amino acids like glutamate, aspartate, NAAG. The mechanisms by which exposure to mercury and other neurotoxic substances cause all of this will be documented.

The main factors determining whether chronic conditions are induced by metals appear to be exposure and genetic susceptibility, which determines individuals immune sensitivity and ability to excrete and detoxify metals (405, 342, 60, 181, 303, 314, 330, 464). 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. Those with the genetic allele ApoE4 protein in the blood have been found to detox metals poorly and to be much more susceptible to chronic neurological conditions than those with types ApoE2 or E3 (437, 577). There are also other similar factors.

Some of the toxic exposures which have been found to be a factor in ALS like symptoms other than mercury include lead (94a), pyrethrin (93), agricultural chemicals (94b), Lyme disease (471, 580), monosodium glutamate (MSG, 580), failed root canaled teeth (35, 200, 437), post-polioymelitis (580), and smoking (94cd). All have been demonstrated to cause some of the mechanisms of damage listed above seen in ALS and since such exposures are common as is exposure to mercury, such exposures appear to synergistically cause the types of damage seen in ALS. This paper will demonstrate that mercury is the most common of toxic substances which are documented to accumulate through chronic exposure in the neurons affected by ALS and which have been documented to cause all of the conditions and symptoms seen in ALS. It will also be noted that chronic infections such

Amyotrophic Lateral Sclerosis (ALS): Lou Gerhig’s Disease

The Mercury Connection

Bernie Windham (Ed.)

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1Informativo: “Susceptibility Factors in Mercury Toxicity: Immune Reactivity, Detoxification System Function, Enzymatic Blockages, Synergistic Exposures”.


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1 Amyotrophic lateral sclerosis (als): Lou Gerhig's disease - the mercury connection

Amyotrophic Lateral Sclerosis (ALS): Lou Gerhig’s Disease - The Mercury Connection.

Bernie Windham (Ed.)

1.1 Introduction

ALS is a systemic motor neuron disease that affects the corticospinal and corticobulbar tracts, ventral horn motor neurons, and motor cranial nerve nuclei (405). 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. Based on studies of groups of monozygous twins, animal studies, and ALS patient case studies, the majority of ALS cases do not appear to be genetic but rather have primarily environmental related causes often affecting genetically susceptible individuals (405, 416, 423, 471, 520, 93, 94, 97, 200, 303, 580, 35, etc.).

ALS is not a unique disease with a single cause or factor, but instead is a result of damage to motorneurons and the support system that they depend on by a variety of factors. Spinal and bulbar-onset subtypes of the disease appear to be biochemically different and have differences in mechanisms of causality (416f). Some of the mechanisms of neural damage found in ALS include increased free radical generation/oxidative damage, impaired electron transport, disrupted calcium channel function, reactive astrogliosis and dysfunctional transporters for L-glutamate, neurotoxicity, oxidative damage to mitochondrial DNA/ inhibition of the mitochondrial respiratory chain, autoimmunity, and generalized disruption of metabolism of neuroexcitotoxic...
cated as major factors in neurological disorders including ALS, motor neuron disease (MND), CFS, FM, Parkinson’s (PD), Multiple Sclerosis (MS), and Alzheimer’s (AD) [13, 43, 56, 84, 145, 169, 207b, 424, 442-444, 453, 462, 496, 577]. Mercury forms conjugates with thiol compounds such as glutathione and cysteine and causes depletion of glutathione [56], which is necessary to mitigate reactive damage. One study found that insertion of amalgam fillings or nickel dental materials causes a suppression of the number of T-lymphocytes (270), and impairs the T-4/T-8 ratio. Low T4/T8 ratio has been found to be a factor in autoimmune conditions. 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, 254, 490, 494-496]. Only a few micrograms of mercury severely disturb cellular function and inhibits nerve growth (305, 147, 175, 226, 255). Metalloprotein (MT) have a major role in regulation of cellular copper and zinc metabolism, metals transport and detoxification, free radical scavenging, and protection against inflammation [114, 442, 464, 602]. Mercury inhibits sulfur ligands in MT and in the case of intestinal cell membranes inactivates MT that normally bind cuprous ions (477, 114), thus allowing buildup of copper to toxic levels in many and malfunction of the Zn/Cu SOD function (495, 13a, 443). Mercury also causes displacement of zinc in MT and SOD, which has been shown to be a factor in neurotoxicity and neuronal diseases (405, 495, 517). Exposure to mercury results in changes in metalloprotein compounds that have genetic effects, having both structural and catalytic effects on gene expression [114, 241, 296, 442, 464, 477, 495, 517]. Some of the processes affected by such MT 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 MT formation also appears to have a relation to autoimmune reactions in significant numbers of people [114, 60, 342, 369, 442, 464]. Of as mycoplasma, echo-7 enterovirus, and candida albicans also usually affect those with chronic immune deficiencies such as ALS patients and need to be dealt with in treatment. Some studies have also found persons with chronic exposure to electromagnetic fields (EMF) to have higher levels of mercury exposure and excretion (28) and higher likelihood of getting chronic conditions like ALS (526).

