 287, 305). Though mercury vapor and organic mercury readily cross the blood-brain barrier, mercury has been found to be taken up into neurons of the brain and CNS without having to cross the blood-brain barrier, since mercury has been found to be taken up and transported along nerve axons as well through calcium and sodium channels and along the olfactory path (329, 288, 333, 34).

1.3 Mercury Toxicity: Summary of Neurological Effects

Mercury has been found to accumulate in the cerebellum and other brain areas, producing reactive oxygen species (ROS), including superoxide that cause damage to those parts of the brain (194, 13). Mercury was also found to cause a reduction in antioxidant function such as superoxide dismutase (SOD) and glutathione peroxidase (GPx) that tries to counter-balance the ROS (13, 56a). Mercury, with exposure either to vapor or organic mercury tends to accumulate in the glial cells in a similar pattern, and the pattern of deposition is the same as that seen from morphological changes (327g, 287a). Mercury (especially mercury vapor or organic mercury) penetrates and damages the blood brain barrier allowing penetration of the barrier by other substances that are neurotoxic (along with reduced amino acid uptake to brain) (22, 38, 85, 604, 162, 301, 311/262). Such damage to the blood brain barrier's function has been found to be a major factor in chronic neurological diseases such as MS (286, 289, 291, 302, 324, 326, 478).

Programmed cell death (apoptosis) is documented to be a major factor in degenerative neurological conditions like MS, ALS, Alzheimer's, Parkinson's, etc. Some of the factors documented to be involved in apoptosis of neurons and immune cells include inducement of the inflammatory cytokine Tumor Necrosis Factor-alpha (TNF α) (126), reactive oxygen species and oxidative stress (13, 43a, 56a, 296b, 495), reduced glutathione levels (56, 126a, 111a), liver enzyme effects and inhibition of protein kinase C and cytochrome P450 (43, 84, 260), nitric oxide and peroxynitrite toxicity (43a, 521, 524), excitotoxicity and lipid peroxidation (490, 496), excess free cysteine levels (56d, 111a, 33, 330), excess glutamate toxicity (13b, 416), excess dopamine toxicity (56d, 13a), beta-amyloid generation (462, 56a), increased calcium influx toxicity (296b, 333, 416, 432, 462c, 507) and DNA fragmentation (296, 42, 114, 142) and mitochondrial membrane dysfunction (56de, 416).

TNF α (tumor necrosis factor-alpha) is a cytokine that controls a wide range of immune cell response in mammals, including cell death (apoptosis). This process is involved in inflammatory and degenerative neurological conditions like ALS, MS, Parkinson's, rheumatoid arthritis, etc. Cell signaling mechanisms like sphingolipids are part of the control mechanism for the TNF α apoptosis mechanism (126a). Glutathione is an amino acid that is a normal cellular mechanism for controlling apoptosis. When glutathione is depleted in the brain, reactive oxidative species increased, and CNS and cell signaling mechanisms are disrupted by toxic exposures such as mercury, neuronal cell apoptosis results and neurological damage. Mercury has been shown to induce TNF α , deplete glutathione, and increase glutamate, dopamine, and calcium related toxicity, causing inflammatory effects and cellular apoptosis in neuronal and immune cells (126b, 126c). Mercury's biochemical damage at the cellular level include DNA damage, inhibition of DNA and RNA synthesis (42, 114, 142, 197, 296, 392); alteration of protein structure (33, 111, 114, 194, 252, 442); alteration of the transport and signaling functions of calcium (333, 43b, 254, 416d, 462, 507); inhibition of glucose

transport (338, 254), and of enzyme function and other essential nutrients (96, 198, 254, 263, 264, 33, 330, 331, 338, 339, 347, 441, 442); induction of free radical formation (13a, 43b, 54, 405, 424), depletion of cellular glutathione (necessary for detoxification processes) (56, 111, 126, 424), inhibition of glutathione peroxidase enzyme (13a, 442), inhibits glutamate uptake (119, 416d), induces peroxynitrite and lipid peroxidation damage (521b), causes abnormal migration of neurons in the cerebral cortex (149), immune system damage (111, 194, 226, 252, 272, 316, 325, 355); inducement of inflammatory cytokines (126, 152, 181) and autoimmunity (181, 226, 272, 314, 369, 405, 507, 100, etc.)

MS patients have been found to have much higher levels of mercury in cerebrospinal fluid compared to controls (163, 291, 35, 139). German studies including studies at German universities have found that MS patients usually have high levels of mercury body burden, with one study finding 300% higher than controls (271, 302). Most recovered after mercury detox, with some requiring additional treatment for viruses and intestinal dysbiosis. Very high levels of mercury are also found in brain memory areas such as the cerebral cortex and hippocampus of patients with diseases with memory related symptoms (158, 34, 207, etc.). Studies have found mercury related neurological effects to be indistinguishable from those of MS (207, 212, 222, 244, 271, 289, 291, 302, 183, 184, 303, 324, 326, 406).

