

Depression and other Neurotransmitter Related Conditions - The
Mercury Connection

Bernard Windham (Ed.)

Contents

1 Depression and other neurotransmitter related conditions - the mercury connection	1
1.1 Introduction	1
1.2 Causes of Depression and Anxiety	3
1.3 Mercury exposure levels from amalgam and other sources	6
1.4 Toxic and immune reactive effects of mercury	7
1.5 The Danger of Vaccinations	10
1.6 Treatment of Depression	10
1.7 Anxiety Disorders include Panic Disorder, OCD, PTSD, Phobias, and General Anxiety Disorder	13
1.8 References	13

1 Depression and other neurotransmitter related conditions - the mercury connection

Depression and other Neurotransmitter Related Conditions - The Mercury Connection

B.Windham (Ed.)

1.1 Introduction

There are several types of depression and mood disorders, including neurotic depression, manic-depression, postpartum depression, anxious depression, agitated depression/panic attacks, obsessive-compulsive disorder, attention deficit disorder, etc. This review covers all of these disorders to some degree. Prescription and over the counter drugs that commonly are a factor in depressive disorders include Accutane, Acid blockers, Alprazolam, Ambien, Anabolic steroids, Beta-blockers, birth control pills, butalbital, chemotherapy, digoxin, hormone replacement drugs, pednisone, Quinalone antibiotics, Valium, etc., so this should be taken into account (20).

According to Dr. Gerald Klerman, based on National Institute of Health studies there has been a huge increase (over 500%) in the rate of depression and chronic neurological problems over the last 3 decades. A random sample of Oregon high school students found that over 16% had been diagnosed with depression (10). According to ECA samples, otherwise healthy people born in recent decades face a 10 fold increase in incidence of major depressive episodes compared to those sampled who were born in earlier decades. Over 6 million Americans over 65 suffer from major depression while another 5 million suffer from depressive symptoms (598). Every year, at least 230 million prescriptions for antidepressants are filled, making them one of the most prescribed drugs in the United States. The psychiatric industry itself is a \$330 billion industry.

Several factors appear to be contributing to this:

1. neurological birth defects and developmental conditions due to increased levels of vaccinations, fetal exposure to alcohol, tobacco smoke, drugs, *toxic metals*¹ such as lead, mercury, cadmium,

¹**Informative:** "Effects of Toxic Metals on Learning Ability and Behavior".

etc., other neurotoxic chemicals such as pesticides (552, 585), nitrates, etc., and other *endocrine system*² / hormonal system disrupting chemicals such as dioxins, phythalates (12), etc. Studies by the *National Academy of Sciences*³ indicate that these affect close to 40% of all children in the U.S., more in some populations than others

2. changes in dietary habits resulting in nutrient, vitamin, and mineral deficiencies or imbalances and blood sugar imbalances (596), and increased consumption of inflammatory excitotoxins such as aspartame, MSG, and high fructose corn syrup
3. stress in family and workplace environments.

Groups of primary care patients aged 18-65 years from 333 randomly chosen public or private clinics throughout the whole country of Poland, totaling 7289, coming for a regular visit were asked to participate in a study of the prevalence of depressive disorders (6). 71% of the sample were female. All patients filled in the Beck Depression Inventory (BDI). The prevalence of depressive disorders in the whole sample was 23.3%.

The number of people with anxiety disorders is close to the number with mood disorders (584). The primary types of anxiety disorders are phobias, panic attacks, generalized anxiety disorder (GAD), and obsessive-compulsive disorder (OCD). At least 20 million people are affected at some time by these conditions. Similar large numbers are affected by attention disorders, including attention deficit hyperactive disorder (ADHD), dyslexia, and schizophrenia (580, 584). “The Centers for Disease Control is out with a new survey that shows 5.4 million schoolchildren have been diagnosed with attention-deficit/hyperactivity disorder (AD/HD). That’s 10%.” In fact “from the years 2003 to 2007, the number of kids between four and ... 17 with AD/HD jumped by one million. That’s a 22% increase.” (180) However large surveys of elementary level student records finds much higher levels - with over 20% of elementary school boys in some areas being treated for ADD (143, 180). Similar levels of children have been found to have mood or anxiety disorders. At least 4% of adults have also been found to have ADHD symptoms (176). Studies have found that long term use of stimulant drugs commonly are not effective in the long run and causes significant adverse neurological and health effects (145, 172, 594b), There are more effective options available to deal with such conditions without such adverse effects including dealing with the underlying causes (172, 173, 176, 177) and diet, exercise, and supplement options that deal with underlying deficiencies (172).

Twenty-plus years of research on antidepressants, from the old tricyclics to the newer selective serotonin reuptake inhibitors (SSRIs) show that their benefit is hardly more than what patients get when they take a placebo (30, 31, etc.) Also that they don’t deal with some of the main causes of depression. Long-term increased stress hormones such as cortisol appear to often be a larger factor in depressive conditions than reduced serotonin (20, 594, etc.). In Britain, the agency that assesses which treatments are effective enough for the government to pay for stopped recommending antidepressants as a first-line treatment, especially for mild or moderate depression. A spokesperson for Pfizer, which makes Zoloft, added that the fact that antidepressants “commonly fail to separate from placebo is a fact well known by the FDA, academia, and industry.” Antidepressants are significantly more effective than a placebo in patients suffering only from the most severe depression (31). The serotonin-deficit theory of depression is built on a hypothesis that has little support. And a new drug, tianeptine, which is sold in France and some other countries (but not the U.S.), turns out to be as effective as Prozac-like antidepressants that keep the synapses well supplied with serotonin even though the mechanism of the new drug is to lower brain levels of serotonin. “If depression can be equally affected by drugs that increase serotonin and by drugs that decrease it”, says Kirsch (30c), “it’s hard to imagine how the benefits can be due to their chemical activity.” SSRIs often provide temporary improvement in some depressive conditions, but their effects usually don’t last over time

²**Internet:** “<http://www.flcv.com/endocrin.html>”.

³**Informative:** “Effects of Toxic Metals on Learning Ability and Behavior”.

and the often cause loss of sex drive and other adverse effects (20, 594b). Exercise, diet modification including reduction of sweets, and supplementing deficient vitamins and minerals have been found more effective treatments in the long term (20, 594, etc.) Supplements found to often help adrenal fatigue include, licorice extract, Panax ginseng, DHEA, Rhodiola, pantehine, and Eleuthero (20). Exercise routines found to be helpful include walking, yoga, and pilates (20). Since 1996, scientific researchers and doctors in clinical practice have been studying the effects of EMPowerplus mineral supplementation program on mental and mood disorders such as bipolar disorder. Results have been very encouraging and significant (522). Low cellular levels of the omega-3 oil DHA have also been found to be associated with bipolar disorder (21b).

1.2 Causes of Depression and Anxiety

There appears to be both a psychological/mind basis as well as physical/chemical basis for depression and anxiety. Nutritional deficiencies, environmental factors, methylation deficiencies, hormonal imbalances, and stress clearly can lead to depression and anxiety, but they also facilitate psychological factors (386, 493, 580, etc.). Based on clinical experience, anxiety and hyperventilation and panic attacks appear to often be related to a person burying their feelings about their circumstances (583). Depression often occurs where a person has suppressed anger, anger turned inward. Chronic anger has been found to be linked to increased risk of recurrent heart attacks and cardiac death (583b). The brain amygdala controls fear and anger and inflammatory conditions such as excess glutamate or stress have been found to reduce its control and to increase anger or fear (594). Other heart risks have also been linked to depression, anxiety, repressed anger and isolation or infrequent social interactions (582b). These factors, which lead to increased risks of heart disease, have been correlated with elevated cholesterol, blood pressure, variable heart rate plus increased arterial thickness and plaque accumulation. And studies estimate that 20 to 40 percent of all sudden cardiac deaths will be triggered by some type of acute emotional stressor (582c). Dealing with nutritional deficiencies and environmental factors, along with being honest with yourself, acknowledging anger or feelings rather than assigning blame, and doing what makes you feel good usually leads to reduced depression or anxiety (583a, 493).

The levels of brain neurotransmitters such as dopamine, norepinephrine, and serotonin, appear to be major factors in controlling moods, and appear to be affected by lifestyle, diet, philosophy, and environmental factors. Some are more susceptible to depression than others, and thus more affected by diet and environmental factors (580).

Chronic or acute brain inflammation appears to be a primary factor in depression. 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 depressive disorders and other chronic neurological conditions (592, 593, 598, 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, 594, 596-599) Overexposure to heavy metals like lead, mercury, copper, and zinc have been shown to induce anxiety or depression (386a, 586, 493, 494, 593, 594). Accumulation of mercury in the brain limbic system with resulting oxidative stress and inflammation has been found to commonly be a factor in depression (303).

Studies have found that oxidative stress from reactive oxygen species (such as caused by mercury and toxic metals) causes increased insulin resistance, whereas reducing reactive oxygen species lowers insulin resistance. (15). Insulin resistance has been found to be a significant factor in metabolic syndrome, cognitive decline, cardiovascular disease, depression, cancer, etc. Mercury and cadmium

inhibit magnesium and zinc levels as well as inhibiting glucose transfer. Reduced levels of magnesium and zinc are related to metabolic syndrome, insulin resistance, and brain inflammation and are protective against these conditions (599, 43). These are additional mechanisms by which mercury and toxic metals are factors in metabolic syndrome and insulin resistance and conditions such as diabetes, depression, etc. (43, 196, 338, 597, 15a). As documented later, for those who have several amalgam fillings, replacement of the amalgam greatly lowers mercury and toxic metal exposure, lowers reactive oxygen species and related damage, and brings significant improvement in the health of people with conditions caused by oxidative damage and insulin resistance. It has also been documented that supplementation with antioxidants such as green tea extract, bilberries, curcumin, N-acetyl-cysteine, etc. and supplements such as DHEA, Goat's Rue, cinnamon, quercetin, and vanadyl sulfate reduces inflammatory cytokine effects and lowers insulin resistance (15a).

Many studies have found toxic metal exposure such as mercury, lead, cadmium, and manganese commonly causes depression and other mood and neurological disorders (586). Young adults with higher blood lead levels are more likely to have major depressive disorder (MDD) or panic disorder, even if they have exposure to lead levels generally considered safe (586b)

The brain has elaborate protective mechanisms for regulating neurotransmitters such as glutamate, which is the most abundant of all neurotransmitters. When these protective regulatory mechanisms are damaged or affected, chronic neurological conditions such as Parkinson's can result (593). 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, 594). 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). Excess extracellular glutamate has been found to be strongly related to neurological conditions such as Alzheimer's, Parkinson's, ALS, OCD, depression, etc.(587b, 594). Psychotropic drugs that were thought to alleviate depression by raising monoamine levels have now been found to work by inhibiting glutamate receptors, thus reducing inflammation (587c). Hypericin, the active ingredient in St John's Wort used to treat depression also has been found to inhibit the release of glutamate into the brain and protect against excitotoxicity (588).

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. Microglia are the main immune cells in the brain. 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 (594, 598). This has been called immunoexcitotoxicity (594), which has been demonstrated to be a significant factor in many chronic psychiatric disorders including schizophrenia, PTSD, autism, suicides. 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, aspartame, other food additives, etc. (593, 594, 598, 600) Studies have shown that an increase in the inflammatory biomarker CRP (C-reactive protein) predicts the onset of depression in elderly people who had no prior history of depression (587a), and that depression is also linked with high levels of other inflammatory biomarkers - such as IL-6 (594). Inflammation also causes reduced levels and/or reduced effectiveness of the main brain-calming neurotransmitter, GABA (594). It is the balance between brain excitatory neurotransmitters like Glutamate and the calming neurotransmitters like GABA that allows normal functioning, and imbalances lead to psychiatric disorders.

Excitotoxic exposures and food additives are extremely common and affect most children, and can have major impacts on the brain over time, resulting in faulty brain-wiring, magnified aggressiveness,

rage reactions, obsessions, panic attacks, and other neurological and mood disorders (594). Studies have found that food-based excitotoxins can raise brain glutamate levels by as much as a factor of 50, causing inflammation and resulting in damage to the brain and brain regulatory mechanisms over time. This is especially true of the prefrontal cortex which controls judgement, regulates risk-taking, and suppresses socially inappropriate behavior. A study found that those with bipolar disorder have much lower levels than normal of the omega-3 DHA in the orbitofrontal cortex area of the brain which regulates behavior (21b). Those most susceptible to such excitotoxic effects are babies and the elderly, and also especially damaging for those who suffer from reactive hypoglycemia. Studies have found that eliminating such food-based excitotoxins in school diets resulted in greatly reduced behavioral problems and inattention problems (594b). The majority of the body's immune system is found in the digestive system, and inflammatory bowel diseases and food intolerances which induce inflammation in the intestines have also been found to be factors in brain inflammation and related psychiatric disorders (594b).

It had been thought that low serotonin levels in the brain were a major factor in depression, because inflammatory disorders (or infections) cause measured serotonin levels in the blood to fall significantly. However further studies have found that inflammation activates microglia (589a), which metabolize the serotonin precursor tryptophan into the highly brain-toxic excitotoxin quinolinic acid (589b); while also reducing the number of astrocytes, which metabolize tryptophan into a brain protective chemical kynurenine (589c). This imbalance has been found to be associated with psychiatric conditions such as depression and anxiety disorders (594). It has also been found that those with depression or anxiety disorders have low levels of a the brain-protective substance brain growth stimulator factor (BDGF) (589d). This is supplied by the astrocytes, which have been seen to be decreased in inflammatory conditions such as depression. Serotonin, which is also decreased, stimulates the release of BDGF. The mineral zinc has also been found to increase BDGF as well as the protective substance BDNF, and to reduce excitotoxicity (594), though its also possible to get too much zinc. Zinc deficiency can cause conditions such as depression, and zinc supplementation can improve depression in such circumstances. A persons zinc status can be determined through hair test or red blood cell test.

Hormone imbalance has been found to be a common factor in depression and learning disabilities (488, 12b), and thyroid imbalances have also been found to cause depression and ADHD (386a, 553, 20, 12b). Mercury and other endocrine disrupting chemicals such as phthalates have been found to commonly cause hypothyroidism (553, 12b). Imbalances in DHEA and cortisol may underlie depression, particularly when stress and obesity are present. Estrogen imbalances in post-menopausal women, low testosterone levels in some men, low DHEA levels, and hypothyroid conditions have been found to be common factors in depression. Subclinical hypothyroidism and/or the presence of thyroid peroxidase antibodies (TPOAb) has been found to be associated with subfertility, infertility, spontaneous abortion, placental abruption, preterm delivery, gestational hypertension, preeclampsia, postpartum thyroid dysfunction, depression (including postpartum depression), and impaired cognitive and psychomotor child development (7). It is recommended to suspect thyroid pathology if such conditions are present.