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

Amalgam dental fillings are the largest source of mercury in most people with daily exposures documented to commonly be above government health guidelines (49, 79, 183, 506, 599, 600). 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 (600). Mercury has been found in autopsy studies to accumulate in the brain of those with chronic exposures, and levels are directly proportional to the number of amalgam filling surfaces (85, 270). 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 sludge9, 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 (20). People also commonly get exposures to mercury

\footnote{Internet: \texttt{http://www.flev.com/emfesf.html}.}
\footnote{Informativo: “Dental Amalgam Mercury Solutions”.}
\footnote{Internet: \texttt{http://www.flev.com/damspr2l.html}.}
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.

Mercury has been found to accumulate preferentially in the 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 (20, 291, 327, 329, 442, 48).

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). 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, bileary 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.

1.3 Effects of Exposure to Mercury and Toxic Metals

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 such as eczema, psoriasis, rheumatoid arthritis, Lupus, Scleroderma, allergies, autism, schizophrenia, (114c, 181, 303, 330, 331, 411, 412, 152b, 439, 602, 601), as well as to autoimmune conditions such as ALS, Alzheimer’s disease (AD), Chronic Fatigue (CFS), Fibromyalgia (FM), etc. (405, 342, 60, 181, 303, 314b, 513, 580, etc.). For example mercury has been found to strongly inhibit the activity of dipeptidyl peptidase (DPP IV) which is required in the digestion of the milk protein cascin (411, 412) as well as of xanthine oxidase (439).

Additional cellular level enzymatic effects of mercury’s binding with proteins include blockage of sulfur oxidation processes (33, 114c, 194, 330, 331, 412), enzymatic processes involving vitamins B6 (417) and B12 (418), effects on the cytochrome-C energy processes (43, 84, 232, 338c, 35), along with mercury’s adverse effects on cellular mineral levels of calcium, magnesium, copper, zinc, and lithium (43b, 96, 198, 333, 338, 386, 427, 430, 432, 461, 489, 507). And along with these blockages of cellular enzymatic processes, mercury has been found to cause additional neurological and immune system effects in many by causing immune autoimmunity reactions (60, 152c, 181, 2888, 314, 342, 405, 513). Recent studies gives a comprehensive review of studies finding a connection between ALS, toxic metals, and autoimmune (405, 580). Studies have found the presence of antibodies in ALS patients that interact with motor neurons, inhibiting the sprouting of axons. Immune complexates have also been found in the spinal cords of ALS patients (580). T cells, activated microglia, and IgG within the spinal cord may be a primary event that leads to lesions and tissue destruction.

Oxidative stress and reactive oxygen species (ROS) have been impli-
Reduced levels of magnesium and zinc are related to metabolic syndromes, type 2 diabetes, and insulin resistance [15]. Reduced levels of magnesium and zinc levels as well as initiating glucose transport, insulin sensitivity, and insulin resistance [16].