Mercury has been shown to be a factor that can cause rheumatoid arthritis by activating localized CD4+ T-cells which trigger production of immune macrophages and immunoglobulin (Ig) producing cells in joints (405, 513, 514).

1.4 Mercury Related Neurological Damage: Mechanisms of Causality

Exposure to inorganic mercury has significant effects on blood parameters and liver function. Studies have found that in a dose dependent manner, mercury exposure causes reductions in oxygen consumption and availability, perfusion flow, biliary secretion, hepatic ATP concentration, and cytochrome P450 liver content (260), while increasing blood hemolysis products and tissue calcium content and inducing heme oxygenase, porphyria, platelet aggregation through interfering with the sodium pump.

Mercury vapor and methyl mercury penetrate and damage the blood brain barrier (311, 22, 85, 105, 162, 600/262), also facilitating other toxic substances penetration of the BBB. Damage to the blood brain barrier's function has been found to be a major factor in chronic neurological diseases discussed here. Mercury also causes high levels of oxidative stress and reactive oxygen species (ROS) (13), which have been implicated as major factors in neurological disorders including stroke, ALS (501) PD (502), Alzheimer's (503), CFS (504), Lupus (113, 234, 331, 602). Studies have found mercury related neurological effects to be indistinguishable from those of MS (163, 207, 271, 244, 289, 291, 302, 303, 184, 324, 326).

Metals like mercury bind to SH-groups (sulphydryl) in sulfur compounds like amino acids and proteins, changing the structure of the compound that it is attached to. This often results in the immune systems T-cells not recognizing them as appropriate nutrients and attacking them (181, 226, 314, 507). Such binding and autoimmune damage has been documented in the fat-rich proteins of the myelin sheaths and collagen (405), which are affected in MS. Metals by binding to SH radicals in proteins and other such groups can cause autoimmunity by modifying proteins which via T-cells activate B-cells that target the altered proteins inducing autoimmunity as well as causing aberrant MHC II expression on altered target cells (425de, 343). Studies have also found mercury and lead cause autoantibodies to neuronal proteins, neurofilaments, and myelin basic protein (MBP) (269ag, 405, 478, 515, 516). Mercury and cadmium also have been found to interfere with zinc binding to MBP (517b) which affects MS symptoms since zinc stabilizes the association of MBP with brain myelin (517a). MS has also been found to commonly be related to inflammatory activity in the

CNS such as that caused by the reactive oxygen species and cytokine generation caused by mercury and other toxic metals (405, 478, 515, 516). Antioxidants like lipoic acid which counteract such free radical activity have been found to alleviate symptoms and decrease demyelination (494, 572). A group of metal exposed MS patients with amalgam fillings were found to have lower levels of red blood cells, hemoglobin, hemocrit, thyroxine, T-cells, and CD8+ suppressor immune cells than a group of MS patients with amalgam replaced, and more exacerbations of MS than those without (102a). Immune and autoimmune mechanisms are thus seen to be a major factor in neurotoxicity of metals.

Na(+), K(+)-ATPase is a transmembrane protein that transports sodium and potassium ions across cell membranes during an activity cycle that uses the energy released by ATP hydrolysis. Mercury is documented to inhibit Na(+), K(+)-ATPase function at very low levels of exposure (288ab). Studies have found that in Ms cases there was an elevation in plasma serum digoxin and a reduction in serum magnesium and RBC membrane Na(+)-K+ ATPase activity (263). The activity of all serum free-radical scavenging enzymes, concentration of glutathione, alpha tocopherol, iron binding capacity, and ceruloplasmin decreased significantly in Ms, while the concentration of serum lipid peroxidation products and nitric oxide increased. The inhibition of Na+-K+ ATPase can contribute to increase in intracellular calcium and decrease in magnesium, which can result in 1) defective neurotransmitter transport mechanism, 2) neuronal degeneration and apoptosis, 3) mitochondrial dysfunction, 4) defective golgi body function and protein processing dysfunction. It is documented in this paper that mercury is a cause of most of these conditions seen in MS (13a, 111, 288, 442, 521b, 43, 56, 263etc.)

