Toxic Exposures and Parkinsons:

the Mercury Connection

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1.1 Introduction

There has been a huge increase in the incidence of degenerative neurological conditions in virtually all Western countries over the last 2 decades (574, 303). The increase in Parkinson's and other motor neuron disease has been over 50%. The primary cause appears to be increased exposures to toxic pollutants such as toxic metals, pesticides, etc. resulting in brain *inflammation*[1] and oxidative damage of free-radicals (574, 580, 598).

Dental amalgam fillings[2] are the largest source of mercury in most people with daily exposures documented to commonly be above government health guidelines (49, 79, 183, 199, 506, 600, 217). This is due to continuous vaporization of mercury from amalgam in the mouth, along with *galvanic currents*[3] from mixed metals in the mouth that deposit the mercury in the gums and oral cavity (605, 580). 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*[4], 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 and other toxic metals such as lead, arsenic, nickel, and aluminum from food, water, and other sources (601, 303, 592). 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 (580, 598, 601, 602, 303). A study found that those with occupational exposure to lead, arsenic, or copper have more than double the incidence of Parkinson's than normal (560).

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

[1] **Internet:** "http://www.flcv.com/inflamhg.html".
[2] **Informativo:** "Dental Amalgam Mercury Solutions".
[3] **Informativo:** "Oral galvanism and Electromagnetic Fields (EMF)".
[4] **Internet:** "http://www.flcv.com/damspr2f.html".

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 high vapor pressure 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 the directly correlated to the number of amalgam surfaces and the largest source of mercury in the majority of people (49, 183, 199, 209, 79, 99, 600), 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 μg/kg body weight/day, and the oral mercury level commonly exceeds the mercury MRL of the U.S.ATSDR of 0.2 μg/ cubic meter of air (217, 600). When amalgam fillings are replaced, levels of mercury in the blood, urine, saliva, and feces typically rise temporarily but decline between 60 to 90% within 6 to 9 months (79, 600).

The main factors determining whether chronic conditions are induced by metals appear to be exposure and genetic *susceptibility*[5], 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

1.2 Mechanisms by which mercury causes neurological conditions found in Parkinson's and neurodegenerative diseases

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 inducement of the inflamatory cytokine Tumor Necrosis Factor-alpha (TNFa) (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), increased calcium influx toxicity (296b, 333, 416, 432, 462c, 507) and DNA fragmentation (296, 42, 114, 142) and mitochondrial membrane dysfunction (56de, 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 less susceptibility to mitochondrial dysfunction. This has been found to be a factor in conditions like Parkinson's. The mechanisms by which mercury causes (often *synergistically*[6] along with other toxic exposures) all of these conditions and neuronal apoptosis will be documented.

TNFa (tumor necrosis factor-alpha) is a cytokine that controls a wide range of immune cell response in mammals, including cell death (apoptosis) in neuronal and immune cells. 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 mechansim for the TNFa apoptosis mechanism (126a). Gluthathione 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 mechinsisms are disrupted by toxic exposures such as mercury, neuronal cell apoptosis results and neurological damage. **Mercury has been shown to induce TNFa and deplete glutathione, causing inflamatory effects and cellular apoptosis**

[5]**Internet:** "http://www.home.earthlink.net/%7Eberniew1/suscept.html".
[6]**Internet:** "http://www.flcv.com/synergis.html".

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 of calcium (333, 43b, 254, 263, 416, 462, 507); inhibitation of glucose transport (338, 254), and of enzyme function, protein transport, and other essential nutrient transport (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 gluthathione (necessary for detoxification processes) (111, 126, 424), inhibition of glutathione peroxidase enzyme (13a, 442), inhibits glutamate uptake (119, 416), induces peroxynitrite and lipid peroxidation damage (521b, 119b), causes abnormal migration of neurons in the cerebral cortex (149), immune system damage (34, 111, 194, 226, 252, 272, 316, 325, 355); and inducement of inflammatory cytokines (126, 181).

Oxidative stress and reactive oxygen species (ROS) have been implicated as major factors in neurological disorders including stroke, Parkinson's Disease (PD), Alzheimer's, ALS, etc.(13, 424, 442, 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 dismustase (SOD) (13, 441, 443). Only a few micrograms of mercury severely disturb cellular function and inhibit nerve growth (147, 149, 226, 255, 305, 442). Exposure to mercury results in metalloprotein compounds that have genetic effects, having both structural and catalytic effects on gene expression (114, 241, 296, 442). 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). 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 have a relation to autoimmune reactions in significant numbers of people (114, 60, 313, 342, 368, 369, 405, 442). Increased formation of reactive oxygen species (ROS) has also been found to increase formation of advanced glycation end products (AGEs) that have been found to cause activation of glial cells to produce superoxide and nitric oxide, they can be considered part of a vicious cycle, which finally leads to neuronal cell death in the substantia nigra in PD (424).