The TNFα (tumor necrosis factor-alpha) is a cytokine that controls a wide range of immune responses in mammalian cells and is an important factor in the control of cell death and inflammation (17). Glutathione levels are disrupted by toxic exposures such as mercury, indicating that the control mechanisms for the TNFα-apoptosis mechanism (18) are inactivated by these exposures. When glutathione is depleted, the brain becomes more vulnerable to neuronal damage. Glutathione depletion has been shown to increase the risk of developing Alzheimer's disease (19). The pathogenesis of Alzheimer's disease involves oxidative stress and neuroinflammation, which cause the release of proinflammatory cytokines such as TNFα and IL-1β. These cytokines activate microglia, which produce ROS and RNS, contributing to neurodegeneration. Glutathione depletion has been shown to exacerbate the neuroinflammatory response and increase the risk of developing Alzheimer's disease (20).
Chronic neurological conditions such as ALS appear to be primarily caused by chronic or acute brain inflammation. The brain is very sensitive to inflammation. Disturbances in metabolic networks: e.g., immuno-inflammatory processes, insulin-glucose homeostasis, adipokine synthesis and secretion, intra-cellular signaling cascades, and mitochondrial respiration have been shown to be major factors in chronic neurological conditions (592, 593, 598, 580, etc.). Inflammatory chemicals such as mercury, aluminum, and other toxic metals as well as other excitotoxins including MSG and aspartame cause high levels of free radicals, lipid peroxidation, inflammatory cytokines, and oxidative stress in the brain and cardiovascular systems (13, 582, 595-598, etc.)

In amyotrophic lateral sclerosis (ALS) non-neuronal cells play key roles in disease etiology and loss of motoneurons via noncell-autonomous mechanisms. Reactive astrogliosis and dysfunctional transporters for L-glutamate are common hallmarks of ALS pathology (416d). Oxidative and excitotoxic insults exert differential effects on spinal motoneurons and astrocytic glutamate transporters in the progression of ALS. Excitotoxicity in ALS affects both motor neurons and astrocytes, favouring their local interactive degeneration. 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). Mercury and increased glutamate in the plasma activate free radical forming processes like xanthine oxidase which produce oxygen radicals and oxidative neurological damage (142, 416, 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 Dimutase (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). A recent study has linked some cases of sporadic ALS with the failure to edit key residues in ionotropic glutamate receptors, resulting in excessive influx of calcium ions into motor neurones which in turn triggers cell death. The study suggests that edited AMPA glutamate (GluR2) receptor subunits serve as gatekeepers for motor neurone survival. (525)

These inflammatory processes damage cell structures including DNA, mitochondria, and cell membranes. They also activate microglia cells in the brain, which control brain inflammation and immunity. Once activated, the microglia secrete large amounts of neurotoxic substances such as glutamate, an excitotoxin, which adds to inflammation and stimulates the area of the brain associated with anxiety (598). Inflammation also disrupts brain neurotransmitters resulting in reduced levels of serotonin, dopamine, and norepinephrine. Some of the main causes of such disturbances that have been documented include vaccines, mercury, aluminum, other toxic metals, MSG, aspartate, etc. (582, 593, 598, 600, etc.) High levels of aluminum exposure along with low levels of other minerals such as calcium and magnesium have been documented to cause neurological degeneration and appear to be the cause of high ALS and Parkinson’s in the past in Guam (518). There is evidence that aluminum hydroxide in vaccines can cause symptoms such as those seen in ALS (582). Aluminum has been found to be a factor in some Alzheimer’s and Parkinson’s cases.

Programmed cell death (apoptosis) is documented to be a major factor in degenerative neurological conditions like ALS, Alzheimer’s, MS, Parkinson’s, etc. Some of the factors documented to be involved in apoptosis of neurons and immune cells include mitochondrial membrane dysfunction (56bc, 416). Mitochondrial DNA mutations or dysfunction is fairly common, found in at least 1 in every 200 people (275), and toxicity effects affect this population more than those with

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Informativo: “Alzheimer’s Disease and Other Autoimmune Degenerative Conditions: the Mercury Connection”.

Informativo: “Toxic Exposures and Parkinsons: the Mercury Connection”.
also interrupts the cytochrome oxidase system, blocking the ATP energy function (35, 43, 84), lowering immune growth factor IGF-I levels and impairing astrocyte function (119, 152, 416d, 497). Astrocytes are common cells in the CNS involved in the feeding and detox of nerve cells. Increases in inflammatory cytokines such as caused by toxic metals trigger increased free radical activity and damage to astrocyte and astrocyte function (152, 416d). IGF-I protects against brain and neuronal pathologies like ALS, MS, and Fibromyalgia by protecting the astrocytes from this destructive process.