Autoimmunity has also been found to be a factor in chronic degenerative autoimmune conditions such as MS, ALS, etc., with genetic susceptibility a major factor in who is affected. One genetic factor in Hg induced autoimmunity is major histocompatibility complex (MHC) linked. Both immune cell type Th1 and Th2 cytokine responses are involved in autoimmunity (425c). One genetic difference found in animals and humans is cellular retention differences for metals related to the ability to excrete mercury (426). For example it has been found that individuals with genetic blood factor type APOE-4 do not excrete mercury readily and bioaccumulate mercury, resulting in susceptibility to chronic autoimmune conditions such as Alzheimer's, Parkinson's, etc. as early as age 40 (437), whereas those with type APOE-2 readily excrete mercury and are less susceptible (437, 35). Those with type APOE-3 are intermediate to the other 2 types. The incidence of autoimmune conditions has increased to the extent this is now one of the leading causes of death among women (450). Also when a condition has been initiated and exposure levels decline, autoimmune antibodies also decline in animals or humans (233, 234d, 369, 60, 118, 303, 368, 405)

Calcium plays a major role in the extreme neurotoxicity of mercury and methyl mercury. Both inhibit cellular calcium ATPase and calcium uptake by brain microsomes at very low levels of exposure (333). Protein Kinase C (PKC) regulates intracellular and extra cellular signals across neuronal membranes, and both forms of mercury inhibit PKC at micromolar levels, as well as inhibiting phorbol ester binding (43). They also block or inhibit calcium L-channel currents in the brain in an irreversible and concentration dependent manner. Metallic mercury is much more potent than methyl mercury in these actions, with 50 % inhibition in animal studies at 13 ppb (333).

A direct mechanism involving mercury's inhibition of cellular enzymatic processes by binding with the hydroxyl radical (SH) in amino acids appears to be a major part of the connection to allergic/immune reactive conditions. The binding of mercury from amalgam to the -SH groups often results in inactivation of sulfur and blocking of enzyme function, producing sulfur metabolites with extreme toxicity that the body is unable to properly detoxify (33, 114). Sulfur is essential in enzymes, hormones, nerve tissue, and red blood cells. These exist in almost every enzymatic process in the body. Blocked or inhibited sulfur oxidation at the cellular level has been found in most with many of the chronic degenerative diseases, including Parkinson's, Alzheimer's, ALS, lupus, rheumatoid arthritis, CFS, FMS, MCS, autism, etc.(33, 234, 330, 331, 501-505, 602)

Some studies of patients with major neurological or degenerative diseases have found evidence amalgam fillings may play a major role in development of conditions such as such as, MS (102, 163, 170, 184, 212, 285, 291, 302, 303, 324, 326), ALS (92, 97, 325, 501), RA (600), AD (66, 67, 158, 166, 204, 207, 221, 238, 242, 244, 258, 296, 300, 303, 503), SLE (234, 60, 405), PD (56, 84, 98, 169, 218, 248, 250, 258, 303, 502), and many other conditions (600, 303). Mercury induced lipid peroxidation has been found to be a major factor in mercury's neurotoxicity, along with leading to decreased levels of glutathione peroxidation and superoxide dismutase (SOD) (13). Only a few micrograms of mercury severely disturb cellular function (33, 56, 226).

Mercury exposure causes high levels of oxidative stress/reactive oxygen species (ROS) (13), which has been found to be a major factor in neurological disease (56, 501-505). Mercury and quinones form conjugates with thiol compounds such as glutathione and cysteine and cause depletion of glutathione, which is necessary to mitigate reactive damage. Such conjugates are found to be highest in the brain substantia nigra with similar conjugates formed with L-Dopa and dopamine in Parkinson's disease (56, 502). Mercury depletion of GSH and damage to cellular mitochondria and the increased lipid peroxidation in protein and DNA oxidation in the brain appear to be a major factor in Parkinson's disease (33). A Canadian study found those with 15 or more amalgam fillings to have more than 250% greater risk of MS than controls, and likewise higher risk for those who have had amalgam fillings more than 15 years, and another study also found higher mercury body burden in those with more fillings and increased risk of MS with more fillings (324). Another study (169) found blood and urine mercury levels to be very strongly related to Parkinson's with odds ratios of approx. 20.

Exposure to mercury results in metalloprotein compounds that have genetic effects, having both structural and catalytic effects on gene expression (114). Some of the processes affected by such metalloprotein control of genes include cellular respiration, metabolism, enzymatic processes, metal-specific homeostasis, and adrenal stress response systems. Significant physiological changes occur when metal ion concentrations exceed threshold levels. Such metalloprotein formation also appears to cause a change in antigenicity and autoimmune reactions in significant numbers of people (114, 60, 342, 405). Much mercury in saliva and the brain is also organic, the most neurotoxic form (506, 51, 220, 272), since mouth bacteria and other organisms in the body methylate inorganic mercury to organic mercury (506, 51, 254). Dental amalgam has been found to be the largest source of methyl mercury in most with mercury amalgam fillings (506, etc.).