Most studies support a relationship between thyroid state and cognition, particularly slowed information processing speed, reduced efficiency in executive functions, and poor learning (11). Furthermore, hypo-thyroidism is associated with an increased susceptibility to depression and reductions in health-related quality of life. Controlled studies suggest that cognitive and mood symptoms improve with thyroid treatment, though the data are limited by diverse treatment methodologies. Functional neuroimaging data provide support for the mood and cognitive findings and treatment reversibility for both overt and subclinical hypothyroidism (11a). 94 patients with subclinical hypothyroidism and a control group were evaluated to determine the prevalence of psychiatric disorders (11b). The prevalence of depressive symptoms based on Beck's Scale among subclinical hypothyroidism patients was about 2.3 times higher than among controls (45.6% vs 20.9%, $p = 0.006$). Anxiety symptoms

were also more frequent in the hypothyroid group.

Postpartum thyroiditis (PPT) is the occurrence, in the postpartum period, of transient hyperthyroidism and/or transient hypothyroidism, with most women returning to the euthyroid state by 1 year postpartum (8a). However PPT frequently reoccurs in subsequent pregnancies and approximately 25% of women with a history of PPT will develop permanent hypothyroidism in the ensuing 10 years. The mean prevalence of PPT in 2 studies was 7.5%. Postpartum thyroiditis is an autoimmune disorder, and thyroid antibody-positive women in the first trimester have a 33% to 50% chance of developing thyroiditis in the postpartum period. There was a 70% chance of developing recurrent PPT after a first attack, and a 25% risk even in women who were only anti-TPO positive without thyroid dysfunction during the first postpartum period (8b). For this group of women with PPT, 46% had postpartum depression in one or more pregnancies.

In a study of effects of hypothyroid or thyroiditis during pregnancy, infants of women with hypothyroxinemia at 12 weeks' gestation had significantly lower scores on the Neonatal Behavioral Assessment Scale orientation index compared with normal subjects (9). Regression analysis showed that first-trimester maternal free thyroid hormone was a significant predictor of orientation scores. This study confirmed that maternal hypothyroxinemia constitutes a serious risk factor for neurodevelopmental difficulties that can be identified in neonates as young as 3 weeks of age. **Because of such evidence**, in November 2002, the American Association of Clinical Endocrinologists (AACE) recommended screening all women considering conception and/or all pregnant women in the first trimester for thyroid dysfunction (7b).

For a group of women with PPT, 46% had postpartum depression in one or more pregnancies (8b).

As will be shown, there is considerable evidence that depression/neurological problems can be caused by many physiological problems related to past toxic exposures or combinations of these. Where physiological problems are contributing factors, determination of the underlying cause from assessing the persons past medical history, diet, blood tests, hair tests, etc. can be useful to identifying and correcting any nutritional deficiencies or imbalances (386a) or identifying other problems to be dealt with. There is considerable evidence mercury exposure is among the most common significant exposures that commonly cause such effects, although many are also exposed to lead (586), arsenic, and pesticides (552, 585) that have similar effects and effects are *synergistic*⁴ or cumulative.

1.3 Mercury exposure levels from amalgam and other sources

Amalgam fillings have been documented to leak significant levels of mercury continuously due to high vapor pressure of mercury and galvanic action between mixed metals in the mouth (600, 602). The average person with several fillings gets significant exposure of mercury daily, much *more than from any other source*⁵ and more than that prescribed by U.S. Government health guidelines (602). Mercury in pregnant women is also documented to cross the placenta and accumulate in the *fetus*⁶ to levels higher than in the mother (603). Since mercury from amalgam fillings of a mother is also transmitted to nursing infants in significant amounts, mercury from their mom's dental fillings has been found to be the *largest source of mercury*⁷ to the fetus and a significant source of mercury in infants, which has produced developmental problems that affect children later in life (603). Young children also have been receiving significant levels of mercury (thimerosal which is used as a preserva-

⁴**Internet:** "<http://www.flcv.com/synergis.html>".

⁵**Informative:** "Dental Amalgam Mercury Solutions".

⁶**Informative:** "Infertility, Birth Defects, and Fetal Developmental Effects Related to Mercury from Amalgam Dental Fillings & Other Toxins".

⁷**Informative:** "Infertility, Birth Defects, and Fetal Developmental Effects Related to Mercury from Amalgam Dental Fillings & Other Toxins".

tive in vaccines) and large numbers have been found to be significantly adversely affected because of receiving larger numbers of vaccinations, especially at very early ages before the blood-brain barrier matures (602). People also get significant prenatal and postnatal exposures to other toxic metals such as lead, arsenic, cadmium, aluminum, etc. which have also been found to commonly cause significant neurological effects (586, 604). The top 3 toxic substances affecting large numbers of people in the U.S. adversely according to EPA/ATSDR are mercury, lead, and arsenic. (600, 604).

A 2009 study found that inorganic mercury levels in people have been increasing rapidly in recent years (543b). It used data from the U.S. Centers for Disease Control and Prevention's National Health Nutrition Examination Survey (NHANES) finding that while inorganic mercury was detected in the blood of 2 percent of women aged 18 to 49 in the 1999-2000 NHANES survey, that level rose to 30 percent of women by 2005-2006. Surveys in all states using hair tests have found dangerous levels of mercury in an average of 22% of the population, with over 30% in some states like Florida and New York (543c). A large U.S. Centers for Disease Control epidemiological study, NHANES III, found that those with more amalgam fillings (more mercury exposure) have significantly higher levels of chronic health conditions (543). The conditions in which the number of dental amalgam surfaces were most highly correlated with disease incidence were MS, epilepsy, migraines, mental disorders, diseases of the nervous system, disorders of the thyroid gland, cancer, and infectious diseases (543).

1.4 Toxic and immune reactive effects of mercury

Mercury is neurotoxic (kills or damages brain and nerve cells): (19, 27, 34, 36, 43, 69, 70, 147, 148, 175, 207, 211, 273, 291, 295, 327, 329, 301, 303, 395, 600/ 39, 262, 274, 303); generates high levels of reactive oxygen species (ROS) and oxidative stress, depletes glutathione and thiols causing increased neurotoxicity from interactions of ROS, glutamate, and dopamine (13, 56, 98, 102, 126, 145, 169, 170, 184, 213, 218, 219, 250, 257, 259, 286, 290, 291, 302, 324, 326, 329, 594, 600); kills or inhibits production of brain tubulin cells (66, 67, 161, 166, 207, 300); inhibits production of neurotransmitters by inhibiting: calcium-dependent neurotransmitter release (372), dihydropteridine reductase (27, 122, 257), nitric oxide synthase (259), blocking neurotransmitter amino acids (438, 601), and effecting phenylalanine, tyrosine and tryptophan transport to neurons) (34, 122, 126, 257, 285, 288, 333, 438, 495/255, 333). Toxic metals as well as genetic factors commonly cause systemic methylation deficiencies (88), which are documented to commonly be a factor in chronic conditions such as depression, autism, etc. (386a)

Numerous studies have found long-term chronic low doses of mercury cause neurological, memory, behaviour, sleep, and mood problems (5, 72, 74, 107, 109, 290, etc.). Neurological problems are among the most common and serious effects of mercury, and include memory loss, moodiness, depression, anger and sudden bursts of anger/rage, self-effacement, suicidal thoughts, lack of strength/force to resolve doubts or resist obsessions or compulsions, etc. Many studies of patients with major neurological diseases have found evidence amalgam fillings may play a major role in development of conditions such as depression (94, 107, 109, 212, 222, 229, 233, 285c, 294, 317, 320, 322, 372, 374, 453), schizophrenia (34, 35, 295, 601), memory problems (70, 94, 212, 222, 600), and other more serious neurological diseases such as MS, ALS, Parkinson's, and Alzheimer's (13, 33, 66, 98, 207b, 330, 331, 424, 438, 483, 600). Some factors that have been documented in depression are low serotonin levels, abnormal glucose tolerance (hypoglycemia), and low folate levels (480-83), which mercury has also been found to be a cause of. Occupational exposure to mercury has been documented to cause depression and anxiety (534). Acute exposure to mercury vapor has been found to cause chronic depression, anxiety, and obsessive-compulsive behavior (487). One mechanism by which mercury has been found to be a factor in aggressiveness and violence is its documented inhibition of the brain transmitter acetylcholinesterase (175, 451, 465, 254). Low serotonin levels and/or hypoglycemia have also been found in the majority of those with impulsive and violent behavior (481, 482).

Mercury (and other toxic metals) has been found to accumulate in the pineal gland and reduce melatonin levels, which is thought to be a significant factor in mercury's toxic effects (569). Melatonin has found to have a significant protective action against methyl mercury toxicity, likely from antioxidative effect of melatonin on the MMC induced neurotoxicity (567). Disrupted sleep from low melatonin, or 'Seasonal Affective Disorder' with excessive melatonin production, can result in depression (386a). Melatonin is important in regulating mood and improving sleep and increasing quality of life by regulating your body's circadian rhythms-while scientific evidence indicates that it has helpful anti-inflammatory and antioxidant properties that can support your heart, too (564).

There is also evidence that mercury affects neurotransmitter levels which have effects on conditions such as depression, mood disorders, ADHD, etc. There is evidence that mercury can block the dopamine- β -hydroxylase (DBH) enzyme (571). This enzyme synthesizes noradrenaline, and low noradrenaline can cause fatigue and depression. Mercury molecules can block all copper-catalysed dithiolane oxidases, such as coproporphyrin oxidase and DBH. Mercury and other toxic metals have been found to accumulate in the pineal gland and reduce melatonin levels, which is thought to be a significant factor in mercury's toxic effects (569).

There is evidence that mercury can block the dopamine-beta-hydroxylase (DBH) enzyme (571). DBH is used to make the noradrenaline neurotransmitter and low noradrenaline can cause fatigue and depression. Mercury molecules can block all copper catalyzed dithiolane oxidases, such as coproporphyrin oxidase (260) and DBH.

Workers occupationally exposed to mercury at levels within guidelines have been found to have impairment of lytic activity of neutrophils and reduced ability of neutrophils to kill invaders such as candida (285, 404). The balance of yeasts found in the intestine can be a factor in neurological conditions such as depression (386a, 404). Evidence suggests *Candida albicans* may activate depressive symptoms and fatigue by promoting ethanol production, a known central nervous system depressant. Behavior changes are also associated with *Candida's* inherent toxin - candidotoxin - and/or by its tendency to compete with the host organism for essential dietary nutrients.(460) Immune Th1 cells inhibit candida by cytokine related activation of macrophages and neutrophils. Development of Th2 type immune responses deactivate such defenses (404b, 285). Mercury inhibits macrophage and neutrophil defense against candida by its affects on Th1 and Th2 cytokine effects (181, 285). *Candida* overgrowth results in production of the highly toxic candidotoxin and ethanol which are known to cause fatigue, toxicity, and depressive symptoms (460).

Mercury causes decreased lithium levels, which is a factor in neurological diseases such as depression and Alzheimer's. Lithium protects brain cells against excess glutamate and calcium, and low levels cause abnormal brain cell balance and neurological disturbances (280, 294, 333, 33, 56). Medical texts on neurology (27, 295) point out that chronic mercurialism is often not recognized by diagnosticians and misdiagnosed as dementia or neurosis or functional psychosis or just "nerves". "Early manifestations are likely to be subtle and diagnosis difficult: Insomnia, nervousness, mild tremor, impaired judgment and coordination, decreased mental efficiency, emotional lability, headache, fatigue, loss of sexual drive, depression, etc. are often mistakenly ascribed to psychogenic causes". Very high levels of mercury are found in brain memory areas such as the cerebral cortex and hippocampus of patients with diseases with memory related symptoms (158, 34, 207, etc).

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 neurological conditions such as autism, schizophrenia, manic-depressive, ADD, depression (294, 375, 408, 438, 601). For example mercury has been found to strongly inhibit the activity of dipeptyl peptidase (DPP IV) which is required in the digestion of the milk protein casein (411, 412, 602). Studies involving a large sample of schizophrenic or autistic patients found that over 90% of those tested had high levels of the milk protein beta-casomorphin-7 in their blood and urine and defective enzymatic processes for digesting milk protein (410). Similar findings have been confirmed for ADD

and mania patients. Elimination of milk products from the diet has been found to improve these conditions in large numbers of patients (5). Such populations have also been found to have high levels of mercury and to recover after mercury detoxification. (413, 60, 313, 600). As mercury levels are reduced the protein binding is reduced and ment in the enzymatic process occurs (5). Additional cellular level enzymatic effects of mercury's binding with proteins include blockage of sulfur oxidation processes and neurotransmitter amino acids (33, 114, 438, 5), enzymatic processes involving vitamins B6 and B12 (418, 5), effects on the cytochrome-C energy processes (232, 35), along with mercury's adverse effects on cellular mineral levels of calcium, magnesium, zinc, chromium, and lithium (43, 96, 198, 333, 386, 427, 432, 484, 38).

When a pathological state exists, the body's finely balanced symbiosis may be damaged and cease to function normally. Beneficial essential bacteria may be damaged, causing the malabsorption of critical vitamins and minerals. If the damage is extensive and/or long lasting, pathogens including pathogenic yeast and gram negative bacilli will begin to fill the vacuum left by the healthy bacteria. The metabolism of these pathogens is different and foods are no longer broken down in the same way. Proteins that previously would be broken down to their constituent amino acids are only partially digested, leaving long chains of amino acids called peptides. Our entire body is built from proteins, which are themselves built from chains of peptides. Certain peptides are extremely bioactive i.e they interact strongly with other proteins in the body. Mercury and toxic metals cause dysbiosis and inhibits the function of the enzymes needed to digest gluten and casein, resulting in peptides in the blood which have significant neurological effects including depression, anxiety, and schizophrenia (404, 405). A side effect of dysbiosis (incorrect gut microorganisms) is that the gut becomes leaky i.e it passes larger molecules than would normally be the case. Thus peptides, which should normally be broken down to amino acids, leave the gut and enter the blood stream intact, where they are delivered to other organs. *Casein and Gluten*⁸, proteins and mixture of proteins common in many foods break down to form very potent opio-peptides when acted on by certain pathogenic bacteria. These peptides have a narcotic action and act on opiate receptors in the brain, triggering major changes in brain function including depression, anxiety, schizophrenia, etc.(406, etc.) Certain pathogens more plentiful during dysbiosis also have been found to methylate mercury to its organic form which is more readily taken up by the blood and redistributed. Taking antibiotics is another cause of such dysbiosis.