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 congugates are found to be highest in the brain substantia nigra with similar congugates formed with L-Dopa and dopamine in Parkinson's disease (56). Mercury depletion of GSH and damage to cellular mitochrondria 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). Exposure to mercury vapor and methyl mercury is well documented to commonly cause conditions involving tremor and/or ataxia, with populations exposed to mercury experiencing tremor on average proportional to exposure level (250, 565, 98). One study found higher than average levels of mercury in the blood, urine, and hair of Parkinson's disease patients (363). Another study (169) found blood and urine mercury levels to be very strongly related to Parkinson's with odds ratios of approx. 20 at high levels of Hg exposure. Other studies (145) that reviewed occupational exposure data found that occupational exposure to manganese and copper have high odds rations for relation to PD, as well as multiple exposures to these and lead, but one study noted that this effect was only seen for exposure of over 20 years. Occupational exposure to mercury has been found to cause Parkinson's (98). One study found the EDTA chelation was effective in reducing some of the effects (145b).

Glutamate is the most abundant amino acid in the body and in the CNS acts as excitory neurotransmitter (346, 386), which also causes inflow 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 neurotoxic effects (119, 333). Mercury and other toxic metals inhibit astrocyte function in the brain and CNS (119), causing increased glutamate and calcium related neurotoxicity (119, 333, 226) which are responsible for much of the fibromylgia symptoms. This is also a factor in conditions such as CFS, Parkinson's, and ALS (346, 416).

Parkinson's disease involves the aggregation of alpha-synuclein to form fibrils, which are the major constituent of intracellular protein inclusions (Lewy bodies and Lewy neurites) in dopaminergic neurons of the substantia nigra (564). Occupational exposure to specific metals, especially manganese, copper, lead, iron, mercury, aluminum, appears to be a risk factor for Parkinson's disease based on epidemiological studies (98, 145, 518, 564, 580). Elevated levels of several of these metals have also been reported in the substantia nigra of Parkinson's disease subjects (564, 580, 518).

Exposure to aluminum hydroxide in vaccines also appears to sometimes cause symptoms similar to Parkinson's or other neurological conditions (592).

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 Parkinson's cases there was an elevation in plasma serum digoxin and a reduction in serum magnesium, 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 PD, 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 Parkinson's (13a, 111, 288, 442, 521b, 43, 56, etc.)

Many 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 Alzheimers (66, 67, 158, 166, 204, 207, 221, 242, 244, 257, 295, 300), ALS (92, 97, 325, 442), MS (102, 163, 170, 184, 212, 213, 285, 291, 302, 324, 326), Parkinson's (98, 145, 169, 248, 250, 256, 258, 363, 405, 56, 84), etc. 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). 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 congugates are found to be highest in the brain substantia nigra with similar congugates formed with L-Dopa and dopamine in Parkinson's disease (56, 442). Mercury depletion of GSH and damage to cellular mitochrondria and the increased lipid perxodation in protein and DNA oxidation in the brain appear to be a major factor in Parkinson's disease (33, 56, 442).

An EKM system for evaluating nerve and muscle function ability using a set of 5 measures (precision, imprecision, tremor, Fitts' constant, and irregularity) and tested on a group of Cree Indians with mercury exposure from fish eating (565). Ninety-six participants, including 30 controls subjects, 36 Cree subjects exposed to mercury, 21 subjects with Parkinson disease, 6 with presumed cerebellar deficit, and 3 with essential tremor, participated in the study. An ANOVA on the three largest groups generated significant results for tremor, Fitts' constant, and irregularity between the Cree and the control subjects and on Fitts' constant and irregularity between the subjects with Parkinson's disease

and the control subjects. Three subgroups of the same mean age composed of six subjects each were selected. One was composed of Cree subjects with the highest level of mercury exposure, another with Cree subjects having a low level of mercury exposure, and a third with control subjects. An ANOVA on these three groups revealed a significant difference between both groups of Cree subjects and the control group for Fitts' constant and irregularity. These preliminary results suggest that the EKM system is able to discriminate the performance of different groups of subjects and found significant evidence that mercury exposure is related to nerve and muscle function conditions such as tremor and Parkinson's (565).