Spatial and temporal changes in intracellular calcium concentrations are critical for controlling neurotransmitter release in neurons (432). Mercury alters calcium homeostasis and calcium levels in the brain and affects neurotransmitter release through its effects on calcium levels (270c, 333, 372, 43). Low levels of toxic metals have been found to inhibit dihydropteridine reductase, which affects the neural system function by inhibiting neurotransmitters through its effect on phenylalanine, tyrosine and tryptophan transport into neurons (257, 258). This was found to cause severe impaired amine synthesis and hypokinesia. Tetrahydro-biopterin, which is essential in production of neurotransmitters, is significantly decreased in patients with Alzheimer's, Parkinson', and MS. Such patients have abnormal inhibition of neurotransmitter production.

Mercury at extremely low levels also interferes with formation of tubulin producing neurofibrillary tangles in the brain, similar to those observed in Alzheimer's patients with high levels of mercury in the brain (207, 303). Mercury and the induced neurofibrillary tangles also appear to produce a functional zinc deficiency in the of AD sufferers (242), as well as causing reduced lithium levels which is another factor in such diseases. The low Zn levels result in deficient CuZnSuperoxide dismutase (CuZnSOD), which in turn leads to increased levels of superoxide (463). Lithium protects brain cells against excess glutamate induced excitability and calcium influx (280). Also mercury binds with cell membranes interfering with sodium and potassium enzyme functions, causing excess membrane permeability, especially in terms of the blood-brain barrier (159, 207, 311]. Less than 1ppm mercury in the blood stream can impair the blood-brain barrier. Mercury was also found to accumulate in the mitochondria and interfere with their vital functions, and to inhibit cytochrome C enzymes

which affect energy supply to the brain. Persons with extra Apo-E4 gene copies appear especially susceptible to this damage (207, 221)

Mercury blocks the immune function of magnesium and zinc (198, 427, 43, 38), whose deficiencies are known to cause significant neurological effects (461, 463, 430). The low Zn levels result in deficient CuZnSuperoxide dismutase (CuZnSOD), which in turn leads to increased levels of superoxide due to toxic metal exposure. This is in addition to mercury's effect on metallothionein and copper homeostasis as previously discussed (477). Copper is an essential trace metal which plays a fundamental role in the biochemistry of the nervous system (489, 495, 463, 464). Several chronic neurological conditions involving copper metabolic disorders are well documented like Wilson's Disease and Menkes Disease. Mutations in the copper/zinc enzyme superoxide dismutase (SOD) have been shown to be a major factor in the motor neuron degeneration in conditions like familial ALS. Exposures to toxic metals such as mercury and cadmium have been found to cause such effects, and similar effects on Cu/Zn SOD have been found to be a factor in other conditions such as autism, Alzheimer's, Parkinson's, and ALS (489, 495, 464, 469, 111, 501-504). This condition can result in zinc deficient SOD and oxidative damage involving nitric oxide, peroxynitrite, and lipid peroxidation (495, 496, 489), which have been found to affect glutamate mediated excitability and apoptosis of nerve cells and effects on mitochondria (495, 496, 119) These effects can be reduced by zinc supplementation (464, 495, 430), as well as supplementation with antioxidants and nitric oxide-suppressing agents and peroxynitrite scavengers such as Vit C, Vit E, lipoic acid, Coenzyme Q10, carnosine, ginkgo biloba, N-acetylcysteine, etc. (444, 464, 494, 495, 469, 470, 572). Some of the antioxidants such as ginkgo biloba were also found to have protective effects through increasing catalase and SOD action, while reducing lipid peroxidations (494a) Ceruloplasmin in plasma can be similarly affected by copper metabolism dysfunction, like SOD function, and is often a factor in neurodegeneration (489).

Excess zinc from products such as GSK Superpolygrip (before reformulated) can also cause demyelinating conditions with effects similar to MS, Demyelinating Syndrome, and Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) (530)

Mercury and other toxic metals inhibit astrocyte function in the brain and CNS (119, 131), causing increased glutamate and calcium related neurotoxicity (119, 152, 333, 226a, 496) which are responsible for much of the Fibromyalgia symptoms and a factor in neural degeneration in MS and ALS. There is some evidence that astrocyte damage/malfunction is the main factor in MS (544). This is also a factor in conditions such as CFS, Parkinson's, and ALS (346, 416, 496). Animal studies have confirmed that increased levels of glutamate (or aspartate, another amino acid excitatory neurotransmitter) cause increased sensitivity to pain, as well as higher body temperature - both found in CFS/Fibromyalgia. Mercury and increased glutamate activate free radical forming processes like xanthine oxidase which produce oxygen radicals and oxidative neurological damage (346, 142, 13). Medical studies and doctors treating Fibromyalgia have found that supplements which cause a decrease in glutamate or protect against its effects have a positive effect on Fibromyalgia and other chronic neurological conditions. Some that have been found to be effective include CoQ10 (444), ginkgo biloba and pycnogenol (494a), NAC (54, 494a), Vit B6, methyl cobalamine (B12), L-carnitine, choline, ginseng, vitamins C and E, nicotine, and omega 3 fatty acids (fish and flaxseed oil) (417, 495e).