Studies have shown a significant association between hypothyroidism and mood disorders such as depression (391, 8). Mercury from dental amalgam has been documented to cause hypothyroidism (50, 91, 212, 222, 369, 382, 390, 35ab). The majority of patients tested with hypothyroidism or thyroiditis and treated with dental amalgam replacement significantly improved after replacement (91, 369, 303).

Numerous studies have found long term chronic low doses of mercury cause neurological, memory, behavior, sleep, and mood problems (34, 69, 70, 71, 72, 74, 95, 107, 108, 109, 115, 119, 140, 141, 196, 199, 222, 252, 255, 257, 258, 282, 290, 303, 304]. Neurological effects have been documented at very low levels of exposure (urine Hg ; 4 $\mu\text{g}/\text{L}$), levels commonly received by those with amalgam fillings (290). One of the studies at a German University (199) assessed 20,000 people. There is also evidence that fetal or infant exposure causes delayed neurotoxicity evidenced in serious effect at middle age (255). Studies of groups of patients with amalgam fillings found significantly more neurological, memory, mood, and behavioral problems than the control groups. (34, 107, 108, 109, 140, 141, 196, 199, 222, 290]. Increased mercury levels from amalgam are documented to cause increased neurological problems related to lowered levels of neurotransmitters dopamine, serotonin, norepinephrine, and acetylcholinesterase (35, 107, 140, 141, 175, 251, 254, 288, 290, 296, 305, 372, 451, 465, 412). The reduced neurotransmitter levels in those with amalgam appear to be a factor encouraging smoking since nicotine increases these neurotransmitter levels and a much higher

⁸**Internet:** "<http://www.flcv.com/autismgc.html>".

percentage of those with amalgam smoke than in those without amalgam (141).

Based on thousands of clinically followed cases by doctors, replacement of amalgam fillings resulted in the cure or significant improvement in the majority of cases for: depression (35, 94, 95, 107, 222, 271, 294, 212, 229, 230, 233, 303, 317, 320, 322, 376, 407), schizophrenia (294, 34, 35), insomnia (94, 95, 212, 222, 271, 304, 317, 322, 376, 407), anger (212, 233, 320, 407, 102), anxiety & mental confusion (94, 95, 212, 222, 229, 233, 271, 304, 317, 320, 322, 407, 57), memory disorders (94, 95, 222, 304, 407). For example, in a study of amalgam replacement for 56 persons who suffered from chronic depression, 16 had the condition eliminated and 34 had significant improvement after a year or 4 years (95).

One of the most common causes of depression and mood disorders has been documented to be past toxic exposures such as mercury or pesticides (585), and the majority treated for these at clinics that deal with such conditions have either recovered or shown significant improvement (600, 601, 552). Amalgam dental fillings have been found the most common source of such toxic exposures, with mercury thimerosal from vaccinations also affecting millions of children (600, 601). Many doctors treating depression and mood disorder conditions related to toxic exposures also usually recommend supplementing the deficient essential minerals that mercury affects by affecting cell membrane permeability and blocking cellular enzymatic processes, often obtaining a hair element test to determine imbalances and needs (560, 600). The body requires adequate, but not excessive, amounts of trace minerals and nutrients for proper functioning. Under certain conditions, excesses or deficiencies of many of these elements can set off symptoms of depression (560). Subnormal levels of zinc, for example, are associated with treatment resistant depression (561). And deficiencies of magnesium can provoke a wide range of psychiatric symptoms related to depression, ranging from apathy to psychosis (562). Research on manic patients, on the other hand, has revealed elevated vanadium in the hair—significantly higher levels than those measured in both a control group and a group of recovered manic patients (563).

1.5 The Danger of Vaccinations

Chronic over activation of the immune system has been found to be a major factor in neurological and cardiovascular conditions (593, 598, etc.) Immune adjuvants in vaccines including aluminum, mercury, special lipids, and even MSG in some cause activation of the immune system which can last for months. This causes inflammation of the brain that is magnified by each additional vaccination with more immune adjuvants. The high number of vaccinations in a short period of time has been found to be a major cause of autism spectrum and other inflammatory conditions in children, and also to be major factors in inflammatory conditions of older adults such as depression, Alzheimer's, Parkinson's, etc. (593, 598, 601, 600, etc.) Flu vaccinations in those over 55 years of age have been found to increase the risk of Alzheimer's by over 500%, along with increased risk of major depression (598).

1.6 Treatment of Depression

Anyone with depression should be examined and tested for toxic metal exposure or exposures to other toxics. Detoxification should be carried out as appropriate. Those with several amalgam fillings or metal crowns over amalgam are getting high exposures of extremely toxic substances that are highly inflammatory so should have the problematic dental work replaced. Everyone should also be checked for problematic root-canal teeth and jawbone cavitations, which likewise are highly inflammatory and can have major impacts on the immune system and health (605, 303). Reducing glutamate levels and blocking glutamate receptors can significantly improve depression (592, 593, 598).

Diet and lifestyle are important factors in preventing or controlling depression. One should avoid

alcohol, sugar, caffeine, and inflammatory substances such as MSG or aspartame, high-fructose corn syrup, fluoride, pesticides, aluminum in foods, mercury fillings, most vaccinations (esp. flu vax.), etc. (580, 594, 598). Stress causes increased stress hormones and inflammation, which can be major factors in depression and anxiety disorders.(594b) Reduce stress and get regular exercise. Yoga and meditation have been found to be helpful for many. Studies have found that dietary choices play a major role in psychological well being, so proper diet is important (594). Behavioral problems and *criminal behavior*⁹ are correlated to toxic or excitotoxic exposures and diet choices (594b). Properly formulated nutritional supplements and diet modification have been found to be effective in treating ADHD, depression, and anxiety disorders (522, 20, 593, 594).

Studies and clinical experience have found that diet plays a role in depression and diet measures commonly avoid, cure, or significantly improve depression (565, 566, 580, 583, 591, 20). B Vitamins and magnesium deficiencies have been found to be factors in depression and anxiety. Supplementaion to assure proper levels is beneficial in treatment (565, 566, 583, 20). Many people, particularly women over 65, have B-12 deficiencies and respond dramatically to injections of the vitamin. But all B vitamins can boost mood; they work by facilitating neurotransmitter function. Other pluses: B vitamins are critical for preventing other maladies, including heart disease, cancer, and Alzheimer's. Suggested Dosage: Take at least 800 micrograms of folate, 1,000 mcg of B-12, and 25 to 50 milligrams of B-6. A B-complex vitamin should do the trick, says Hyman, and if you're depressed, take more. Take them in combination because otherwise one can mask another B vitamin deficiency (565).

The supplement 5-HTP has been shown by many studies and clinical experience to often be effective in treating or controlling depression (530, 20). Double blind studies have found 5-HTP to be as effective as SSRIs and other types of antidepressives at treating depression. Tryptophan likewise has been found beneficial in some with depression (495). But studies have also cast doubt on serotonin levels as the main cause in depression and found both 5-HTP and SSRIs have limited effect on many with depression. SSRIs appear to be attempting to suppress symptoms related to one type of imbalance found in many with depression rather than the underlying causes.

SAMe (400-1600 mg) and Inositol have been found to be effective in treating depression and anxiety with effectiveness at least as much as pharmaceutical antidepressants and much less adverse effects (565, 566, 580, 590, 20). SAMe is an amino acid combination produced by humans, animals, and plants. Supplements come from a synthetic version produced in a lab that has shown a lot of promise in European studies. May affect the synthesis of neurotransmitters. Has fewer side effects than 5-HTP and fewer drug interactions than Saint-John's-wort. Dosage: Can range from 400 to 1, 200 mg a day, though high doses can cause jitteriness and insomnia. Risks: People with bipolar disorder shouldn't use it without supervision because it can trigger mania. (566) Inositol has been found to be effective for treating OCD, panic disorders, and bipolar depression (591), with effectiveness at least as much as SSRIs and less adverse effects (591). St. Johns Wort (300 mg x 3) also has been found effective for many (565, 580, 20) and is one of the best-known remedies. Best for mild to moderate depression. Suggested Dosage (566): Start on a dose of 300 mg (standardized to 0.3 percent hypericin extract) two to three times a day, depending on severity of depression; it can take three weeks to show benefits. Risks: It may interfere with up to half of all drugs, prescription and over-the-counter.

Amino acids are the building blocks of neurotransmitters; **5-HTP** is the most popular. Taking it can elevate mood in cases of depression, anxiety, and panic attacks, and relieve insomnia. Increases production of the neurotransmitter serotonin. Suggested Dosage (566): Start with a low dose, 50 mg two to three times a day; after two weeks, increase the dose to 100 mg three times a day. Risks: Mild nausea or diarrhea. Before starting, get off antidepressants (under a doctor's supervision); the combination can produce an overload of serotonin. Tyrosine is another amino acid found to often be useful in overcoming depression (20).

⁹**Internet:** "<http://www.flcv.com/violence.html>".

Lower levels of fish oil (EPA) has been found to be significantly related to depression. (20) Elderly people have been found to be of special risk regarding depression. Studies have found higher levels of EPA to be associated with lower likelihood of depression or dementia (580b) in the elderly. Theoflavins from black or green tea and curcumin (turmeric) have also been found to be significantly effective against inflammation, which is a major factor in depression (580). Poor digestion results in poor mineral and nutrient absorption and is a factor in many chronic conditions. Digestive problems often increase with aging, due to reductions in digestive enzyme production and availability as well as increased proliferation of pathogenic organisms. Supplementation with digestive enzymes and probiotics often significantly improves digestion and improves digestive related conditions (580).

Adrenal fatigue and long-term increased stress hormones such as cortisol have been found to be common factors in depressive disorders (20). Prescription hydrocortisone can help in the short term, but supplements found to often help adrenal fatigue include, licorice extract, Panax ginseng, DHEA, Rhodiola, pantehine, and Eleuthero (20). Exercise routines found to be helpful with depressive disorders include walking, yoga, and pilates (20). Deep breathing exercises and meditation have also been found to be beneficial in alleviation of depressive disorders (20).

Hypothyroidism is also often a factor in depressive conditions, and treatments such as mercury detoxification and supplements such as iodine, zinc, copper, selenium, tyrosine, vitamins C, E, B12, and Ashwagandha extract are often helpful when this is a factor (20).

Birth control pills and artificial hormone replacement drugs can deplete nutrients such as vitamin B6 and create estrogen/progestin imbalances, which can be a factor in depression. Supplementing with Vitamin C, multivitamin B complex, magnesium, iodine, and tyrosine have been found to be helpful in this situation (20).

Essential fatty acids (EPA/DHA) benefits are among the best documented. (20, 21, 22) The reason they're so effective is EFAs are part of every cell membrane, and if those membranes aren't functioning well, then neither is your brain. Suggested Dosage (566): For depression, take at least 2,000 to 4,000 mg of fish oil a day. Should be purified or distilled so it's free of heavy metals. Risks: Very safe, albeit unstable. Since it can oxidize in your body, take it along with other antioxidants, like natural vitamin E (400 IUs a day).

DHEA is a hormone marketed in Europe specifically for postmenopausal depression, though it may be helpful for other forms as well. It has been used in conjunction with estrogen to treat hot flashes. Suggested Dosage (566): 10 to 200 mg a day. Risks: Any hormonal supplement not properly monitored has the potential to increase cancer risk.

Rhodiola rosea is considered an adaptogen, which means it can increase your resistance to a variety of stressors. It may be good for mild to moderately depressed patients (20). Suggested Dosage (566): Take 100 to 200 mg three times a day, standardized to 3 percent rosavin. Risks: More than 1, 500 mg a day can cause irritability or insomnia.

Other nutrients found to cause depression when low or to usually be low in depression or to be effective additions in treating depression include ginkgo biloba, DHEA, natural progesterone, pregnenolone, DMAE, L-Carnitine, NADH, Phenylalanine, Folic Acid, Vit B12 (cobalamine), B6, other B vitamins, choline, vit D, vit C, potassium, testosterone in men over 40 (580, 582, 565, 566). A product that contains several of these nutrients is Happiness 1-2-3 (vit B complex, magnesium, St.Johns Wort, L-Theanine, 5-HTP, magnolia) (583). Other companies referenced here have similar combinations (580, 582).

1.7 Anxiety Disorders include Panic Disorder, OCD, PTSD, Phobias, and General Anxiety Disorder

(584) As previously noted, anxiety or panic disorder can be related to not acknowledging or burying feelings (583). Panic disorder is characterized by repeated episodes of intense fear. Affects 3 to 6 million. Obsessive-Compulsive Disorder (OCD) is characterized by anxious thoughts and uncontrollable ritualistic behavior. Affects 2% of the population. Some studies have suggested OCD patients usually have high glutamate levels, which overexcites areas of the brain (581). Post-Traumatic Stress Disorder (PTSD) is a debilitating illness resulting from a traumatic event or events. It affects a large number of people. Phobias are irrational fears of things or situations. Affects over 10% of the population. Generalized Anxiety Disorder (GAD) is chronic, daily worrying about health, finances, work, family, etc. Stress is a psychological and physical response to the demands of daily life that exceed the person's ability to cope successfully. Stress can have physical effects prolonged stress can have debilitating effects. Two conventional non-pharmaceutical treatments for anxiety are behavioral therapy (breathing techniques, exposure therapy, etc.) and cognitive therapy (modification of thinking patterns).

As previously note, environmental toxins can be a factor in causing nutritional deficiencies, imbalances, and inflammation related to anxiety disorders and reductions in exposures have been found to be beneficial. Hypoglycemia may be a factor in some anxiety disorders - eat more frequent small quantities including protein, nuts, etc. Many are adversely affected by stimulants such as caffeine. Irregular or insufficient sleep patterns can be a significant factor. Regular exercise is generally beneficial in anxiety disorders. Massage therapy, including aromatherapy is often helpful, along with meditation and deep breathing exercises. Music, yoga, muscle relaxation techniques, biofeedback, etc. are also often helpful.