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, 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, and platelet aggregation through interfering with the sodium pump.

Studies have found mercury and lead cause autoantibodies to neuronal proteins, neurofilaments, and myelin basic protein (MBP) (39b, 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, 126, 303, 516, 35c). 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+ suppresser 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. Mercury penetrates and damages the blood brain barrier allowing penetration of the barrier by other substances that are neurotoxic (20, 38, 85, 105, 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 and studies have found mercury related mental effects to be indistinguishable from those of MS patients (207, 212, 222, 244, 271, 286, 289, 291, 302, 324, 326, 183, 184). MS patients have been found to have much higher levels of mercury in cerebrospinal fluid compared to controls (163, 35, 139). Large 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). Most recovered after mercury detox (369), with some requiring additional treatment for viruses and intestinal dysbiosis. Similarly *thousands of MS patients*[7] have been documented to have recovered or significantly improved after amalgam replacement (35, 212, 228, 291, 302, 600, etc.)

Mercury has been found to accumulate preferentially in the primary motor function related areas 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). There is considerable indication this may be a factor in development of ALS and other neurodegenerative conditions (48, 325, 405, 442). Treatment using IV glutathione, vitamin C, and minerals has been found to be very effective in the stabilizing and amelioration of some of these chronic neurological conditions by neurologists such as Perlmutter in Florida (469).

Low levels of toxic metals have been found to inhibit dihydroteridine reductase, which affects the

[7]**Informativo:** "Mercury from Amalgam Fillings is a Common Cause of MS, ALS, PD, SLE, RA, MCS, AD, etc.".

neural system function by inhibiting brain transmitters through its effect on phenylalanine, tyrosine and tryptophan transport into neurons (122, 257, 258, 289, 372). This was found to cause severe impaired amine synthesis and hypokinesis. Tetrahydro-biopterin, which is essential in production of nerurotransmitters, is significantly decreased in patients with Alzheimer's, Parkinson's, and MS. Such patients have abnormal inhibition of neurotransmitter production (432).(supplements which inhibit breach of the blood brain barrier such as bioflavonoids have been found to slow such neurological damage).

Clinical tests of patients with MND, ALS, Parkinson's, Alzheimer's, Lupus (SLE), and rheumatoid arthritis have found that the patients generally have elevated plasma cysteine to sulphate ratios, with the average being 500% higher than controls (330, 331, 56), and in general being poor sulphur oxidizers. Mercury has been shown to diminish and block sulphur oxidation and thus reducing glutathione levels which is the part of this process involved in detoxifying and excretion of toxics like mercury (33, 442). Glutathione is produced through the sulphur oxidation side of this process. Low levels of available glutathione have been shown to increase mercury retention and increase toxic effects (111), while high levels of free cysteine have been demonstrated to make toxicity due to inorganic mercury more severe (333, 194, 56). Mercury has also been found to play a part in neuronal problems through blockage of the P-450 enzymatic process (84). Other toxic metals and toxics such as pesticides have also been found to cause the types of damage seen in Parkinson's and to exposure to have positive correlation to Parkinson's (400, 98, 145). Another exposure that affects some appears to be hexane (505). There are *synergistic effects*[8] of various toxics that result in conditions like Parkinson's (524b, 13c). Determination of one's factors by history assessment and tests is a first step in improving the condition.

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, Parkinsons, etc. as early as age 40, whereas those with type APOE-2 readily excrete mercury and are less susceptible. Those with type APOE-3 are intermediate to the other 2 types (437, 35).

The Huggins Clinic (35) using total dental revision (TDR) has successfully treated over a thousand patients with chronic autoimmune conditions like MS, Parkinson's, Lupus, ALS, AD, diabetes, etc., including himself with the population of over 1000 (approx. 85%) who experienced significant improvement in MS. Jaw bone cavitations were found to be common significant factors in some of these conditions such as Parkinson's (35, 580).

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.

(c) 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).

(f) avoid acute exposures/challenges to the immune system on a weekly 7/14/21 day pattern.

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)

[8]**Internet:** "http://www.flcv.com/synergis.html".