1.5 Endocrine System and Metabolic Enzymatic System Impairments

Mercury has been well documented to be an endocrine system-disrupting chemical affecting hormonal processes (85, 146, 149, 199, 312, 604) and enzyme production processes (33, 111, 194) at very low levels. The pituitary gland, in which mercury has been documented to accumulate, controls many of the body's endocrine system functions and secretes hormones involved in control of most bodily

processes. The hypothalamus regulates body temperature and many metabolic processes. Such hormonal secretions are affected at levels of mercury exposure much lower than the acute toxicity effects normally tested for (146, 199). Some of the common effects of mercury on the endocrine system include inhibiting human growth hormone, causing hormonal imbalances that affect the reproductive system and body temperature regulation, and causing hormonal imbalances resulting in imbalances in metabolism of important minerals such as calcium (333, 21, 25, 35, 280). Calcium flux is inhibited in synaptic plasma membranes of the cerebellum and cerebrum cortex. A permanent increase in cytosolic calcium levels appears to be associated with various pathological conditions which result in cell death (333). All of the effects on hormonal regulation of the various bodily processes add to and reinforce the imbalances caused in the metabolic enzymatic processes.

All body functions depend on cellular enzymatic and respiratory processes that use Nutrients delivered by the blood, detoxify toxic substances, and eliminate waste products through the cellular respiratory process back through the lymph and blood to the lungs, kidneys, or liver for excretion. Proteins are converted by enzymatic processes to amino acids such as cysteine, cystine, glutamic acid, methionine, etc. for cellular metabolic processes and to organic compounds such as glutathione which is necessary to detoxify toxic substances such as mercury (13, 111, 194). Imbalances or blockages in any of several of these enzymatic processes have been documented to cause major neurological and immune damage that appears to be involved in most of the diseases being discussed here.

Mercury vapor of those with chronic exposure is continuously released into the blood stream through the lungs and distributed to cells throughout the body, where it creates metal-protein compounds and reactive oxidative species (ROS) such as superoxide, which must be detoxified. Cysteine and glutathione, which are produced and interchanged as required through enzymatic processes, are necessary for detoxification. Blockages or impairments caused by mercury or other toxic substances or processes can then result in cellular toxicity and damage to vital organs such as the brain, CNS, liver, or kidneys.

Clinical tests of patients with motor neurone disease (MND), ALS, PD, AD, SLE, and RA have found that the patients generally have damaged enzymatic processes resulting in elevated plasma cysteine to sulphate ratios, with the average being 500% higher than controls (330, 331), and in general are poor sulphur oxidizers (33, 331). High levels of free cysteine have been found to result in major neurological damage to the brain, CNS, and cellular processes (194, 330, 331). The two main enzymatic processes that down regulate cysteine to taurine, sulfates, and glutathione are cysteine dioxygenase (CDO) and gamma-glutamylcysteine synthetase (GGCS). Impairment in CDO can result in high cysteine levels, high cysteine to sulfate ratio, low taurine levels, and neurological damage (194, 330, 331). GGCS converts cysteine to glutathione, which has been demonstrated to be necessary to detoxification of toxic substances like mercury (111). If this enzymatic process is blocked, inhibited, or overloaded by chronic high toxicity levels or autoimmune reactions, there is insufficient glutathione and toxic damage occurs due to immune inability to process the metal-organic compounds and the ROS created by exposure to mercury or other toxic substances (111, 33, 60, 56). Another enzymatic process necessary for proper cellular metabolism is sulfite oxidase (SO) which is involved in conversion of toxic sulfur forms such as sulfites, sulfur dioxide (SO₂), hydrogen sulfide (H₂S), etc. to nontoxic sulfates (33). SO can be blocked or inhibited by mercury or other toxic exposures, resulting in more of these very toxic sulfur compounds. SO is commonly found to be totally blocked or inhibited in patients with MND, PD, AD, SLE, RA, etc.(330, 331). Glutathione peroxidase (GPx) is another enzymatic process in this loop that is often affected, as well as the process involved in converting Vitamin B6 through the essential coenzyme pyrodoxal 5-phosphate (P5P) in the synthesis of neurotransmitters. Impairment in this process results in brain neurotransmitter imbalances. Individual patients with any of these diseases who commonly have been shown to have high ratio of cysteine to sulfate can thus have several different individual enzymatic blockages or imbalances that result in such high ratios, and different levels of neurological, immune, and cellular damage due to high cysteine levels or low glutathione levels. Autoimmune reactions have also been

found to be commonly involved in such blockages or imbalances, particularly for those with the major diseases being considered here. This aspect will thus be further discussed.