Deficiency of B vitamins and magnesium have been found to be common factors in anxiety disorders. (583). Adapton (fish oil) is commonly used helpful treatment for anxiety in Europe. (580) Very successful for fatigue, etc. Theanine (green tea extract) - calming and lowers blood pressure. (580, 582, 583)

Ginseng has been found effective for many post-menopausal women's anxiety, fatigue, depression. Reishi has helped some and Ashwagunda (Indian Ginseng). (580) A product with several of these nutrients is Calming Balance (vit B complex, magnesium, L-Theanine, Magnolia extract). (583). The other sources referenced here have similar products (580, 582).

1.8 References

(5) Consensus paper of the WFSBP Task Force on Biological Markers:

Biological Markers in Depression, R. MOSSNER, O. MIKOVA,, E. KOUTSILIERI, M. SAOUD, A-C EHLIS1, N. MULLER5, A. J. FALLGATTER1 & P. RIEDERER

The World Journal of Biological Psychiatry, 2007; 8(3): 141_174; wfsbp-verband.globit.com/fileadmin/pdf

(6) The study of the prevalence of depressive disorders in primary care patients in Poland], Wiad Lek. 2007; 60(3-4):109-13. Drózd W, Wojnar M, Araszkiwicz A, Nawacka-Pawlaczyk D, Urbański R, Cwiklińska-Jurkowska M, Rybakowski J

¹⁰**Internet:** "http://wfsbp-verband.globit.com/fileadmin/pdf/guides/WFSBP_Consensus_Paper_Biological_Markers_in_Depr

(7) Thyroid malfunction in women; *Ginecol Obstet Mex.* 2001 May; 69:200-5, Zárata A, Basurto L, Hernández M; & (b) Clinical controversies in screening women for thyroid disorders during pregnancy. Wier FA, Farley CL. *J Midwifery Womens Health.* 2006 May-Jun; 51(3):152-8.

(8) Postpartum thyroiditis. *Best Pract Res Clin Endocrinol Metab.* 2004 Jun; 18(2):303-16. Stagnaro-Green A; & Recognizing, understanding, and treating postpartum thyroiditis. *Endocrinol Metab Clin North Am.* 2000 Jun; 29(2):417-30, ix. Stagnaro-Green A; & (b) Postpartum depression and thyroid antibody status. *Thyroid.* 1999 Jul; 9(7):699-703, Harris B.

(9) Neonatal effects of maternal hypothyroxinemia during early pregnancy. *Pediatrics.* 2006 Jan; 117(1):161-7. Kooistra L, Crawford S, van Baar AL, Brouwers EP, Pop VJ.

(10) *Science News*, Vol 158, Oct 14, 2000

(11) Neuropsychiatric aspects of hypothyroidism and treatment reversibility. *Minerva Endocrine.* 2007 Mar; 32(1):49-65, Davis JD, Tremont G; & (b) Subclinical hypothyroidism: psychiatric disorders and symptoms. *Rev Bras Psiquiatr.* 2007 Jun; 29(2):157-9, Almeida C, Brasil MA, Costa AJ et al.

(12) Prenatal phthalate exposure is associated with childhood behavior and executive functioning. Engel SM, Miodovnik A, Canfield RL, Zhu C, Silva MJ, Calafat AM, Wolff MS. *Environ Health Perspect.* 2010 Apr; 118(4):565-71.

(13) S.Hussain et al, "Mercuric chloride-induced reactive oxygen species and its effect on antioxidant enzymes in different regions of rat brain", *J Environ Sci Health B* 1997 May; 32(3):395-409; & S.Tan et al, "Oxidative stress induces programmed cell death in neuronal cells", *J Neurochem*, 1998, 71(1):95-105. & J.S. Bains et al, "Neurodegenerative disorders in humans and role of glutathione in oxidative stress mediated neuronal death", *Brain Res Rev*, 1999, 25(3):335-58; & P.Bulat, "Activity of Gpx and SOD in workers occupationally exposed to mercury", *Arch Occup Environ Health*, 1998, Sept, 71 Suppl:S37-9; & Stohs SJ, Bagchi D. Oxidative mechanisms in the toxicity of metal ions. *Free Radic Biol Med* 1995; 18(2): 321-36; & Pocernich CB, Cardin AL, Racine CL, Lauderback CM, Allan Butterfield D. Glutathione elevation and its protective role in acrolein-induced protein damage in synaptosomal membranes: relevance to brain lipid peroxidation in neurodegenerative disease. *Neurochem Int* 2001 Aug; 39(2):141-9;

(15) Insulin Resistance: the Surprising Cause Behind This Highly Destructive Process, *Vitamin Research News*, Vol 22, No. 6, June 2008; & Houstis N, Rosen ED, Lander ES, Reactive oxygen species have a causal role in multiple forms of insulin resistance, *Nature*, 2006, Apr 13; 440(7086): 944-8; & Meigs JB, Larson MG, et al, Association of oxidative stress, insulin resistance, and diabetes risk phenotypes: the Framingham Offspring Study, *Diabetes Care*. 2007, Oct; 30(10):2539-35.

(20) The 24 Hour Pharmacist, S. Cohen, Rodale Books, 2007

(21) Horrocks LA, Yeo YK: Health benefits of docosahexaenoic acid (DHA). *Pharmacol Res* 1999; 40(3):211-25; & (b) DHA levels in people with bipolar disorder, McNamara RK, et al, *Psychiatry Res*, 2008, 160: 285-299; & (c) Hibbeln JR, et al.: Do plasma polyunsaturates predict hostility and violence? *World Rev Nutr Diet* 1996; 82:175-86.

(22) Improved behavior and attention associated with higher levels of cellular omega-3 levels, Kirby A, et al, *Res Dev Disabl* 2010, 31:731-742; & (b) Excess omega-6 oils correlated to depressive disorders, Coklin SM, et al, *Psychosom Med* 2007, 69: 932-934.

(30) Irving Kirsch and Guy Sapirstein, *Prevention & Treatment*, 1998 & Listening to Prozac but Hearing Placebo. 2002; & *The Emperor's New Drugs: Exploding the Anti-depressant Myth*. 2010

(31) *The Journal of the American Medical Association (JAMA)* January 6, 2010; 303(1):47-53

(33) B. Windham, Multiple Sclerosis (MS): the mercury connection; www.flcv.com/ms.html¹¹

¹¹ "... gaia/en/vital/medoral/fatosmer/ms_dams.htm".

(34) Patrick Störtebecker, Associate Professor of Neurology, Karolinska Institute, Stockholm. Mercury Poisoning from Dental Amalgam - A Hazard to the Human Brain, Bio-Probe, Inc. ISBN: 0-941011001-1

(35).Huggins HA, Levy, TE, Uniformed Consent: the hidden dangers in dental care, 1999, Hampton Roads Publishing Company Inc; & Hal Huggins, Its All in Your Head, 1997; & Center for Progressive Medicine, 1999, <http://www.hugnet.com>

(43) B.Rajanna et al, "Modulation of protein kinase C by heavy metals", *Toxicol Lett*, 1995, 81(2-3):197-203; & A.Badou et al, "HgCl₂-induced IL-4 gene expression in T cells involves a protein kinase C-dependent calcium influx through L-type calcium channels", *J Biol Chem*. 1997 Dec 19; 272(51):32411-8., &

(50) (a) Sin YM, Teh WF, Wong MK, Reddy PK - "Effect of Mercury on Glutathione and Thyroid Hormones" *Bulletin of Environmental Contamination and Toxicology* 44(4):616-622 (1990); & (b) J.Kawada et al, "Effects of inorganic and methyl mercury on thyroidal function", *J Pharmacobiodyn*, 1980, 3(3):149-59; & (c) Ghosh N. Thyrotoxicity of cadmium and mercury. *Biomed Environ Sci* 1992, 5(3): 236-40; & (d) Goldman, Blackburn, *The Effect of Mercuric Chloride on Thyroid Function of the Rat*, *Toxicol and Applied Pharm* 1979, 48: 49-55; & (e) Kabuto M - "Chronic effects of methylmercury on the urinary excretion of catecholamines and their responses to hypoglycemic stress" *Arch Toxicol* 65(2):164-7 (1991)

(61) E.Lutz et al, "Concentrations of mercury in brain and kidney of fetuses and infants", *Journal of Trace Elements in Medicine and Biology*, 1996, 10:61-67; & G.Drasch et al, "Mercury Burden of Human Fetal and Infant Tissues", *Eur J Pediatr* 153:607-610, 1994;

(66) B.Windham, *Alzheimer's Disease: the mercury connection*, www.flcv.com/alzhg.html¹²; (over 150 peer-reviewed medical studies)

(72) D.L.Smith, "Mental effects of mercury poisoning", *South Med J* 71:904-5, 1978.

(74) A.C.Bittner et al, "Behavior effects of low level mercury exposure among dental professionals", *Neurotoxicology & Teratology*, 1998, 20(4):429-39.

(88) Activation of methionine synthase by insulin-like growth factor-1 and dopamine: a target for neurodevelopmental toxins and thimerosal, Waly M, Olteanu H, Deth RC et al, *Mol Psychiatry*. 2004 Apr; 9(4):358-70

(91) B.Lindqvist et al, "Effects of removing amalgam fillings from patients with diseases affecting the immune system", *Med Sci Res* 24(5): 355-356, 1996.

(94) F.Berglund, Case reports spanning 150 years on the adverse effects of dental amalgam, Bio-Probe, Inc., Orlando, FL, 1995; ISBN 0-9410011-14-3(245 cured)

(95) Lichtenberg, HJ "Elimination of symptoms by removal of dental amalgam from mercury poisoned patients", *J Orthomol Med* 8:145-148, 1993; & Lichtenberg H, "Symptoms before and after proper amalgam removal in relation to serum-globulin reaction to metals", *Journal of Orthomolecular Medicine*, 1996, 11(4): 195-203. (119 cases)

(98) B.Windham, *Parkinson's Disease: the mercury connection*; www.flcv.com/parkins.html¹³ (over 100 peer-reviewed medical studies)

(107) R.L.Siblerud et al, "Psychometric evidence that mercury from dental fillings may be a factor in depression, anger, and anxiety", *Psychol Rep*, v74, n1, 1994; & *Amer. J. Of Psychotherapy*, 1989; 58:575-87; & Poisoning and Toxicology Compendium, Leikin & Palouchek, Lexi-Comp, 1998, p705

¹²**Informative:** "Alzheimer's Disease and Other Autoimmune Degenerative Conditions: the Mercury Connection".

¹³**Informative:** "Toxic Exposures and Parkinsons: the Mercury Connection".

(108) M.Henningsson et al, "Defensive characteristics in individuals with amalgam illness", *Acta Odont Scand* 54(3): 176-181, 1996.

(109) Y.X. Liang et al, "Psychological effects of low exposure to mercury vapor", *Environmental Med Research*, 60(2): 320-327, 1993; & T.Kampe et al, "Personality traits of adolescents with intact and repaired dentitions", *Acta Odont Scand*, 44:95-, 1986; & R.Kishi et al, 1994, "Residual neurobehavioral effects of chronic exposure to mercury vapor", *Occupat. Envir. Med.*, 1:35-41.

(114) M.Aschner et al, "Metallothionein induction in fetal rat brain by in utero exposure to elemental mercury vapor", *Brain Research*, 1997, dec 5, 778(1):222-32; & T.V. O'Halloran, "Transition metals in control of gene expression", *Science*, 1993, 261(5122):715-25; & Matts RL, Schatz JR, Hurst R, Kagen R. Toxic heavy metal ions inhibit reduction of disulfide bonds. *J Biol Chem* 1991; 266(19): 12695-702; Boot JH. Effects of SH-blocking compounds on the energy metabolism in isolated rat hepatocytes. *Cell Struct Funct* 1995; 20(3): 233-8; & Baauweegers HG, Troost D. Localization of metallothionein in the mammalian central nervous system. *Biol Signals* 1994, 3:181-7.

(115) G.Hall, V-TOX, Mercury levels excreted after Vit C IV as chelator - by number of fillings Int Symposium "Status Quo and Perspectives of Amalgam and Other Dental Materials" European Academy, Ostzenhausen/Germany. April 29 - May 1, 1994; & Heavy Metal Bulletin, Apr 1996, Vol.3, Issue 1, p6-8 (200 cured or significantly improved)

(119) (a) L.Ronnback et al, "Chronic encephalopathies induced by low doses of mercury or lead", *Br J Ind Med* 49: 233-240, 1992; & (b) H.Langauer-Lewowicka", "Changes in the nervous system due to occupational metallic mercury poisoning" *Neurol Neurochir Pol* 1997 Sep-Oct; 31(5):905-13; & (c) Langauer-Lewowicka H. [Chronic toxic encephalopathies] [Polish] *Med Pr.* 1982; 33(1-3):113-7; & (d)[Pneuropsychological disorders after occupational exposure to mercury vapors in El Bagre (Antioquia, Colombia)] *Rev Neurol.* 2000 Oct 16-31; 31(8):712-6. Tirado V, Garcia MA et al; & (f) Neurobehavioral effects of acute exposure to inorganic mercury vapor. *Appl Neuropsychol.* 1999; 6(4):193-200, Haut MW, Morrow LA et al. & (g) Personality traits in miners with past occupational elemental mercury exposure. *Environ Health Perspect.* 2006 Feb; 114(2):290-6; Kobal Grum D, Kobal AB et al

(122) B.Ono et al, "Reduced tyrosine uptake in strains sensitive to inorganic mercury", *Genet*, 1987, 11(5):399-

(140) R.L.Siblerud, "Health Effects After Dental Amalgam Removal", *J Orthomolecular Med* 5(2): 95-106.

(141) R.L.Siblerud et al, "Evidence that mercury from dental fillings may be an etiological factor in smoking", *Toxicol Lett*, v68, n3, 1993, p307- & v69(3):305.