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 ALS cases there was a reduction in serum magnesium and RBC membrane Na (+)-K+ ATPase activity and an elevation in plasma serum digoxin (263, 260d). The activity of all serum free-radical scavenging enzymes, concentration of glutathione, alpha tocopherol, iron binding capacity, and ceruloplasmin decreased significantly in ALS, 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 ALS (13a, 111, 288, 442, 521b, 43, 56, 263, etc.)

Mercury exposure also degrades the immune system resulting in more susceptibility to viral, bacterial, or parasitic effects along with candida albicans which are often present in those with chronic conditions and require treatment (404, 468, 470, 485, 600). Four such commonly found in ALS patients are mycoplasma AND echo-7 enterovirus 33, 330, 331, 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, 416), induces peroxytritrite 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); inhibits functional methylation (504), inducement of inflammatory cytokines (126, 152, 181) and autoimmunity (226, 272, 369, 405, etc.)

Exposure to mercury vapor and methyl mercury is well documented to commonly cause conditions involving tremor, with populations exposed to mercury experiencing tremor levels on average proportional to exposure level (250, 565). However bacteria, yeasts, and Vitamin B12 methylate inorganic mercury to methyl mercury in the mouth and intestines (599, 505) and mercury inhibits functional methylation in the body, a necessary process (504).

Mercury exposure causes high levels of oxidative stress/ reactive oxygen species (ROS) (13), which has been found to be a major factor in apoptosis and neurological disease (56, 250, 441, 442, 443, 13) including dopamine or glutamate related apoptosis (288c).

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). 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, 56, 442) and a factor in other neurological conditions.

Mercury blocks the immune function of magnesium and zinc (198, 427, 38), whose deficiencies are known to cause significant neurologi-
cal effects (461, 463, 430, 601). 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 (477, 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 (580). Exposures to toxic metals such as mercury and cadmium have been found to cause such effects (13a, 495, 517, etc.) 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 non-familial ALS (489, 490, 495, 464, 469, 111). This condition can result in zinc deficient SOD and oxidative damage involving nitric oxide, peroxynitrite, and lipid peroxidation (490, 495, 496, 489, 521, 524), which have been found to affect glutamate mediated excitability and apoptosis of nerve cells and effects on mitochondria (119c, 412, 416, 495, 496, 502, 519, 524). These effects can be reduced by zinc supplementation (464, 495, 517, 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, gingko biloba, N-acetyl cysteine, melatonin, etc. (444, 464, 494, 495, 469, 470, 521, 524, 572). Ceruloplasmin in plasma can be similarly affected by copper metabolism dysfunction, like SOD function, and is often a factor in neurodegeneration (489).

Motor neuron dysfunction and loss in amyotrophic lateral sclerosis (ALS) have been attributed to several different mechanisms, including increased intracellular calcium (333, 496, 507), glutamate dysregulation and excitotoxicity (119c, 412, 416, 496, 502), oxidative stress and free radical damage (13, 43, 56, 442, 490), nitric oxide related toxicity caused by peroxynitrite (524, 521), mitochondrial damage/dysfunction (519), neurofilament aggregation and dysfunction of transport mechanisms (507), and autoimmunity (313, 314, 369, 405, 513). These alterations and effects are not mutually exclusive but rather are synergistic, and increased calcium and altered calcium homeostasis appears to be a common denominator. Mercury forms strong bonds with the SH groups of proteins causing alteration of the transport of calcium (333, 43, 96, 254, 329, 432, 496) and causes mitochondrial release of calcium (21, 35, 43, 329, 333, 432, 496, 519). This results in a rapid and sustained elevation in intracellular levels of calcium (333, 496). 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 (270, 288, 329, 333, 432, 56). Protein Kinase C (PKC) regulates intracellular and extra cellular signals across neuronal membranes, and both forms of mercury inhibit PKC at micro molar levels, as well as inhibiting phorbal ester binding (43, 432). They also block or inhibit calcium L-channel currents in the brain in an irreversible and concentration dependent manner. Mercury vapor or inorganic mercury exposure affects the posterior cingulate cortex and causes major neurological effects with sufficient exposure (428, 453). Metallic mercury is much more potent than methyl mercury in such actions, with 50% inhibition in animal studies at 13 ppb (333, 329). Mercury is seen to be a factor in all of these known mechanisms of neural degeneration seen in ALS and other motor neuron conditions.