1.6 Autoimmunity, Neurological and Immune Diseases, and Mercury

Mercury has been documented to cause autoimmune disease (45, 91, 234, 269, 270, 291, 328, 405) and many researchers have concluded that autoimmunity is a factor in the major chronic neurological diseases such as MS, ALS, PD, SLE, RA, etc. Mercury and other toxic metals also form inorganic compounds with OH, NH₂, CL, in addition to the SH radical and thus inhibits many cellular enzyme processes, coenzymes, hormones, and blood cells (405, 600). Mercury has been found to impair conversion of thyroid T₄ hormone to the active T₃ form as well as causing autoimmune thyroiditis common to such patients (369, 382). In general, immune activation from toxic metals such as mercury resulting in cytokine release and abnormalities of the hypothalamus-pituitary-adrenal (HPA) axis can cause changes in the brain, fatigue, and severe psychological symptoms (342, 369, 379-382, 385, 405, 118) such as profound fatigue, musculoskeletal pain, sleep disturbances, gastrointestinal and neurological problems as are seen in CFS, fibromyalgia, and autoimmune thyroiditis. Such hypersensitivity has been found most common in those with genetic predisposition to heavy metal sensitivity (60, 342, 369, 382, 405), such as found more frequently in patients with human lymphocyte antigens (HLA-DRA) (381-383). A significant portions of the population appear to fall in this category.

The enzymatic processes blocked by such toxic substances as mercury also result in chronic formation of metal-protein compounds (HLA antigens or antigen-presenting macrophages) that the body's immune system (T-lymphocytes) does not recognize, resulting in autoimmune reactions (114, 342, 405). The metals bind to SH-groups on proteins which can then be recognized as "foreign" and attacked by immune lymphocytes. Such has been extensively documented by studies such as the documentation of the autoimmune function test MELISA, a sophisticated immune/autoimmune test which was developed to test for such reactions (60, 405).

Very low doses and short term exposures of inorganic Hg (20-200 mug/kg) exacerbates lupus and accelerates mortality in mice. low dose Hg exposure increases the severity and prevalence of experimental autoimmune myocarditis induced by other factors. A strong significant correlation was found between occupational exposure to mercury or pesticides to lupus (SLE), with dental personal having a very high risk factor (113c). In a study of small-scale gold mining using mercury, there was a positive interaction between Hg autoimmunity and malaria. These results suggest a new model for Hg immunotoxicity, as a co-factor in autoimmune disease, increasing the risks and severity of clinical disease in the presence of other triggering events, either genetic or acquired (234f).

Autoimmune reactions to inorganic and methyl mercury have been found to be relatively independent, occurring in over 10% of controls. In the population of over 3,000 patients tested by MELISA, the following percentages tested positive for lymphocyte reactivity: nickel-34%, inorganic mercury-22%, phenyl mercury-15%, methyl mercury-8%, gold-10%, palladium-10%, cadmium-11%, silver-1%. Groups with autoimmune symptoms such as oral lichen planus, CFS, MS, autoimmune thyroiditis, etc. generally have high percentages with lymphocyte reactivity to metals (60, 342, 369, 405). Among a population of patients being tested for autoimmune problems, 94% of such patients had significant immune reactions to inorganic mercury (MELISA test, 60, 342, 369, 405) and 72% had immune reactions to low concentrations of HgCl₂ ($<0.5 \mu\text{g/ml}$). Of a population of 86 patients with CFS symptoms who had amalgam fillings replaced, 78% reported significant health improvement in a relatively short time period after replacement, and MELISA test scores had a significant reduction in lymphocyte reactivity compared to pre-replacement (369). Similar results were experienced for those with MS, lupus, and autoimmune thyroiditis (369). The MELISA test has proved successful in diagnosing and treating environmentally caused autoimmune diseases such as MS, SLE, oral lichen planus, CFS, etc. (60, 313, 342, 369, 405). A high percentage of patients subjectively diagnosed with

CNS and systemic symptoms suggestive of mercury intoxication have been found to have immune reactivity to inorganic mercury (MELISA test, 118), and likewise for MRI positive patients for brain damage. Controls without CNS problems did not have such positive correlations. Nickel, palladium, and gold have also been found to induce autoimmunity in genetically predisposed or highly exposed individuals (60, 118, 313, 314, 234, 369, 130). Tests have found a significant portion of people (over 10%) to be in this category and thus more affected by exposure to amalgam than others. Once compromised by a toxic substance that depletes the immune protectors and causes autoimmunity, the immune system is more susceptible to being sensitized to other toxic chemicals, a factor in multiple chemical sensitivity (MCS). Mercury also causes a reduction in thyroid production (50) and an accumulation in the thyroid of radiation. Among those with chronic immune system problems with related immune antibodies, the types showing the highest level of antibody reductions after amalgam removal include glomerular basal membrane, thyroglobulin, and microsomal thyroid antigens (91).