(142) Ariza ME; Bijur GN; Williams MV. Lead and mercury mutagenesis: role of H_2O_2 , superoxide dismutase, and xanthine oxidase. *Environ Mol Mutagen* 1998; 31(4):352-61

(143) The extent of drug therapy for attention deficit-hyperactivity disorder among children in public schools. (*American Journal of Public Health.* 1999; 89(9):1359-64); & www.niehs.nih.gov/oc/news.adhd

(145) Adverse health effects of Ritalin and other stimulant drugs: <http://users.cybercity.dk/bbb9582/ritalin> & www.healthysource.com/ritalin.html; & www.breggin.com/RitalinNIHSPEECH.html; & www.healthoption.com & <http://lifefellowship.org/-Updatables/Articles/40.html>; Michael R. Lyon, Healing the Hyperactive Brain through the Science of Functional Medicine, www.pureliving.com/product.html¹⁵

(172) Life Extension Foundation (MDs), *Disease Prevention and Treatment*, Expanded Forth Edition, 2003.

¹⁴**Internet:** "<http://www.niehs.nih.gov/oc/news.adhd.htm>".

¹⁵**Internet:** "<http://www.pureliving.com/product.html>".

- (173) Digestive, Metabolic Problems Abound In Patients with ADHD, Autism, Erik L. Goldman Editor in Chief, Crossroads Institute, www.gordonresearch.com/category_adhd.html¹⁶ & www.gordonresearch.com.
- (174) Iodine: Why You Need It, Why You Can't Live Without It (4th Edition), Dr. David Brownstein, 2008; & Overcoming Thyroid Disorders, Dr. David Brownstein.
- (175) Soderstrom S, Fredriksson A, Dencker L, Ebendal T, "The effect of mercury vapor on cholinergic neurons in the fetal brain", Brain Research & Developmental Brain Res, 1995, 85:96-108; & Toxicol Lett 1995; 75(1-3): 133-44.
- (176) Niederhofer H. Ginkgo biloba treating patients with attention-deficit disorder. Phytother Res. 2009 May 14.
- (177) (a) Parent Ratings of Behavioral Effects of Biomedical Interventions for large group of parents of children who had autism, www.autism.com/treatable/form34qr.htm¹⁸ : & (b) Parent Ratings of Behavioral Effects of Biomedical Interventions for Asperger Syndrome www.autism.com/treatable/form34qrasp.htm & (c) Autism, an extreme challenge to integrative medicine. Part 2: medical management; Kidd PM. Altern Med Rev. 2002 Dec; 7(6):472-99
- (180) Susanna Visser et al, U.S. Center for Disease Control (CDC), and NBC Nightly News, 11-10-2010.
- (181) P.W. Mathieson, "Mercury: god of TH2 cells", 1995, Clinical Exp Immunol., 102(2):229-30;
- (196) Gowdy & Demes, 1978, in B. Wolfe and P. Wolfe, "Fillings, Mercury, and You", Mothering magazine, Summer, 1987.
- (198) Cd²⁺ and Hg²⁺ affect glucose release and cAMP-dependent transduction pathway in isolated eel hepatocytes. Aquat Toxicol. 2003 Jan 10; 62(1):55-65, Fabbri E, Caselli F, Piano A, Sartor G, Capuzzo A. & Fluctuation of trace elements during methylmercury toxication and chelation therapy. Hum Exp Toxicol. 1994 Dec; 13(12):815-23, Bapu C, Purohit RC, Sood PP; & E.S. West et al, Textbook of Biochemistry, MacMillan Co, 1957, p853
- (199) Dr. P.Kraub & M.Deyhle, Universitat Tuingen - Institut fur Organische Chemie, "Field Study on the Mercury Content of Saliva", 1997 <http://www.uni-tuebingen.de/KRAUSS/amalgam.html>; & (b) Dr.I.Gerhard, Dr. E.Roller, et al, Tuingen Univ. Gynecological Clinic, Heidelberg, 1996
- (207) Pendergrass JC, Haley BE, Univ. Of Kentucky Dept. Of Chemistry "The Toxic Effects of Mercury on CNS Proteins: Similarity to Observations in Alzheimer's's Disease", IAOMT Symposium paper, March 1997 & "Mercury Vapor Inhalation Inhibits Binding of GTP . . . - Similarity to Lesions in Alzheimer's Diseased Brains", Neurotoxicology 1997, 18(2)::315-24; & Met Ions Biol Syst, 1997, 34:461-
- (212) Ziff, M.F., "Documented clinical side effects to dental amalgams", ADV Dent. Res., 1992; 1(6):131-134; & Ziff, S., Dentistry without Mercury, 8th Edition, 1996, Bio-Probe, Inc., ISBN 0-941011-04-6; & Dental Mercury Detox, Bio-Probe, Inc. www.bioprobe.com. (cases:FDA Patient Adverse Reaction Reports - 762, Dr.M.Hanson-Swedish patients-519, Dr. H. Lichtenberg-100 Danish patients, Dr. P.Larose - 80 Canadian patients, Dr. R.Siblerud, 86 Colorado patients, Dr. A.V.Zamm, 22 patients)
- (222) M. Daunderer, "Improvement of Nerve and Immunological Damages after Amalgam Removal", Amer. J. Of Probiotic Dentistry and Medicine, Jan 1991
- (229) M.Davis, editor, "Defense Against Mystery Syndromes", Chek Printing Co. March, 1994(case histories documented)

¹⁶**Internet:** "http://www.gordonresearch.com/category_adhd.html".

¹⁷**Internet:** "http://www.gordonresearch.com/articles_adhd/Digestive_&_Metabolic_Problems_Abound_in_Patients_With_AD".

¹⁸**Internet:** "<http://www.autism.com/treatable/form34qr.htm>".

¹⁹**Internet:** "<http://www.autism.com/treatable/form34qraspergersyndrome.htm>".

(230) Sherry A. Rogers, M.D., *Depression - Cured at Last!* (1997), SK Publishing, P. O. Box 40101, Sarasota, FL 34242.

(233) Sven Langworth et al, "Amalgamnews and Amalgamkedefonden", 1997 and Svenska Dogbladet, 1997 (286 cases); & F.Berglund, Bjerner/Helm, Klock, Ripa, Lindforss, Mornstad, Ostlin), "Improved Health after Removal of dental amalgam fillings", Swedish Assoc. Of Dental Mercury Patients, 1998. (www.tf.nu) (over 1000 cases) (Sweden has decided to phase out amalgam fillings & Gov't maintains health records on all citizens)

(251) (c) Omura, Yoshiaki; *Abnormal Deposits of Al, Pb, and Hg in the Brain, Particularly in the Hippocampus, as One of the Main Causes of Decreased Cerebral Acetylcholine, Electromagnetic Field Hypersensitivity, Pre-Alzheimer's Disease, and Autism in Children; Acupuncture & Electro-Therapeutics Research*, 2000, Vol. 25 Issue 3/4, p230, 3p

(252) B.J.Shenker et al, Dept. of Pathology, Univ. of Pennsylvania, "Immunotoxic effects of mercuric compounds on human lymphocytes and monocytes: Alterations in cellular glutathione content", *Immunopharmacol Immunotoxicol* 1993, 15(2-3):273-90.

(254) al-Saleh I, Shinwari N. *Urinary mercury levels in females: influence of dental amalgam fillings. Biometals* 1997; 10(4): 315-23;

(257) I. Smith et al, "Pteridines and mono-amines: relevance to neurological damage", *Postgrad Med J*, 62(724): 113-123, 1986; & A.D.Kay et al, "Cerebrospinal fluid bipterin is decreased in Alzheimer's's disease", *Arch Neurol*, 43(10): 996-9, Oct 1986; & T.Yamiguchi et al, "Effects of tyrosine administration on serum bipterin In patients with Parkinson's Disease and normal controls", *Science*, 219(4580):75-77, Jan 1983; & T.Nagatsu et al, "Catecholoamine-related enzymes and the bipterin cofactor in Parkinson's", *Neurol*, 1984, 40: 467-73.

(258) Ely, J.T.A., "Mercury Induced Alzheimer's Disease: Accelerating Incidence?", *Bull Environ Contam Toxicol*. 2001, 67: 800-6; & Clinical Management of Poisoning, 3rd Ed., (p753) Haddad, Shannon, and Winchester, W.B. Saunders and Company, Philadelphia, 1998;

(259) C.K.Mittal et al, "Interaction of heavy metals with the nitric oxide synthase", *Mol Cell Biochem*, 149-150:263-5, Aug 1995; & J.P.Bolanos et al, "Nitric Oxide mediated mitochondrial damage in the brain", ??

(260) J.S. Woods et al, "Urinary porphyrin profiles as biomarker of mercury exposure: studies on dentists", *J Toxicol Environ Health*, 40(2-3):1993, p235-; & "Altered porphyrin metabolites as a biomarker of mercury exposure and toxicity", *Physiol Pharmacol*, 1996, 74(2):210-15

(280) S.Nonaka et al, Nat. Inst. of Mental Health, Bethesda Md., "Lithium treatment protects neurons in CNS from glutamate induced excitability and calcium influx", *Neurobiology*, Vol 95(5):2642-2647, Mar 3, 1998.

(281) T.W. Clarkson et al, "Transport of elemental mercury into fetal tissues", *Biol. Neonate*. 21:239-244, 1972; & M.R.Greenwood et al, "Transfer of metallic mercury into the fetus", *Experientia*, 28:1455-1456, 1972

(285) R.C.Perlingeiro et al, "Polymorphonuclear phagocytosis in workers exposed to mercuryvapor", *Int J Immunopharmacology*, 16(12):1011-7, 1994;; & Mathieson PW. 1995. Mercury: god of Th2 cells? *Clin Exp Immunol* 102:229_230; & (b) *Hum Exp Toxicol* 1995, 14(3):281-6; & M.L. Queiroz et al, *Pharmacol Toxicol*, 1994, 74(2):72-5; & (b) J.W.Albers et al, "Neurological abnormalities associated with remote occupational elemental mercury exposure", *Ann Neurol* 1988, 24(5):651-9.; & (c) *Effects of low exposure to inorganic mercury on psychological performance. Br J Ind Med*. 1990 Feb; 47(2):105-9. Soleo L, Urbano ML, Petrera V, Ambrosi L. & (e) M.S.Hua et al, "Chronic elemental mercury intoxication", *Brain Inj*, 1996, 10(5):377-84; & (f) Gunther W, et al, *Repeated neurobehavioral investigations in workers . . . , Neurotoxicology* 1996; 17(3-4):605-14;

(288) Rajanna B, Hobson M, Harris L, Ware L, Chetty CS. Effects of cadmium and mercury on Na(+)-K(+)ATPase and uptake of 3H-dopamine in rat brain synaptosomes. Arch Int Physiol Biochem 1990, 98(5):291-6; & M.Hobson & B.Rajanna, "Influence of mercury on uptake of dopamine and norepinephrine", Toxicol Letters, Dep 1985, 27:2-3:7-14; & McKay SJ, Reynolds JN, Racz WJ. Effects of mercury compounds on the spontaneous and potassium-evoked release of [3H]dopamine from mouse striatal slices. Can J Physiol Pharmacol 1986, 64(12):1507-14; & Scheuhammer AM; Cherian MG. Effects of heavy metal cations, sulfhydryl reagents and other chemical agents on striatal D2 dopamine receptors. Biochem Pharmacol 1985 Oct 1; 34(19):3405-13; Lewis RN; Bowler K. Rat brain (Na+-K+)ATPase: modulation of its ouabain-sensitive K+-PNPPase activity by thimerosal. Int J Biochem 1983; 15(1):5-7; & Anner BM, Moosmayer M. Mercury inhibits Na-K-ATPase primarily at the cytoplasmic side. Am J Physiol 1992; 262(5 Pt2):F84308.

(290) D. Echeverria et al, "Neurobehavioral effects from exposure to dental amalgam" FASEB J, Aug 1998, 12(11):971-980.

(294) "Do amalgam fillings influence manic depression?", Journal of Orthomol. Medicine, 1998, www.depression.com/news/news_981116.htm²⁰

(295) Cecil Textbook of Medicine, 20th Ed., Bennett & Plum, W.B. Saunders and Company, Philadelphia, 1996, p 69; & Comprehensive Psychiatry, Vol 18(6), 1977, pp595-598, & poisoning & Toxicology Compendium, Leikin and Paloucek, Lexi-Comp., Cleveland, 1998.

(296) Harrison's Principles Of Internal Medicine, 14th Ed., McGraw-Hill, N.y., 1998.

(300) C.Hock et al, "Increased blood mercury levels in patients with Alzheimer's's disease", J. Neural Transm, 1998, 105(1):59-68.

(303) Heavy Metal and Chemical Toxicity, Dietrich Klinghardt, MD, Ph.D. www.neuraltherapy.com/chemtox & Mercury Toxicity and Systemic Elimination Agents, D. Klinghardt & J Mercola (DO), J of Nutritional and Environmental Medicine, 2001, 11:53-62; & Amalgam Detox, Klinghardt Academy of Neurobiology, 2008

(304) R.F. Kidd, Results of Dental Amalgam Removal and Mercury Detoxification, Alternative Therapies, July 2000, Vol 6, No. 4, p49-55.

(305) Soderstrom S, Fredriksson A, Dencker L, Ebendal T, "The effect of mercury vapor on cholinergic neurons in the fetal brain", Brain Research & Developmental Brain Res, 1995, 85:96-108; & Leong CC, Syed NI, Lorscheider FL. Retrograde degeneration of neurite membrane structural integrity of nerve growth cones following in vitro exposure to mercury. Neuroreport 2001 Mar 26; 12(4):733-7

(313) V.D.M.Stejskal et al, "Mercury-specific Lymphocytes: an indication of mercury allergy in man", J. Of Clinical Immunology, 1996, Vol 16(1); 31-40.

(317) S.Zinecker, "Amalgam: Quecksilberdamme bis ins Gehirn", der Kassenarzt, 1992, 32(4):23; "Praxiproblem Amalgam", Der Allgermeinarzt, 1995, 17(11):1215-1221. (1800 patients)

(320) U.F.Malt et al, "Physical and mental problems attributed to dental amalgam fillings", Psychosomatic medicine, 1997, 59:32-41. (99 cured)

(322) P.Engel, "Beobachtungen uber die gesundheit vor und nach amalgamentfernung", Separatdruck aus Schweiz. Monatsschr Zahnm. 1998, vol 108(8).(75 cases amalgam removal) <http://soho.globalpoint.ch/engel>

(330) B. Windham, ALS: the mercury connection, www.flcv.com/als.htm²²; over 100 peer-reviewed medical study references.

²⁰**Internet:** "http://www.depression.com/news/news_981116.htm".

²¹**Internet:** "<http://www.neuraltherapy.com/chemtox.htm>".

