



DEPRESSION AND OTHER NEUROTRANSMITTER RELATED CONDITIONS: THE MERCURY CONNECTION

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CONTENTS

Section	Page
I. Introduction	2
II. Causes of depression and anxiety	3
III. Mercury exposure levels from amalgam and other sources	7
IV. Toxic and immune reactive effects of mercury	8
V. The danger of vaccinations	13
VI. Treatment of depression	13
VII. References	15

I. Introduction

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).

Several factors appear to be contributing to this:

1. Neurological birth defects and developmental conditions due to increased levels of vaccinations, foetal exposure to alcohol, tobacco smoke, drugs, toxic metals such as lead, mercury and cadmium in addition to other neurotoxic chemicals such as pesticides and nitrates and other endocrine system/hormonal system disrupting chemicals such as dioxins. Studies by the National Academy of Sciences 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 (594), 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 7,289, coming for a regular visit were asked to participate in a study of the prevalence of depressive disorders (6). 71% of the sample was 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, generalised 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).

II. 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, 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. 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 (583).

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).

Inflammatory processes and depression

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, 596-599, etc.) Over-exposure to heavy metals such as lead and mercury have also been shown to induce anxiety or depression (386a).

Oxidative damage and depression

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 such conditions as metabolic syndrome, cognitive decline, cardiovascular disease, depression and cancer.

Nitric oxide related toxicity caused by peroxynitrite formed by the reaction of NO with superoxide anions, which results in nitration of tyrosine residues in neurofilaments and manganese Superoxide Dismutase (SOD) has been found to cause inhibition of the mitochondrial respiratory chain, inhibition of the glutamate transporter, and glutamate-induced neurotoxicity involved in ALS (521, 524).

Metal toxicity and depression

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 (43, 599). These are additional mechanisms by which mercury and toxic metals are factors in metabolic syndrome and insulin resistance and conditions such as diabetes and depression (15a, 43, 196, 338, 597).

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).

Mercury and other toxic metals inhibit astrocyte function in the brain and CNS (119), causing increased glutamate and calcium related neurotoxicity (119, 333, 416, 496). Mercury and increased glutamate activate free radical forming processes like xanthine oxidase which produce oxygen radicals and oxidative neurological damage (13, 142).

These inflammatory processes damage cell structures including DNA, mitochondria, and cell membranes. They also activate microglia cells in the brain, which control brain inflammation and immunity. Once activated, the microglia secrete large amounts of neurotoxic substances such as glutamate, an excitotoxin, which adds to inflammation and stimulates the area of the brain associated with anxiety (598). Inflammation also disrupts brain neurotransmitters resulting in reduced levels of serotonin, dopamine, and norepinephrine. Some of the main causes of such disturbances that have been documented include vaccines, mercury, aluminum, other toxic metals, MSG and aspartame (593, 598, 600, etc.)

Thyroid function and depression

Hormone imbalance has been found to be a common factor in depression (488). Imbalances in DHEA and cortisol may underlie depression, particularly when stress and obesity are present, and thyroid imbalances have also been found to cause depression (386a). Oestrogen 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 sub-fertility, infertility, spontaneous abortion, placental abruption, preterm delivery, gestational hypertension, pre-eclampsia, 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, hypothyroidism 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 neuro-imaging 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% v 20.9%, $p = 0.006$). Anxiety symptoms were also more frequent in the hypothyroid group.

Postpartum thyroiditis

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 hypothyroxinaemia 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 hypothyroxinaemia constitutes a serious risk factor for neuro-developmental 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).

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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 person's past medical history, diet, blood tests, hair tests and so on 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, arsenic, and pesticides that have similar effects and effects are synergistic or cumulative.

III. Mercury Exposure Levels From Amalgam And Other Sources

Amalgam fillings have been documented to leak significant levels of mercury continuously due to high vapour 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 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 foetus 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 to the foetus 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 preservative 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 and aluminum which have also been found to commonly cause significant neurological effects (604).

The top 3 toxic substances affecting large numbers of people in the U.S. adversely according to the US Environmental Protection Agency (EPA) and the Agency for Toxic

Substances and Disease Registry (ATSDR) are mercury, lead, and arsenic (600, 604).