(b) CBC blood test with differential and platelet count

(c) 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 urine test (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

(h) specific gravity of urine (test for pituitary function, s.g¿1.022 normal; s.g.¡ 1.008 consistent with depression and suicidal tendencies (35))

Note: during initial exposure to mercury the body marshals immune system and other measures to try to deal with the challenge, so many test indicators will be high; after prolonged exposure the body and immune system inevitably lose the battle and measures to combat the challenge decrease-so some test indicator scores decline. Chronic conditions are common during this phase. Also high mercury exposures with low hair mercury or urine mercury level usually indicates body is retaining mercury and likely toxicity problem (35). In such cases where (calcium¿ 1100 or ¡ 300 ppm) and low test mercury, manganese, zinc, potassium; mercury toxicity likely and hard to treat since retaining mercury.

Test results indicating mercury/metals toxicity (35):

(a) white blood cell count ¿7500 or ¡ 4500

(b) hemocrit ¿ 50% or ¡ 40%

(c) lymphocyte count ¿ 2800 or ¡ 1800

(d) blood protein level ¿ 7.5 gm/100 ml

(e) triglycerides ¿ 150 mg %ml

(f) BUN ¿ 18 or ¡ 12

(g) hair mercury ¿ 1.5 ppm or ¡ .4 ppm

(h) oxyhemoglobin level ¡ 55% saturated

(i) carboxyhemoglubin ¿ 2.5% saturated

(j) T lymphocyte count ¡ 2000

(k) DNA damage/cancer

(l) TSH ¿ 1 μg

(m) hair aluminum ¿ 10 ppm

(n) hair nickel ¿ 1.5 ppm

(o) hair manganese ¿ 0.3 ppm

(p) immune reactive to mercury, nickel, aluminum, etc.

(q) high hemoglobin and hemocrit and high alkaline phosphatase (alk phos) and lactic dehydrogenese (LDA) during initial phases of exposure; with low/marginal hemoglobin and hemocrit plus low oxyhemoglobin during long term chronic fatigue phase.

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).

There are extensive documented cases (many thousands) where removal of amalgam fillings led to cure of serious health problems such as MS (94, 95, 102, 170, 212, 213, 222, 271, 291, 302, 34, 35, 229, 405), ALS (229, 325, 405, 535, 35), Parkinson's/ muscle tremor (222, 228, 248, 229, 233c, 271, 212, 322, 469, 557, 94, 98, 35), Alzheimer's (204, 35), muscular/joint pain/ fibromyalgia (222, 293, 317, 322, 369, 35, 94), anxiety & mental confusion (94, 212, 222, 229, 233, 271, 317, 303, 320, 322, 57, 35), Chronic Fatigue Syndrome (212, 293, 229, 222, 232, 233, 271, 313, 317, 303, 320, 368, 369, 376, 595, 35), memory disorders (94, 222, 303, 595, 35)

Medical studies and doctors treating fibromylagia 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 such as Parkinson's include Vit B6, CoenzymeQ10, methyl cobalamine (B12), L-carnitine, choline, ginseng, Ginkgo biloba, vitamins C and E, nicotine, octacosanol, phosphatifylserine, and omega 3 fatty acids (fish and flaxseed oil), tumeric, lipoic acid, proteolytic enzymes, and Hydergine (417, 444, 580). Reduced glutathione (GSH) and N-acetyl cysteine (NAC) have been found to be protective against cellular apoptosis seen in Parkinson's and other neurodegenerative conditions (56ab, 462c, 149b). High levels of Vitamins C and E along with zinc (517) have also been found protective against oxidative stress and some effects of mercury toxicity including for Parkinson's (41, 63, 462c, 580, 56a). CoQ10 at 600 mg per day was found effective at reducing Parkinson's effects (580). IGF-1 treatments have also been found to alleviate some of the symptoms of ALS (424). There is also evidence that melatonin and curcumin may have beneficial effects on reducing metal toxicity (591, 497, 580). Turmeric/curcumin has been found to reduce some of the toxic and inflammatory effects of toxic metals. Lithium supplements (lithium carbonate and lithium oratate) have been found to be effective in protecting neurons and brain function from oxidative and excitotoxic effects. A recent study demonstrated that combined treatment with lithium and valproic acid elicits synergistic neuroprotective effects against glutamate excitotoxicity in cultured brain neurons (590).

Doctors affiliated with Life Enhancement Foundation have developed a diet and supplementation protocol to reduce Parkinson's effects and delay the start time of daily levodopa therapy (page 1139) (580). Dietary considerations include avoidance of alcohol, sugar, red meats, cow's milk products, gluten, fried foods, aspartame, MSG, pesticides.

Some clinics have found root canals, cavitations, and amalgam tattoos to also be a factor in such autoimmune conditions and that treatment of them improves prognosis in recovery from these conditions (35, 437, 580).

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