²²**Informativo:** "Amyotrophic Lateral Sclerosis (ALS): Lou Gerhig's Disease - The Mercury Connection".

(331) C.Gordon et al, "Abnormal sulphur oxidation in systemic lupus erythrmatosus (SLE)", *Lancet*, 1992, 339:8784, 25-6; & P.Emory et al, "Poor sulphoxidation in patients with rheumatoid arthritis", *Ann Rheum Dis*, 1992, 51:3, 318-20; & P.Emory et al, *Br J Rheumatol*, 1992, 31:7, 449-51; & Steventon GB, et al; Xenobiotic metabolism in motor neuron disease, *Neurology* 1990, 40:1095-98.

(333) A.J.Freitas et al, "Effects of Hg²⁺ and CH₃Hg⁺ on Ca²⁺ fluxes in the rat brain", *Brain Research*, 1996, 738(2): 257-64; & P.R.Yallapragoda et al, "Inhibition of calcium transport by Hg salts in rat cerebellum and cerebral cortex", *J Appl toxicol*, 1996, 164(4): 325-30; & E.Chavez et al, "Mitochondrial calcium release by Hg²⁺", *J Biol Chem*, 1988, 263:8, 3582-; A. Szucs et al, *Cell Mol Neurobiol*, 1997, 17(3): 273-8; & D.Busselberg, 1995, "Calcium channels as target sites of heavy metals", *Toxicol Lett*, Dec; 82-83:255-61; & *Cell Mol Neurobiol* 1994 Dec; 14(6):675-87; & Rossi AD, et al, Modifications of Ca²⁺ signaling by inorganic mercury in PC12 cells. *FASEB J* 1993, 7:1507-14.

(338) (a) W.Y.Boadi et al, Dept. Of Food Engineering and Biotechnology, T-I Inst of Tech., Haifa, Israel, "In vitro effect of mercury on enzyme activities and its accumulation in the first-trimester human placenta", *Environ Res*, 1992, 57(1):96-106; & "In vitro exposure to mercury and cadmium alters term human placental membrane fluidity", *Pharmacol*, 1992, 116(1): 17-23; & (b) J.Urbach et al, Dept. of Obstetrics & Gynecology, Rambam Medical Center, Haifa, Israel, "Effect of inorganic mercury on in vitro placental nutrient transfer and oxygen consumption", *Reprod Toxicol*, 1992, 6(1):69-75; & (c) Karp W, Gale TF et al, Effect of mercuric acetate on selected enzymes of maternal and fetal hamsters" *Environmental Research*, 36:351-358; & W.B. Karp et al, "Correlation of human placental enzymatic activity with trace metal concentration in placenta", *Environ Res*. 13:470-477, 1977; & (d) Boot JH. Effects of SH-blocking compounds on the energy metabolism and glucose uptake in isolated rat hepatocytes. *Cell Struct Funct* 1995 Jun; 20(3):233-8;

(369) Sterzl I, Prochazkova J, Stejskal VDM et al, Mercury and nickel allergy: risk factors in fatigue and autoimmunity. *Neuroendocrinology Letters* 1999; 20:221-228; & Prochazkova J, Sterzl I, Kucerova H, Bartova J, Stejskal VD; The beneficial effect of amalgam replacement on health in patients with autoimmunity. *Neuro Endocrinol Lett*. 2004 Jun; 25(3):211-8. www.melisa.org²³

(372) Atchison WD. Effects of neurotoxicants on synaptic transmission. *Neurotoxicol Teratol* 1998, 10(5):393-416; & Sidransky H, Verney E, Influence of lead acetate and selected metal salts on tryptophan binding to rat hepatic nuclei. *Toxicol Pathol* 1999, 27(4):441-7; & Shukla GS, Chandra SV, Effect of interaction of Mn²⁺ with Zn²⁺, Hg²⁺, and Cd²⁺ on some neurochemicals in rats. *Toxicol Lett* 1982, 10(2-3):163-8; & Brouwer M et al, Functional changes induced by heavy metal ions. *Biochemistry*, 1982, 21(20): 2529-38.

(374) Benkelfat C et al, Mood lowering effect of tryptophan depletion. *Arch Gen Psychiatry*, 1994, 51(9): 687-97; & Young SN et al, Tryptophan depletion causes a rapid lowering of mood in normal males. *Psychopharmacology*, 1985, 87(2):173-77; & Smith KA et al, Relapse of depression after depletion of tryptophan, *Lancet* 1997, 349(9056):915-19; & Delgado PL et al, Serotonin function, depletion of plasma tryptophan, and the mechanism of antidepressant action. *Arch Gen Psychiatry* 1990, 47(5):411-18.

(375) Stejskal VDM, Danersund A, Lindvall A. Metal-specific memory lymphocytes: biomarkers of sensitivity in man. *Neuroendocrinology Letters* 1999; & Stejskal V, Hudecek R, Mayer W, "Metal-specific lymphocytes: risk factors in CFS and other related diseases", *Neuroendocrinology Letters*, 20: 289-298, 1999; www.melisa.org

(376) Melchart D, Wuhr E, Weidenhammer W, Kremers L. A multicenter survey of amalgam fillings and subjective complaints in non-selected patients in the dental practice. *Eur J Oral Sci* 1998; 106:770-77 (6, 744 patients in 34 clinics)

(382) Sterzl I, Fucikova T, Zamrazil V. The fatigue syndrome in autoimmune thyroiditis with polyglandular activation of autoimmunity. *Vnitřní Lekarství* 1998; 44: 456-60; & (b) Sterzl I, Hrda

²³**Internet:** "<http://www.melisa.org/>".

P, Prochazkova J, Bartova J, Reactions to metals in patients with chronic fatigue and autoimmune endocrinopathy. *Vnitr Lek* 1999 Sep; 45(9):527-31; & & (c) Kolenic J, Palcakova D, Benicky L, Kolenicova M - "The frequency of auto-antibody occurrence in occupational risk (mercury)" *Prac Lek* 45(2):75-77 (1993)

(386) Genova Diagnostics, [click: Tests, Search by Disease, see Disease in question & Heavy Metal Toxicity], www.genovadiagnostics.com²⁴; & Doctors Data Lab, <http://www.doctorsdata.com>, inquiries @doctors data.com, www.doctorsdata.com, & MetaMetrix Lab, www.metametrix.com; & (d) Biospectron Lab, LMI, Lennart Månsson International AB, lmi.analyslab@swipnet.se home.swipnet.se/misac/

(390) Ellingsen DG, Efskind J, Haug E, Thomassen Y, Martinsen I, Gaarder PI - "Effects of low mercury vapour exposure on the thyroid function in chloralkali workers" *J Appl Toxicol* 20(6):483-9 (2000) www.ncbi.nlm.nih.gov/htbin-post/Entrez/query?uid=11180271&form=6&db=m&Dopt=r; & (b) Barregard L, Lindstedt G, Schutz A, Sallsten G - "Endocrine function in mercury exposed chloralkali workers" *Occup Environ Med* 51(8):536-40 (1994)

(391) Subclinical hypothyroidism: psychiatric disorders and symptoms. Almeida C, Brasil MA, Costa AJ, Vaisman M. *Rev Bras Psiquiatr.* 2007 Jun; 29(2):157-9, & Screening for depression disorders in patients with chronic somatic illness. Filipčić I, Popović-Grle S, Hajnaek S, Aganović I. *Coll Antropol.* 2007 Mar; 31(1):139-43; & Neuropsychiatric aspects of hypothyroidism and treatment reversibility. . Davis JD, Tremont G. *Minerva Endocrinol.* 2007 Mar; 32(1):49-65, etc.

(404) M. E. Godfrey, Candida, Dysbiosis and Amalgam. *J. Adv. Med.* vol 9 no 2 (1996); & Romani L, Immunity to Candida Albicans: Th1, Th2 cells and beyond. *Curr Opin Microbiol* 1999, 2(4):363-7; & Alfred V. Zamm. CANDIDA ALBICANS THERAPY: Dental mercury removal, an effective adjunct. *J. Orthmol. Med.* v1#4 pp261-5 (1986)

(405) Neurological Effects of Dysbiosis Involving Gluten and Casein, the Mercury Connection, Review, B Windham (Ed), 2009. www.flcv.com/autismgc.html²⁶ & www.flcv.com/leakyghg.html²⁷

(406) Dept. of Gastroenterology, University of Naples, Italy: Depressive symptoms in adult celiac disease. *Scand J Gastroenterol* 1998; 33(3):247-50.

(407) Eedy DJ, Burrows D, Dliford T, Fay A. Elevated T cell subpopulations in dental students. *J prosthet Dent* 1990; 63(5):593-6; & Yonk LJ et al, CD+4 helper T-cell depression in autism. *Immunol Lett*, 1990, 25(4):341-5; & Jaffe JS, Strober W, Sneller MC, Functional abnormalities of CD8+ T cells define a unique subset of patients with common variable immunodeficiency. *Blood* 1993, 82(1): 192-20.

(408) Yonk LJ et al, CD+4 helper T-cell depression in autism. *Immunol Lett*, 1990, 25(4):341-5; & Jaffe JS, Strober W, Sneller MC, Functional abnormalities of CD8+ T cells define a unique subset of patients with common variable immunodeficiency. *Blood* 1993, 82(1): 192-20.

(409) Bernard S, Enayati A, Redwood L, Roger H, Binstock T. Autism: a novel form of mercury poisoning. *Med Hypotheses* 2001 Apr; 56(4):462-71. <http://www.autism.com/ari/mercurylong.html> : & Yazbak FE (MD, FAAP) Autism 99 : A National Emergency, www.garynull.com/documents/autism_99.htm

(410) J.R. Cade et al, Autism and schizophrenia linked to malfunctioning enzyme for milk protein digestion. *Autism*, Mar 1999.

(411) Puschel G, Mentlein R, Heymann E, 'Isolation and characterization of dipeptyl peptidase IV from human placenta', *Eur J Biochem* 1982 Aug; 126(2):359-65; & Kar NC, Pearson CM. Dipeptyl Peptidases in human muscle disease. *Clin Chim Acta* 1978; 82(1-2): 185-92; & Seroussi K, Autism and Pervasive Developmental Disorders, 1998, p174, etc.

²⁴**Internet:** "<http://www.genovadiagnostics.com>".

²⁵**Internet:** "<http://home.swipnet.se/misac/research11.html#biospectrons>".

²⁶**Internet:** "<http://www.flcv.com/autismgc.html>".

²⁷**Internet:** "<http://www.flcv.com/leakyghg.html>".

(412) (a) Moreno-Fuenmayor H, Borjas L, Arrieta A, Valera V, Plasma excitatory amino acids in autism. *Invest Clin* 1996, 37(2):113-28; & Carlsson ML. Is infantile autism a hypoglutamatergic disorder? *J Neural Transm* 1998, 105(4-5): 525-35. & (b) Rolf LH, Haarman FY, Grotemeyer KH, Kehrer H. Serotonin and amino acid content in platelets of autistic children. *Acta Psychiatr Scand* 1993, 87(5): 312-6; & (c) Naruse H, Hayashi T, Takesada M, Yamazaki K. Metabolic changes in aromatic amino acids and monoamines in infantile autism and a new related treatment, *No To Hattatsu*, 1989, 21(2):181-9;

(413) Autism-Mercury@egroups.com, web group of parents with autistic kids and autism doctors and researchers; & <http://www.edelsoncenter.com>; & Edelson SB, Cantor DS. Autism: xenobiotic influences. *Toxicol Ind Health* 1998; 14(4): 553-63; & Liska, DJ. The detoxification enzyme systems. *Altern Med Rev* 1998. 3(3):187-98

(416) Kim P, Choi BH. "Selective inhibition of glutamate uptake by mercury in cultured mouse astrocytes", *Yonsei Med J* 1995; 36(3): 299-305; & Brookes N. In vitro evidence for the role of glutamate in the CNS toxicity of mercury. *Toxicology* 1992, 76(3):245-56; & Albrecht J, Matyja E. Glutamate: a potential mediator of inorganic mercury toxicity. *Metab Brain Dis* 1996; 11:175-84; & Tirosh O, Sen CK, Roy S, Packer L. Cellular and mitochondrial changes in glutamate-induced HT4 neuronal cell death *Neuroscience*. 2000; 97(3):531-41

(418) Srikantaiah MV; Radhakrishnan AN. Studies on the metabolism of vitamin B6 in the small intestine. Purification and properties of monkey intestinal pyridoxal kinase. *Indian J Biochem* 1970 Sep; 7(3):151-6.; & Spivey-Fox MR. Nutritional influences on metal toxicity. *Environ Health Perspect* 1979; 29: 95-104; & (b) McCarty MF. High-dose pyridoxine as an 'anti-stress' strategy. *Med Hypotheses*. 2000 May; 54(5):803-7.

(424) Munch G; Gerlach M; Sian J; Wong A; Riederer P. Advanced glycation end products in neurodegeneration: more than early markers of oxidative stress? *Ann Neurol* 1998 Sep; 44(3 Suppl 1):S85-8.

(427) Chetty CS, McBride V, Sands S, Rajanna B. Effects in vitro on rat brain Mg (++)-ATPase. *Arch Int Physiol Biochem* 1990, 98(5):261-7; & M.Burk et al, Magnesium, 4(5-6):325-332, 1985

(438) Stefanovic V. et al, Kidney ectopeptidases in mercuric chloride-induced renal failure. *Cell Physiol Biochem* 1998; 8(5): 278-84

(451) Miszta H; Dabrowski Z. Effect of mercury and combined effect of mercury on the activity of acetylcholinesterase of rat lymphocytes during in vitro incubation. *Folia Haematol Int Mag Klin Morphol Blutforsch* 1989; 116(1):151-5; & Bear, David; Rosenbaum, Jerrold; Norman, Robert. Aggression in cat and human precipitated by a cholinesterase inhibitor. *The journal Psychosomatics*, July 1986, vol. 27, #7, pgs. 535-536; & Devinsky, Orrin; Kernan, Jennifer; Bear, David. Aggressive Behavior Following Exposure to Cholinesterase Inhibitors. *Journal of Neuropsychiatry*, vol. 4, #2, Spring 1992, pgs. 189-199.

(460) Edwards AE, Depression and Candida, *JAMA*, 1985, 253(23): 3400; & Crook WG, Depression associated with Candida albicans infections, *JAMA*, 1984, 251:22; & Crook, W. G. 1997. *The Yeast Connection Handbook*. Professional Books, Inc., Jackson, Tennessee; & Genova Diagnostic Lab, www.gdx.net.