Mercury and toxic metals block enzymes required to digest milk casein and wheat gluten, resulting in increased IgA and IgG to gluten and IgA to casein, as well as dumping morphine like substances in the blood that are neurotoxic and psychotic, as a major factor in schizophrenia, autism, ADHD, and MS (24-26). A mechanism in MS occurs due to a reduction in immune system activity. Specifically, it is the reduction in the number of the suppressor T-cells within the immune system that allows CD4 helper T-cells to do damage (102a, 181, 226, 314, 405, 507, 513, 514, 20). Thus, during an acute relapse the overall number of T-cells is reduced, the normal balance of helper and suppressor T-cells is disrupted, and helper T-cells tend to predominate. This is most pronounced during an acute relapse, but a similar situation occurs although perhaps to a lesser extent, in chronic progressive MS. A double blind study using a potent opiate antagonist, naltrexone (NAL), produced significant reduction in neurological symptomology among the 56% most responsive to opioid effects in a population of autism patients (18, 19). The behavioral improvements was accompanied by alterations in the distribution of the major lymphocyte subsets, with a significant increase in the T-helper-inducers and a significant reduction of the T-cytotoxic-suppressors and a normalization of the CD4/CD8 ratio. Low dose naltrexone (LDN) has been found to commonly be effective in reducing MS symptoms and exacerbations, apparently due its opioid suppressive effects (20).

1.7 Recovery from Chronic Neurological and Immune Related Diseases After Amalgam Removal and Mercury Detoxification

There are extensive documented cases (many thousands) where removal of amalgam fillings led to cure of serious health problems such as MS (369, 35, 94, 95, 102, 163, 170, 212, 222, 271, 291, 302, 468, 470, 34, 229, 406, 485, 523), SLE (369, 12, 35, 113, 222, 229, 233, 323, 60), Chronic Fatigue Syndrome (8, 35, 60, 212, 293, 229, 222, 232, 233, 271, 317, 323, 342, 369, 376, 382, 440, 470, 523), muscular/joint pain/Fibromyalgia (35, 222, 293, 317, 322, 369, 440, 468, 470, 523, 94), depression (94, 107, 222, 271, 294, 212, 229, 233, 285e, 317, 322, 376, 453, 465, 468, 485, 523, 35, 40), Rheumatoid Arthritis (35, 95, 103, 212, 222, 271, 322, 358, 470, 523), autoimmune thyroiditis (369, 382, 91), OralLichenPlanus (60, 75, 78, 82, 86, 87, 90, 94, 101, 133, 168, 313), ALS (97, 229, 405, 406, 468-470, 485, 35), Parkinson's/ muscle tremor (222, 248, 229, 271, 470, 212, 94, 98, 35), Alzheimer's (204, 35), and many other chronic conditions (600). In several of the studies, over 75% of those with MS and having amalgams replaced recovered or had significant improvement (369, 212(a), (b), (e), 302, 222, 35). Some of the studies reported similar success rates for SLE and autoimmune thyroiditis, but with lower number of cases treated. There is consensus that dental amalgam is the main cause of oral lichen planus and most recover after amalgam replacement.

In one study all 6 of those tested for autoimmunity by the MELISA blood lymphocyte immune reactivity test were found to be immune reactive to mercury, and all had significant improvement in their condition after amalgam replacement, as well as reduction in immune reactivity (369). Out

of 15 patients with lupus (SLE), 73% had significant improvement in health, and out of 8 with autoimmune thyroiditis 75% had significant improvement after amalgam replacement. The patients who did not have significant improvement were found to have immune reactivity to nickel which did not improve after amalgam replacement as the amalgam was not the source of the nickel exposure (369).

Clinical studies have found that patch testing is not a good predictor of success of amalgam removal, as a high percentage of those testing negative also recovered from chronic conditions after replacement of fillings (86, 87, 90). Follow up tests for autoimmune reaction to inorganic mercury after amalgam replacement have found that in most patients tested, the immune reaction as well as most symptoms disappear over time (60, 313, 405, etc.).

The level of mercury in the gums is often 1200 ppm near a gold cap on an amalgam filling (30, 35, 48, 194). These levels are among the highest levels ever measured in tissues of living organisms, exceeding the highest levels found in chronically exposed chloralkali workers, those who died from mercury in Minamata, or animals that died from mercury poisoning. The FDA/EPA action level for warnings of dangerous levels in fish or food is 1 ppm.