IV. Toxic And Immune Reactive Effects Of Mercury

Mercury:

- Is neurotoxic (kills or damages brain and nerve cells) (19, 27, 34, 36, 39, 43, 69, 70, 147, 148, 175, 207, 211, 262, 273, 274, 291, 295, 301, 303, 327, 329, 395, 600)
- Generates high levels of reactive oxygen species (ROS) and oxidative stress and 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, 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, 255, 257, 285, 288, 333, 438).
- Causes systemic methylation deficiencies (88), which are documented to commonly be a factor in chronic conditions such as depression and autism (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 or rage, self-effacement, suicidal thoughts and lack of strength or force to resolve doubts or resist obsessions or compulsions.

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)
- Serious neurological diseases such as MS, ALS, Parkinson's and Alzheimer's diseases (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 vapour has been found to cause chronic depression, anxiety, and obsessive-compulsive behaviour (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, 254, 451, 465). Low serotonin levels and/or hypoglycaemia have also been found in the majority of those with impulsive and violent behaviour (481,482).

Mercury (and other toxic metals) has been found to accumulate in the pineal gland and reduce melatonin levels and this is thought to be a significant factor in mercury's toxic effects (569). Melatonin has been shown to have a significant protective action against methyl mercury toxicity, likely from the 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).

There is also evidence that mercury affects neurotransmitter levels which have effects on conditions such as depression, mood disorders and ADHD. There is evidence that mercury can block the dopamine- β -hydroxylase (DBH) enzyme (571). This enzyme synthesises 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 and this is thought to be a significant factor in mercury's toxic effects (569).

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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). Evidence suggests *Candida albicans* may activate depressive symptoms and fatigue by promoting ethanol production, a known central nervous system depressant.

Behaviour 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 (285, 404b). Mercury inhibits macrophage and neutrophil defense against candida by its effects 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 (33, 56, 280, 294, 333). Medical texts on neurology (27, 295) point out that chronic mercurialism is often not recognised 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 and depression 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 (34, 158, 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 (60, 313, 413, 600). As mercury levels are reduced the protein binding is reduced and improvement in the enzymatic process occurs (5).

Additional cellular level enzymatic effects of mercury's binding with proteins include blockage of sulphur oxidation processes and neurotransmitter amino acids (5, 33, 114, 438), enzymatic processes involving vitamins B6 and B12 (5, 418), effects on the cytochrome-C energy processes (35, 232), along with mercury's adverse effects on cellular mineral levels of calcium, magnesium, zinc, chromium, and lithium (38, 43, 96, 198, 333, 386, 427, 432, 484).

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

Numerous studies have found long term chronic low doses of mercury cause neurological, memory, behaviour, 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). Neurological effects have been documented at very low levels of exposure (urine Hg < 4 mcg/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 foetal 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, noradrenaline and acetylcholinesterase (35, 107, 140, 141, 175, 251, 254, 288, 290, 296, 305, 372, 412, 451, 465). 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 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, 212, 222, 229, 230, 233, 271, 294, 317, 320, 322, 376, 407)
- Schizophrenia (34, 35, 294)
- Insomnia (94, 95, 212, 222, 271, 317, 322, 376, 407)
- Anger (102, 212, 233, 320, 407)
- Anxiety & mental confusion (57, 94, 95, 212, 222, 229, 233, 271, 317, 320, 322, 407) memory disorders (94, 95, 222, 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, and the majority treated for these at clinics that deal with such conditions have either recovered or shown significant improvement (552, 600, 601). 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). Whereas 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).

V. 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 and Parkinson’s disease (593, 598, 600, 601, 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).

VI. 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 filled teeth and jawbone cavitations, which likewise are highly inflammatory and can have major impacts on the immune system and health (605). Reducing glutamate levels and blocking glutamate receptors can significantly improve depression (598).

Lifestyle and diet

Diet and lifestyle are important factors in preventing or controlling depression. One should avoid alcohol, sugar, caffeine, and inflammatory substances such as MSG and high-fructose corn syrup (580,598). Reduce stress and get regular exercise. Yoga and meditation have been found to be helpful for many.

Supplementation

B Vitamins and magnesium deficiencies have been found to be factors in depression and anxiety. Supplementation to assure proper levels is beneficial in treatment (583). 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 (580, 590). 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 (580). Other nutrients found to cause depression when low or to usually be low in depression or to be effective additions in treating depression include omega-3 fatty acids (EPA/DHA), ginkgo biloba, DHEA, natural progesterone, pregnenolone, DMAE, L-Carnitine, NADH, Phenylalanine, Folic Acid, Vit B12 (cobalamine), B6, other B vitamins, choline, vitamin D, vitamin C, potassium and testosterone in men over 40 (580, 582). 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).