Spatial and temporal changes in intracellular calcium concentrations are critical for controlling gene expression and neurotransmitter release in neurons (432, 496, 43, 114). Mercury alters calcium homeostasis and calcium levels in the brain and affects gene expression and neurotransmitter release through its effects on calcium, etc. Mercury inhibits sodium and potassium (N, K) ATPase in dose dependent manner and inhibits dopamine and norepinephrine uptake by synaptosomes and nerve impulse transfer (288, 270, 56, 43, 35). Mercury
with growth hormone levels and have been found to be the best easily measured marker for levels of growth hormone, but males have been found more responsive to this factor than women (497). IGF-I controls the survival of spinal motor neurons affected in ALS during development as well as later in life (497, 498). IGF-I and insulin levels have been found to be reduced in ALS patients with evidence this is a factor in ALS (497, 498). Several clinical trials have found IGF-I treatment is effective at reducing the damage and slowing the progression of ALS and Alzheimer’s with no medically important adverse effects (498). It has also been found that in chronically ill patients the levels of pituitary and thyroid hormones that control many bodily processes are low, and that supplementing both thyrotropin-releasing hormone and growth control hormone is more effective at increasing all of these hormone levels in the patient (499).

Extremely toxic anaerobic bacteria from root canals or cavitations formed at incompletely healed tooth extraction sites have also been found to be common factors in Fibromyalgia and other chronic neurological conditions such as Parkinson’s and ALS, with condensing osteitis which must be removed with a surgical burr along with 1 mm of bone around it (35, 200, 437, 600). Cavitations have been found in 80% of sites from wisdom tooth extractions tested and 50% of molar extraction sites tested (35, 200, 437). The incidence is likely somewhat less in the general population. 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 neurologic conditions like ALS. Some that have been found to be effective include Vit B6, methyl cobalamin (B12), L-carnitine, choline, ginseng, Ginkgo biloba, vitamins C and E, CoQ10, nicotine, and omega 3 fatty acids (fish and flaxseed oil) (417, 468).

Clinical tests of patients with ALS, MND, Parkinson’s, Alzheimer’s, Lupus (SLE), and rheumatoid arthritis have found that the patients (468, 470), candida albicans (404), and parasites (485). One clinic found that over 85% of patients with ALS tested have mycoplasma infection, often M. Pneumoniae (470), but in Gulf War veterans mostly a manmade used in bioterrorism agents- M. fermentans. Mercury from amalgam interferes with production of cytokines that activate macrophage and neutrophils, disabling early control of viruses or other pathogens and leading to enhanced infection (131). While the others are also being commonly found, mycoplasma has been found in 85% of ALS patients by clinics treating such conditions (470). Mycoplasma appears to be a cofactor with mercury in the majority of cases and shifts the immune T cell balance toward inflammatory cytokines (470b). Treatment of these chronic infections are required and documented to cause improvement in such patients (470).

Mercury lymphocyte reactivity and effects on amino acids such as glutamate in the CNS induce CFS type symptoms including profound tiredness, musculoskeletal pain, sleep disturbances, gastrointestinal and neurological problems along with other CFS symptoms and Fibromyalgia11 (346, 342, 369, 416, 496, 513, 119b, 152, 314). Mercury has been found to be a common cause of Fibromyalgia (293, 346, 369), which based on a Swedish survey occurs in about 12% of women over 35 and 5.5% of men (342). ALS patients have been found to have a generalized deficiency in metabolism of the neuroexcitotoxic amino acids like glutamate, aspartate, NAAG, etc.(416). Glutamate is the most abundant amino acid in the body and in the CNS acts as excitory neurotransmitter (346, 412, 416, 438, 496, 119c), which also causes influx of calcium. Astrocytes, a type of cell in the brain and CNS with the task of keeping clean the area around nerve cells, have a function of neutralizing excess glutamate by transforming it to glutamic acid. If astrocytes are not able to rapidly neutralize excess glutamate, then a buildup of glutamate and calcium occurs, causing swelling and

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11Informativo: “Chronic Fatigue Syndrome, Fibromyalgia, Scleroderma, Lupus, Rheumatoid Arthritis, MCS: The Mercury Connection”.