Tests and Treatment

In a large German study of MS patients after amalgam revision, extraction resulted in 85% recovery rate versus only 16% for filling replacement alone (302, 222). Another large clinic in Colorado has likewise found that more seriously affected cases often require more than simple replacement for successful treatment (35). Other clinics have found that recovery from serious autoimmune diseases, dementia, or cancer may require more aggressive mercury removal techniques than simple filling replacement due to body burden. This appears to be due to migration of mercury into roots & gums that is not eliminated by simple filling replacement. Also toxic metals, formaldehyde, and other toxic substances have been documented to accumulate in the jaw bone and tissue near teeth with multiple metals, as well as in pockets from extracted teeth and form cavitations (areas of toxic materials and diseased bone). Such cavitations and toxic bacteria accumulating from root-canaled teeth sometimes must be cleaned out before significant recovery can occur (200, 35, 302, 222, 207, etc.). There is a direct connection between the teeth and gums with the brain and CNS by both travel along nerve fibers and through the cranio-vertebral venous system for either toxic substances such as mercury or for bacteria (34, 325, 207, etc.), The following protocol is perhaps the most used protocol for treating these conditions and has had considerable success:

Huggins Total Dental Revision Protocol (35)

- (a) history questionnaire and panel of tests.
- (b) replace amalgam fillings starting with filling with highest negative current or highest negative quadrant, with supportive vitamin/mineral supplements.
- ©extract all root canaled teeth using proper finish protocol.
- (d) test and treat cavitations and amalgam tattoos where relevant
- (e) supportive supplementation, periodic monitoring tests, evaluate need for further treatment (not usually needed).

note: after treatment of many cases of chronic autoimmune conditions such as MS, ALS, Parkinson's, Alzheimer's, CFS, Lupus, Rheumatoid Arthritis, etc., it has been observed that often mercury along with root canal toxicity or cavitation toxicity are major factors in these conditions, and most with these conditions improve after TDR if protocol is followed carefully (35, 200, 600). Other measures in addition to TDR that have been found to help in treatment of MS in clinical experience are avoidance of milk products, get lots of sunlight, supplementation of calcium AEP (448) and alpha lipoic acid (448b). Progesterone creme has been found to promote regrowth of myelin sheaths in animals (448c).

Tests suggested by Huggins/Levy (35) for evaluation and treatment of mercury toxicity:

(a) hair element test (386) (low hair mercury level does not indicate low body level) (more than 3 essential minerals out of normal range indicates likely metals toxicity)

(b) CBC blood test with differential and platelet count

©blood serum profile

(d) urinary mercury (for person with average exposure with amalgam fillings, average mercury level is 3 to 4 ppm; lower test level than this likely means person is poor excretor and accumulating mercury, often mercury toxic (35))

(e) fractionated porphyrin (note test results sensitive to light, temperature, shaking)

(f) individual tooth electric currents (replace high negative current teeth first)

(g) patient questionnaire on exposure and symptom history

Based on the known mechanisms of damage found in these conditions, the authors of the study (463) suggest that supplementation with 100 mg MG, 25 mg vit B6, 10 mg vit B2, 15 mg Zn and 400 IU vit D and E, 100 & mgr; g Se, 180 mg EPA and 120 mg DHA per day between 14 and 16 years of age may prevent MS, and reduce further damage for those with the condition.

An Oregon researcher, Dr. R. Swank, found a significant correlation between MS and dietary fat (274). He developed a low fat diet, with animal meat mostly replaced by fish or fish oil (with EPA/DHA) and olive oil. Studies found the Swank diet effective at reducing the effects of MS. European studies have confirmed his findings regarding connection of MS to high fat animal diets, and effectiveness of the Swank diet. Studies have also found deficiency in essential fatty acids to be associated with demyelination, again consistent with the Swank findings. Studies have also found protective effects of diets high in vegetable protein, dietary fiber, cereal fiber, vit C, vit D, thiamin, riboflavin, calcium, potassium, and magnesium. A study found increased vit D helpful in reducing MS effects. Additionally curcumin and Acetyl-L-Carnitine were found by studies to be neuroprotective. Both reduce inflammation/ oxidative stress. Extracts of green tea (EGCG) and black tea (theaflavins) also have been found to be highly effective at reducing inflammatory effects (274). A study comparing alternative treatment of MS to conventional treatment found the majority using alternative treatments were satisfied with their treatment, and much lower adverse health effects from alternative treatments compared to convention treatments (273). Amalgam replacement was one of the alternatives used by some.

More information on causes, prevention, and treatment of autoimmune conditions can be found at the following *review*⁵ (100). Information on test and treatment options and doctors and dentists with experience at dealing with toxic metal related conditions can be obtained from DAMS⁶ (800-311-6265) or the dental and medical association IAOMT (www.iaomt.org/⁷).

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