(465) Walsh WJ, Health Research Institute, Biochemical Treatment of Mental Illness and Behavior Disorders, Minnesota Brain Bio Assoc, Nov 17, 1997; www.hriptc.org/Minnesota.htm²⁸; & William J. Walsh, Laura B. Glab, and Mary L. Haakenson; Pfeiffer Treatment Center, Biochemical Therapy and Behavior Outcomes; 2000, www.hriptc.org/btbres.htm²⁹

²⁸**Internet:** "<http://www.hriptc.org/Minnesota.htm>".

²⁹**Internet:** "<http://www.hriptc.org/btbres.htm>".

(480) Salzer HM, Relative hypoglycemia as a cause of neuropsychiatric illness, J National Med Assoc, 1996. 58(1): 12-17; & Heninger GR et al, Depressive symptoms, glucose tolerance, and insulin tolerance, J Nervous and Mental Dis, 1975; 161(6):421-32; & Winokur A et al, Insulin resistance in patients with major depression, Am J Psychiatry, 1988, 145(3): 325-30.

(481) Virkkunen M, Huttunen MO; Evidence for abnormal glucose tolerance among violent offenders, Neuropsychobiology, 1982, 8:30-40; & (b) Markku I, Virkkunen L; Aggression, suicidality, and serotonin, J Clinical Psy 1992, 53(10): 46-51; & (c) Assessment of chronic neuropsychological effects of mercury vapour poisoning in chloral-alkali plant workers. Bosn J Basic Med Sci. 2002 Dec; 2(1-2):29-34. Pranjic N, Sinanovic O, et al.

(482) Linnoila M et al, Low serotonin metabolite differentiates impulsive from nonimpulsive violent behavior, Life Sciences, 1983, 33(26): 2609-2614; & Lopez-Ibor JJ, Serotonin and psychiatric disorders. Int Clinical Psychopharm, 1992, 7(2): 5-11.

(483) Thomas DE et al, Tryptophan and nutritional status in patients with senile dementia, Psychological Med 1986, 16:297-305; & Yaryura-Tobias JA et al, Changes in serum tryptophan and glucose in psychotics and neurotics. Nutrition, No.4557, p1132; Carney MWP, Brit Med J, 1967, 4:512-516.

(484) Urberg M, Zemel MB; Evidence for synergism between chromium and nicotinic acid in the control of glucose tolerance in elderly humans, Metabolism, 1987, 36(9): 896-899; & J Family Practice, 1988, 27(6): 603-606; & Anderson RA et al, Effects of supplemental chromium on patients with reactive hypoglycemia, Metabolism. 1987, 36(4): 351-355; & Metabolism, 1983, 32(9): 894-99.

(487) Haut MW; Morrow LA; Pool D; Callahan TS; Haut JS; Franzen MD. Neurobehavioral effects of acute exposure to inorganic mercury vapor. Appl Neuropsychol 1999; 6(4):193-200.

(488) Depression causes and treatments, Life Extension Foundation, www.lef.org/protocols/emotional_health

(490) Fava M, Giannelli A, Rapisarda V, Patrاليا A, Guaraldi GP. Rapidity of onset of the antidepressant effect of parenteral S-adenosyl-L-methionine. Psychiatry Res 1995 Apr 28; 56(3):295-7; & Rosenbaum JF, Fava M, Falk WE, Pollack MH, Cohen LS, Cohen BM, Zubenko GS. The antidepressant potential of oral S-adenosyl-l-methionine. Acta Psychiatr Scand 1990 May; 81(5):432-6

(491) Levine J. Controlled trials of inositol in psychiatry. Eur Neuropsychopharmacol 1997 May; 7(2):147-55; & Inositol versus placebo augmentation of serotonin reuptake inhibitors in the treatment of obsessive-compulsive disorder: a double-blind cross-over study. Inositol versus placebo augmentation of serotonin reuptake inhibitors in the treatment of obsessive-compulsive disorder: a double-blind cross-over study. Int J Neuropsychopharmacol 1999 Sep; 2(3):193-195; & Palatnik A, Frolov K, Fux M, Benjamin J. Double-blind, controlled, crossover trial of inositol versus fluvoxamine for the treatment of panic disorder. J Clin Psychopharmacol 2001 Jun; 21(3):335-9; & Chengappa KN, Levine J, Kupfer DJ. Inositol as an add-on treatment for bipolar depression. Bipolar Disord 2000 Mar; 2(1):47-55

(493) *Bibliography for Depression*³¹

(494) Narang RL, Gupta, KR: Levels of copper and zinc in depression. Indian J of Physiol Pharmacol 1991; 35(4):272-4; & McLoughlin IJ, Hodge JS: Zinc in depressive disorder. Acta Psychiatr Scand 1990; 82(6):451-3;

(495) Miller HL, et al.: Acute tryptophan depletion: a method of studying antidepressant action. J Clin Psychiatry 1992; 53 Suppl: 28-35; & Boman B: L-tryptophan: A rational anti-depressant and a natural hypnotic? Aust N Z J Psychiatry 1988; 22(1):83-97; & (c) Young SN: The use of diet and dietary components in the study of factors controlling affect in humans: a review. J Psychiatry

³⁰Internet: "http://www.lef.org/protocols/emotional_health/depression_01.htm".

³¹Internet: "<http://www.hriptc.org/content/depression.php>".

Neurosci 1993; 18(5):235-44.

(496) Doble A. The role of excitotoxicity in neurodegenerative disease: implications for therapy. *Pharmacol Ther* 1999 Mar; 81(3):163-221

(521) Guermonprez L, Ducrocq C, Gaudry-Talarmain YM. Inhibition of acetylcholine synthesis and tyrosine nitration induced by peroxynitrite are differentially prevented by antioxidants. *Mol Pharmacol* 2001 Oct; 60(4):838-46; & Mahboob M, Shireen KF, Atkinson A, Khan AT. Lipid peroxidation and antioxidant enzyme activity in different organs of mice exposed to low level of mercury. *J Environ Sci Health B*. 2001 Sep; 36(5):687-97.

(522) Nutrition Supplements Found Effective for Metal Disorders, Dr. Julia Rucklidge et al, University of Canterbury, *Journal of Attention Disorders*, January 2010 (EMPowerPlus, TrueHope) www.truehope.com/truehope_bipolar_disorder_research_empowerplus_1.aspx³²

(524) Torreilles F, Salman-Tabcheh S, Guerin M, Torreilles J. Neurodegenerative disorders: the role of peroxynitrite. *Brain Res Brain Res Rev* 1999 Aug; 30(2):153-63; & (b) Aoyama K, Matsubara K, Kobayashi S. Nitration of manganese superoxide dismutase in cerebrospinal fluids is a marker for peroxynitrite-mediated oxidative stress in neurodegenerative diseases. *Ann Neurol* 2000 Apr; 47(4):524-7; & (c) Guermonprez L, Ducrocq C, Gaudry-Talarmain YM. Inhibition of acetylcholine synthesis and tyrosine nitration induced by peroxynitrite are differentially prevented by antioxidants. *Mol Pharmacol* 2001 Oct; 60(4):838-46

(530) 5-HTP Archives, Dr. G. Valentine and W. Block, Life-Enhancement, www.life-enhancement.com³³; & (b) Birdsall TC. 5-Hydroxytryptophan: a clinically-effective serotonin precursor. *Altern Med Rev*. 1998 Aug; 3(4):271-80.

(534) Tirado V, Garcia MA, Franco A., Pneuropsychological disorders after occupational exposure to mercury vapors, *Rev Neurol* 2000 Oct 16-31; 31(8):712-6; & Powell TJ. Chronic neurobehavioural effects of mercury poisoning on a group of chemical workers. *Brain Inj* 2000 Sep; 14(9):797-814

(543) U.S. Centers for Disease Control, National Center for Health Statistics, NHANES III study (thousands of people's health monitored), www.flcv.com/NHanes3.htm³⁴; & www.mercola.com/article/mercury & Review: cancer related to mercury exposure, B. Windham (Ed) www.flcv.com/cancerhg.htm³⁵; & (b) Laks, Dan R. Assessment of chronic mercury exposure within the U.S. population, National Health and Nutrition Examination Survey, 1999-2006. *Biometals*. August 2009; & Laks, D.R. et al, Mercury has an affinity for pituitary hormones, *Medical Hypotheses*, Dec 2009; & (c) An Investigation of Factors Related to Levels of Mercury in Human Hair, Environmental Quality Institute. October 01, 2005. www.greenpeace.org/raw/content/usa/press/reports/mercury-report.pdf³⁷, www.greenpeace.org/usa/assets/binaries/addendum-to-mercury-report³⁸

(551) B. Windham, Children's neurological conditions: the toxic exposure connection, 2001. www.flcv.com/indexk.html (over 150 peer-reviewed studies referenced)

(552) B. Windham, Toxic effects of pesticides, 2001, www.flcv.com/pesticid.html³⁹

(553) Endocrine effects of mercury, B. Windham (Ed), www.flcv.com/endohg.html⁴⁰ & Effects of endocrine disrupting chemicals, B. Windham (Ed), www.flcv.com/endocrin.html⁴¹

³²**Internet:** "http://www.truehope.com/truehope_bipolar_disorder_research_empowerplus_1.aspx".

³³**Internet:** "http://www.life-enhancement.com".

³⁴**Internet:** "http://www.flcv.com/NHanes3.htm".

³⁵**Internet:** "http://www.mercola.com/article/mercury/no_mercury.htm".

³⁶**Informative:** "Cancer Connection to Mercury, Toxic Metals, and Dental Cavitations".

³⁷**Internet:** "http://www.greenpeace.org/raw/content/usa/press/reports/mercury-report.pdf".

³⁸**Internet:** "http://www.greenpeace.org/usa/assets/binaries/addendum-to-mercury-report".

³⁹**Internet:** "http://www.flcv.com/pesticid.html".

⁴⁰**Internet:** "http://www.flcv.com/endohg.html".

⁴¹**Internet:** "http://www.flcv.com/endocrin.html".

- (560) Great Smokies Diagnostic Lab, (search news & (by condition: depression) www.gsdl.com.
- (561) Maes M, Vandoolaeghe E, Neels H, Demedts P, Wauters A, Meltzer HY, Altamura C, Desnyder R. Lower serum zinc in major depression is a sensitive marker of treatment resistance and of the immune/inflammatory response in that illness. *Biol Psychiatry* 1997; 42(5):349-358.
- (562) Rasmussen HH, Mortensen PB, Jensen IW. Depression and magnesium deficiency. *Int J Psychiatry Med* 1989; 19(1):57-63; & (b) Levine J, Stein D: High serum and cerebrospinal fluid Ca/Mg ratio in recently hospitalized acutely depressed patients. *Neuropsychobiology* 1999; 39(2):63-70.
- (563) Naylor GJ, Smith AH, Bryce-Smith D, Ward NI. Elevated vanadium content of hair and mania. *Biol Psychiatry* 1984; 19(5):759-764.
- (564) McIntyre IM, Judd FK, Marriott PM, et al. Plasma melatonin levels in affective states. *Int J Clin Pharmacol Res.* 1989; 9(2):159-64; & Riemann D, Klein T, Rodenbeck A, et al. Nocturnal cortisol and melatonin secretion in primary insomnia. *Psychiatry Res.* 2002 Dec 15; 113(1-2):17-27; & Wade AG, Ford I, Crawford G, McMahan AD, Nir T, Laudon M, Zisapel N. Efficacy of prolonged release melatonin in insomnia patients aged 55-80 years: quality of sleep and next-day alertness outcomes. *Curr Med Res Opin.* 2007 Oct; 23(10):2597-605.
- (565) Dr. Andrew Weil, www.drweilselfhealing.com⁴²; & www.drweil.com/drw/u/ART00696/depression-treatment⁴³, www.drweil.com/drw/u/QAA400692/Tyrosine-Good-Supplement-for-Depression.html⁴⁴, www.healthyplace.com/depression/alternative-treatments/natural-remedies-for-depression/menu-id-68⁴⁵
- (566) Depression Diet Dr. Mark Hyman, Editor - Alternative Therapies in Health & Medicine and Alternative Medicine, and author of many books on overcoming depression and other mental health issues, www.drhyman.com⁴⁶ & www.naturalsolutionsmag.com/article-display/8697/subTopicID/181/Brain-Food-The-Natural-Cure-for-Depression⁴⁷
- (567) Kim CY, Satoh H, et al, Protective effect of melatonin on methylmercury-Induced mortality in mice. *Tohoku J Exp Med.* 2000 Aug; 191(4):241-6; & Olivieri G, Hock C, et al, Mercury induces cell cytotoxicity and oxidative stress and increases beta-amyloid secretion and tau phosphorylation in SHSY5Y neuroblastoma cells. *J Neurochem.* 2000 Jan; 74(1):231-6.
- (568) Bemis JC, Seegal RF; 2000, PCBs and methylmercury alter intracellular calcium concentrations in rat cerebellar granule cells. *Neurotoxicology*, 21(6): 1123-1134.
- (569) Baccarelli A, Pesatori AC, Bertazzi PA. Occupational and environmental agents as endocrine disruptors: experimental and human evidence. *J Endocrinol Invest.* 2000 Dec; 23(11):771-81; & Libe R, Baccarelli A, et al, Long-term follow-up study of patients with adrenal incidentalomas. *Eur J Endocrinol.* 2002 Oct; 147(4):489-94.
- (571) Manzo L, Candura SM, Costa LG, et al; Biochemical markers of neurotoxicity. A review of mechanistic studies and applications. *Hum Exp Toxicol*, 1996 Mar, 15 Suppl 1:, S20-35.
- (580) Life Extension Foundation (MDs), Disease Prevention and Treatment, Expanded 4th Edition, 2003; & (b) American Journal of Clinical Nutrition, 2008 & Life Extension Foundation, Life Extension, Jan 2009,, www.life-enhancement.com⁴⁸; & (c) Volz HP. Controlled clinical trials of hypericum extracts in depressed patients—an overview. *Pharmacopsychiatry.* 1997 Sep; 30 Suppl 2:72-6; &
-
- ⁴²**Internet:** “<http://www.drweilselfhealing.com>”.
- ⁴³**Internet:** “<http://www.drweil.com/drw/u/ART00696/depression-treatment>”.
- ⁴⁴**Internet:** “<http://www.drweil.com/drw/u/QAA400692/Tyrosine-Good-Supplement-for-Depression.html>”.
- ⁴⁵**Internet:** “<http://www.healthyplace.com/depression/alternative-treatments/natural-remedies-for-depression/menu-id-68>”.
- ⁴⁶**Internet:** “<http://www.drhyman.com>”.
- ⁴⁷**Internet:** “<http://www.naturalsolutionsmag.com/article-display/8697/subTopicID/181/Brain-Food-The-Natural-Cure-for-Depression>”.
- ⁴⁸**Internet:** “<http://www.life-enhancement.com>”.