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neurotoxic effects (119, 152, 333, 416, 496, 524). Mercury and other toxic metals inhibit astrocyte function in the brain and CNS (119, 152, 416), 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. This is also a factor in conditions such as CFS, Parkinson’s, and ALS (346, 416, 496, 524, 600). 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 radicals forming processes like xanthine oxidase which produce oxygen radicals and oxidative neurological damage (346, 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 Dismutase (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).

In addition to the documentation showing the mechanisms by which mercury causes the conditions and symptoms seen in ALS and other neurodegenerative diseases, many studies of patients with major neurological or degenerative diseases have found direct evidence mercury and amalgam fillings play a major role in development of conditions such as ALS (92, 97, 207, 229b, 305, 325, 327, 416, 423, 442, 468, 470, 520, 35). Such supplements including N-acetylcysteine (NAC), Vitamins E and C, zinc, and creatine have been found to offer significant protection against cell apoptosis and neurodegeneration in neurological conditions such as ALS (13c, 56a, 517, 524, 564, 494).

Medical studies and doctors treating chronic conditions like 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 neurologic conditions. Some that have been found to be effective include CoQ10 (444), ginkgo biloba and pycnogenol (494a), NAC (54, 494a), Vit B6, methyl cobalamin (B12), L-carnitine, choline, ginseng, vitamins C and E, nicotine, and omega 3 fatty acids (fish and flaxseed oil) (417, 495e). A study demonstrated protective effects of methylcobalamin, a vitamin B12 analog, against glutamate-induced neurotoxicity (503), and similarly for iron in those who are iron deficient.

In a study of the brains of persons dying of ALS, spherical and crescent-shaped intraneuronal inclusions (SCI) were distributed in association with each other among the parahippocampal gyrus, dentate gyrus of the hippocampus and amygdala, but not any non-motor-associated brain regions (522). The occurrence of SCI in both the second and third layers of the parahippocampal gyrus and amygdala was significantly correlated to the presence of dementia in ALS cases. Mercury has been found to accumulate in these areas of the brain and to cause adverse behavioral effects in animal studies and humans (66, 287, 305).

Another neurological effect of mercury that occurs at very low levels is inhibition of nerve growth factors, for which deficiencies result in nerve degeneration. Only a few micrograms of mercury severely disturb cellular function and inhibits nerve growth (175, 147, 226, 255, 305, 149). Prenatal or neonatal exposures have been found to have life long effects on nerve function and susceptibility to toxic effects. Prenatal mercury vapor exposure that results in levels of only 4 parts per billion in newborn rat brains was found to cause decreases in nerve growth factor and other effects (305). This is a level that is common in the population with several amalgam fillings or other exposures (600). There is also evidence that fetal or infant exposure causes delayed neurotoxicity evidenced in serious effect at middle age (255). Insulin-like-growth factor I (IGF-I) are positively correlated
mercury (54). Experienced doctors have also found additional zinc to be useful when chelating mercury (222) as well as counteracting mercury’s oxidative damage (43). Zinc induces metallothionein which protects against oxidative damage and increases protective enzyme activities and glutathione which tend to inhibit lipid peroxidation and suppress mercury toxicity (430, 464). Also lipoic acid, LA, has been found to dramatically increase excretion of inorganic mercury (over 12 fold), but to cause decreased excretion of organic mercury (572a) and copper. Lipoic acid has a protective effect regarding lead or inorganic mercury toxicity through its antioxidant properties (572), but should not be used with high copper until copper levels are reduced. LA and NAC (N-acetyl cysteine) also increase glutathione levels and protect against superoxide radical/ peroxynitrite damage, so thus have an additional neuroprotective effect (494ab, 521, 572c; 54). Zinc is a mercury and copper antagonist and can be used to lower copper levels and protect against mercury damage. Lipoic acid has been found to have protective effects against cerebral ischemic-reperfusion, excitotoxic amino acid (glutamate) brain injury, mitochondrial dysfunction, diabetic neuropathy (494).