Melzer J, Brignoli R, Keck ME, et al. A hypericum extract in the treatment of depressive symptoms in outpatients: an open study. *Forsch Komplementmed*. 2010; 17(1):7-14;

(581) 1H magnetic resonance spectroscopy study in adults with obsessive compulsive disorder: relationship between metabolite concentrations and symptom severity, Starck G, Carlsson ML, et al, *J Neural Transm*. 2008 Jul; 115(7):1051-62. Epub 2008 Jun 5; & On the role of prefrontal cortex glutamate for the antithetical phenomenology of obsessive compulsive disorder and attention deficit hyperactivity disorder, Carlsson ML, *Biol Psychiatry*. 2001 Jan; 25(1):5-26

(582) Vitamin Research News, weekly journal (several editions), 2003-2009, www.vrp.com⁴⁹ & (b) Chikani V, Reding D, Gunderson P, et al. Wisconsin rural women's health study psychological factors and blood cholesterol level: difference between normal and overweight rural women. *Clin Med Res*. 2004 Feb; 2(1):47-53; & Rääkkönen K, Matthews KA, Kuller LH. Trajectory of psychological risk and incident hypertension in middle-aged women. *Hypertension*. 2001 Oct; 38(4):798-802; & Matthews KA, Owens JF, Kuller LH, et al. Are hostility and anxiety associated with carotid atherosclerosis in healthy postmenopausal women? *Psychosom Med*. 1998 Sep-Oct; 60(5):633-8; & Horsten M, Ericson M, Perski A, et al. Psychosocial factors and heart rate variability in healthy women. *Psychosom Med*. 1999 Jan-Feb; 61(1):49-57; & (c) Vlastelica M. Emotional stress as a trigger in sudden cardiac death. *Psychiatr Danub*. 2008 Sep; 20(3):411-4; & (d) Birkmayer JGD, Birkmayer W: The coenzyme nicotinamide adenine dinucleotide (NADH) as a biological antidepressive agent. *New Trends in Clinical Neuropharmacology* 1992; 6:1-7.

(583) Dr. J. Teitelbaum, *Health & Wellness Update*, Issue 198, Jan 2009; & (b) László KD, Janszky I, Ahnve S. Anger expression and prognosis after a coronary event in women. *Int J Cardiol*. 2010 Apr 1; 140(1):60-5; & Olson MB, Krantz DS, Kelsey SF, et al. Hostility scores are associated with increased risk of cardiovascular events in women undergoing coronary angiography: a report from the NHLBI-Sponsored WISE Study. *Psychosom Med*. 2005 Jul-Aug; 67(4):546-52.

(584) An Invitation to Health: 2009-2010 Edition, Dianne Hales, 2009.

(585) Pesticide poisoning and depressive symptoms among farm residents. Stallones L, Beseler C. *Ann Epidemiol*. 2002 Aug; 12(6):389-94; & Depression among victims of south Mississippi's methyl parathion disaster. Rehner TA, Kolbo JR, Trump R, Smith C, Reid D. *Health Soc Work*. 2000 Feb; 25(1):33-40.

(586) Environmental and occupational medicine, William N. Rom, Steven B. Markowitz, Review, 2007; & Neurobehavioural evaluation of Venezuelan workers exposed to inorganic lead. N.A. Maizlish, GParra, O Feo, *Occup Environ Med* 1995; 52:408-414 & (b) Major depressive disorder and panic disorder related to lead exposure, Dr. M.F. Bouchard & Dr. van Wijngaarden, *Arch Gen Psychiatry*. 2009; 66:1313-1319

(587) Zhu CB, et al, *Neuropsychopharmacology*, 2006, 31:2121-2131; & (b) McNally L, et al, *CNS Spectr* 2008, 13:6 & (c) Smith SE, et al, *J Neurosci* 2007, 27:10695-10702.

(588) Hypericin as glutamate control, Chang Y and Wang SJ, *Eur J Pharmacol* 2010, 634:53-61.

(589) (a) R Dantzer, et al, *Nat Rev Neurosci* 2008, 9:45-56; & (b) C Pittenger, et al, *CNS Neurol Disord Drug Targets*, 2007, 6:101-115; & (c) G Rajkowska, et al, *CNS Neurol Disord Drug Targets*, 2007, 6:219-233.

(590) Fava M, Giannelli A, Rapisarda V, Patrاليا A, Guaraldi GP. Rapidity of onset of the antidepressant effect of parenteral S-adenosyl-L-methionine. *Psychiatry Res* 1995 Apr 28; 56(3):295-7; & Rosenbaum JF, Fava M, Falk WE, Pollack MH, Cohen LS, Cohen BM, Zubenko GS. The antidepressant potential of oral S-adenosyl-l-methionine. *Acta Psychiatr Scand* 1990 May; 81(5):432-6; & Kagan BL, et. al.: Oral S-adenosylmethionine in depression: a randomized, double blind, placebo-controlled trial. *Am J Psychiatry* 1990; 147(5):591-5.

⁴⁹Internet: "<http://www.vrp.com>".

(591) Levine J. Controlled trials of inositol in psychiatry. *Eur Neuropsychopharmacol* 1997 May; 7(2):147-55; & Inositol versus placebo augmentation of serotonin reuptake inhibitors in the treatment of obsessive-compulsive disorder: a double-blind cross-over study. *Inositol versus placebo augmentation of serotonin reuptake inhibitors in the treatment of obsessive-compulsive disorder: a double-blind cross-over study. Int J Neuropsychopharmacol* 1999 Sep; 2(3):193-195; & Palatnik A, Frolov K, Fux M, Benjamin J. Double-blind, controlled, crossover trial of inositol versus fluvoxamine for the treatment of panic disorder. *J Clin Psychopharmacol* 2001 Jun; 21(3):335-9; & Chengappa KN, Levine J, Kupfer DJ. Inositol as an add-on treatment for bipolar depression. *Bipolar Disord* 2000 Mar; 2(1):47-55

(592) Should Depressive Syndromes Be Reclassified as “Metabolic Syndrome Type II”? *Ann Clin Psychiatry*. 2007 Oct-Dec; 19(4):257-64. McIntyre RS, Soczynska JK, Kennedy SH et al; & Inflammation, depression and dementia: are they connected? *Neurochem Res*. 2007 Oct; 32(10):1749-56. Epub 2007 Aug 20 Leonard BE.

(593) Vaccines, Depression and Neurodegeneration After Age 50, By Russell L. Blaylock, www.flcv.com/vax

(594) Immunoexcitotoxicity, R L Blaylock, *Alt Ther Health Med*, 2008, 14:46-53; & (b) Beat Depression and Anxiety with Diet/Nutrition, Blaylock Report, Dec 2010; & (c) Microglial Activation and Neurodegeneration, Dr. Russell Blaylock, www.blaylockwellnesscenter.com⁵¹; & (d) Nutrition and Behavior (DVD), Dr. Russell Blaylock, www.blaylockwellnesscenter.com; & (e) Sudden Cardiac Death and Excitotoxic Foods, Dr. Russell Blaylock, www.blaylockwellnesscenter.com⁵²

(596) How I Beat Depression through Diet: www.squidoo.com/i_beat_depression⁵³ & *The Paleo Diet: Lose Weight and Get Healthy by Eating the Food You Were Designed to Eat*⁵⁴ by Loren Cordain

(597) Effects of mercuric chloride on glucose transport in 3T3-L1 adipocytes. *Toxicol In Vitro*. 2005 Mar; 19(2):207-14. Barnes DM, Kircher EA; & Effects of inorganic HgCl₂ on adipogenesis. *Toxicol Sci*. 2003 Oct; 75(2):368-77. Epub 2003 Jul 25, Barnes DM, Hanlon PR, Kircher EA; & (b) Heavy metal-induced inhibition of active transport in the rat small intestine in vitro. Interaction with other ions. *Comp Biochem Physiol C*. 1986; 84(2):363-8, Iturri SJ, Peña A; & Interaction of the sugar carrier of intestinal brush-border membranes with HgCl₂. *Biochim Biophys Acta*. 1980 May 8; 598(1):100-14, Klip A, Grinstein S, Biber J, Semenza G.

(598) Overcoming Depression, Dr. Russell Blaylock, *The Blaylock Wellness Report*, Vol 5, No. 3, March 2008, & Food Additives, What you eat can kill you, Vol 4, No. 10, www.blaylockreport.com⁵⁵

(599) High fructose consumption combined with low dietary magnesium intake may increase the incidence of the metabolic syndrome by inducing inflammation. *Magnes Res*. 2006 Dec; 19(4):237-43. Rayssiguier Y, Gueux E, et al; & (b) Dietary magnesium and fiber intakes and inflammatory and metabolic indicators in middle-aged subjects from a population-based cohort. *Am J Clin Nutr*. 2006 Nov; 84(5):1062-9 Bo S, Durazzo M, Pagano G. et al; & (c) Hypomagnesemia, oxidative stress, inflammation, and metabolic syndrome. *Diabetes Metab Res Rev*. 2006 Nov-Dec; 22(6):471-6. Guerrero-Romero F, Rodríguez-Morán

(600) B.Windham, Health Effects of Mercury/Amalgam and Results after Replacement of Amalgam Fillings. (contains over 3000 medical study references and approx. 60,000 cases of amalgam replacement documenting recovery from 40 chronic health conditions, as documented by the treating

⁵⁰**Internet:** “<http://www.flcv.com/vaxinfla.html>”.

⁵¹**Internet:** “<http://www.blaylockwellnesscenter.com>”.

⁵²**Internet:** “<http://www.blaylockwellnesscenter.com>”.

⁵³**Internet:** “http://www.squidoo.com/i_beat_depression”.

⁵⁴**Internet:** “<http://www.amazon.com/gp/redirect.html%3FASIN=0471267554%26tag=squidoo8454-20%26lcode=xm2%26cID=2025%26ccmID=165953%26location=/o/ASIN/0471267554%253FSubscriptionId=19BAZMZQFZJ>”.

⁵⁵**Internet:** “<http://www.blaylockreport.com>”.

doctor or dentist. www.flcv.com/amalg6.htm⁵⁶

(601) B. Windham, Autism, PDD - the mercury connection, www.flcv.com/kidshg.html⁵⁷

(602) Mercury exposure levels from dental amalgam - review, B Windham (Ed), www.flcv.com/damspr1.htm

(603) Effects of prenatal and neonatal mercury exposures on the fetus and infants, B Windham (Ed), www.flcv.com/fetaln.htm⁵⁹

(604) Neurological effects of toxic metal exposures, B Windham (Ed), www.flcv.com/tmlbn.html⁶⁰

(605) Health Effects of Root-Canal Teeth and Cavitations: Review www.flcv.com/damspr11.html⁶¹ & www.flcv.com/RHealth.html⁶²

NOTE: all references not included here can be found in (600). You can find abstracts of the medical studies at the National Library of Medicine. National Institute of Health (Medline) and obtain the papers there. (<http://www.nlm.nih.gov/>)

%%%%%%%%%

Mercury impairs alfa-1-adrenergic receptors, astrocytic dopamine uptake, and serotonergic 5-HT2 receptor. The last one is stimulated by cocaine and LSD, so at least those drugs may be abused more due to mercury. We can remember that PhD Alfred Stock, leading early century mercury/chelator chemist stated that only cocaine was able to reverse his mental impairments form mercury, which as a chemist was easily available, and it was also legal at the time yet, in the early century.

%%%%%%%%%

Psychometric Evidence that Dental Amalgam Mercury may be an Etiological Factor in Manic Depression. Siblingrud, Motl and Kienholz. J. Orthmol Med. vol 13 no 1 p 31 ff (1998). MMPI-2 scores for 11 subjects with amalgams removed vs 9 with amalgams in.

&&&&&&&&&&&&&&&&&&&&&&&&&

Many of my patients reported the lifting of depression, anxiety, moodiness within a very short time of the total mercury decontamination of their mouths. I do not know the mechanism for that, and I am reporting this point so that those able to study the link between psychiatric illness and mercury would tell me one day what the mechanism is. The question here is that mercury, though out of the mouth, is not out of the brain in such a short time (two wks.) so, could these psychiatric illnesses be caused by the galvanic currents alone? I do not know.

Virtually 100% of the dozens of patients I've had suffering depression improve within 2 wks. One patient, who was depressed before amalgam removal, told me today that shw now has a positive attitude to life that she did not have before, and that she feels like a child!

Kindest regards. Hesham. DDS
Hesham El-Essawy [pop@EL-ESSAWY.COM]

⁵⁶**Informative:** "Mercury Exposure Levels from Amalgam Dental Fillings; Documentation of Mechanisms by Which Mercury Causes over 30 Chronic Health Conditions; Results of Replacement of Amalgam Fillings; and Occupational Effects on Dental Staff".
⁵⁷**Informative:** "Neurological and Immune Reactive Conditions Affecting Kids: The mercury connection to neurological pervasive developmental disorders (**autism, schizophrenia, dyslexia, ADD, childhood depression, learning disabilities, OCD, etc.**) and developmental immune conditions (**eczema, asthma, and allergies**)".
⁵⁸**Informative:** "Dental Amalgam Mercury Solutions".
⁵⁹**Informative:** "Infertility, Birth Defects, and Fetal Developmental Effects Related to Mercury from Amalgam Dental Fillings & Other Toxins".
⁶⁰**Informative:** "Effects of Toxic Metals on Learning Ability and Behavior".
⁶¹**Informative:** "Dental Amalgam Mercury Syndrome - DAMS".
⁶²**Internet:** "<http://www.flcv.com/RHealth.html>".

(This was was mostly snipped from a much larger paper (600) with over 3000 medical study references regarding common toxic exposures to mercury that are affecting large numbers of people with neurological effects)