Antioxidants such as carnosine (495a), Coenzyme Q10, Vitamins B & C & E & D, ginkgo biloba, superoxide dismutase (SOD), N-acetyl-cysteine (NAC), Alpha Lipoic Acid, and pyrgeenol have also been found protective against degenerative neurological conditions (494, 495e, 444, 580). Other supplements found to be protective against neuronal degenerative conditions include Acetyl-L-Carnitine, EFAs (DHA/EPA), DHEA, CoQ10, magnesium, Vit B1 & B5, hydrgine, and octacosanol (580). Such supplements only offer limited protection and reductions in progression of ALS without other measures that deal with underlying mechanisms of causality.

Other supplements that appear useful in conditions involving neurotoxicity or muscle function degeneration include creatine (502, 580) and lithium (590). In the motor cortex of the ALS group the N-
chronic neurological conditions by neurologist such as Perlmutter in Florida (469).

In one subtype of ALS, damaged, blocked, or faulty enzymatic superoxide dismutase (SOD) processes appear to be a major factor in cell apoptosis involved in the condition (443, 495). Mercury is known to damage or inhibit SOD activity (13, 33, 111).

1.4 Prevention and Treatment of ALS

Tick-borne encephalitis, such as Lyme Disease, has been found to cause ALS symptoms in a significant portion of untreated acute cases (471). Lyme disease is widespread in the U.S. Large numbers of patients diagnosed with ALS and other neurological conditions have been found to have treatable tick-borne encephalitis, and many have recovered after treatment. Anyone diagnosed with degenerative neurological symptoms should investigate the possibility of lyme disease or post-polio encephalitis. Poliomyelitis also has a chronic state that resembles ALS (580).

Since elevated plasma cysteine has been reported in some ALS patients, sulfite and cysteine toxicity may be involved in other cases of ALS. Patients with ALS with nonmutant-SOD should be tested for sulfite toxicity, cysteine, glutamate and GSH levels, and whether they have low levels of GSH metabolism enzymes. During the time when strict dietary and supplement measures normalized a patient’s whole blood GSH, blood cysteine, and urine sulfite, the patient did not experience additional physical decline (330b).

Total dental revision (TDR) which includes replacing amalgam fillings, extracting root canaled teeth, and treating cavitations has been found to offer significant health improvements to many with ALS and other autoimmune conditions (35, 200, 293, 437). Root canals and cavitations have been found to harbor anaerobic bacteria which give off toxins of extreme toxicity which block enzymatic processes at the cellular level causing degenerative processes according to the medical labs that do the tests (437, 200, 35), similar to mercury’s effects but in some cases even more toxic. 1GF-1 treatments have also been found to alleviate some of the symptoms of ALS (424). 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. Some that have been found to be effective in treating metals related autoimmune conditions include Vit B6, CoenzymeQ10, methyl cobalamine (B12), SAMe, L-carnitine, choline, ginseng, Ginkgo biloba, vitamins C and E, nicotine, and omega 3 fatty acids (fish and flaxseed oil) (417, 444, 468, 580).

One dentist with severe symptoms similar to ALS improved after treatment for mercury poisoning (246), and others treated for mercury poisoning or using TDR have also recovered or significantly improved (97, 229, 405, 406, 437, 468-470, 485, 35). The Edelson Clinic in Atlanta which treats ALS patients reports similar experience (406), and the Perlmutter Clinic has also had some success with treatment of ALS and other degenerative neurological conditions (469).

While there are many studies documenting effectiveness of chemical chelators like DMSA and DMPS at reducing metals levels and alleviating adverse effects for most conditions, and many thousands of clinical case results (600, 601); there is also some evidence from animal studies that these chelators can result in higher levels of mercury in the motor neurons in the short term which might be a problem for ALS patients (600). Thus other detox options might be preferable for ALS patients until enough clinical evidence is available treating ALS patients with them with mercury toxicity. Another chelator used for clogged arteries, EDTA, forms toxic compounds with mercury and can damage brain function (307). Use of EDTA may need to be restricted in those with high Hg levels. N-acetyl cysteine (NAC) has been found to be effective at increasing cellular glutathione levels and chelating...


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Two experimental treatment for ALS that has shown some effectiveness at reducing disease progression is recombinant human insulin-like growth factor and Orap (Pimozide) (580).

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