Lower levels of fish oil (EPA) have been found to be significantly related to depression. 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).

Anxiety Disorders

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 to burying feelings (583). Panic disorder is characterised by repeated

episodes of intense fear and affects 3 to 6 million people in the US. Obsessive-Compulsive Disorder (OCD) is characterised by anxious thoughts and uncontrollable ritualistic behavior and 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 and affect over 10% of the population.

Generalized Anxiety Disorder (GAD) is chronic, daily worrying about health, finances, work and family. 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 and prolonged stress can have debilitating effects. Two conventional non-pharmaceutical treatments for anxiety are behavioural therapy (breathing techniques, exposure therapy, etc.) and cognitive therapy (modification of thinking patterns).

As previously noted, 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. Hypoglycaemia may be a factor in some anxiety disorders so it is advisable to eat more frequent, small quantities including protein and nuts. 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 and biofeedback are also often helpful.

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

VII. References

- (5) Consensus paper of the WFSBP Task Force on Biological Markers: Biological Markers in Depression, R. Mossner et al, *The World Journal of Biological Psychiatry*, 2007; 8(3): 141-174;
http://wfsbpverband.globit.com/fileadmin/pdf/guides/WFSBP_Consensus_Paper_Biological_Markers_in_Depression.pdf
- (6) Drózd W, Wojnar M et al. [The study of the prevalence of depressive disorders in primary care patients in Poland], *Wiad Lek.* 2007; 60(3-4): 109-13.
- (7) Zárata A, Basurto L et al. Thyroid malfunction in women; *Ginecol Obstet Mex.* 2001 May; 69: 200-5; & (b) Wier FA, Farley C L. Clinical controversies in screening women for thyroid disorders during pregnancy. *J Midwifery Women's Health.* 2006 May-Jun; 51(3): 152-8.
- (8) Stagnaro-Green A; 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 & (b) Harris B. Postpartum depression and thyroid antibody status. *Thyroid.* 1999 Jul; 9(7): 699-703
- (9) Kooistra L, Crawford S, van Baar AL, Brouwers EP, Pop VJ. Neonatal effects of maternal hypothyroxinemia during early pregnancy. *Pediatrics.* 2006 Jan; 117(1): 161-7.
- (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) Almeida C, Brasil MA, Costa AJ et al. Subclinical hypothyroidism: Psychiatric disorders and symptoms. *Rev Bras Psiquiatr.* 2007 Jun; 29(2): 157-9
- (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*, 199, 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 S J, Bagchi D. Oxidative mechanisms in the toxicity of metal ions. *Free Radic Biol Med* 1995; 18(2): 321-36; & Pocerlich 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 E D, Lander E S, Reactive oxygen species have a causal role in multiple forms of insulin resistance, *Nature*, 2006, Apr 13; 440(7086): 944-8; & Meigs J B, Larson M G, et al, Association of oxidative stress, insulin resistance, and diabetes risk phenotypes: the Framingham Offspring Study, *Diabetes Care.* 2007, Oct; 30(10): 2539-35
- (33) B. Windham, Multiple Sclerosis (MS): The mercury connection; www.flcv.com/ms.html

- (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 H A, Levy, T E, Uniformed Consent: The hidden dangers in dental care, 1999, Hampton Roads Publishing Company Inc; & Hal Huggins, It's 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 Y M, Teh W F, Wong M K, Reddy P K "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, 1991; 65(2): 164-7.
- (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, 1994; 153: 607-610
- (66) B. Windham, Alzheimer's Disease: The mercury connection, www.flcv.com/alzhg.html ; (over 150 peer-reviewed medical studies)
- (72) D. L. Smith, "Mental effects of mercury poisoning" South Med J, 1987; 71: 904-5
- (74) A. C. Bittner et al, "Behavior effects of low level mercury exposure among dental professionals", Neurotoxicology & Teratology, 1998, 20(4): 429-39
- (88) Waly M, Olteanu H, Deth R C et al. Activation of methionine synthase by insulin-like growth factor-1 and dopamine: A target for neurodevelopmental toxins and thimerosal, Mol Psychiatry, Apr 2004; 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, 1996; 24(5): 355-356
- (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, H J "Elimination of symptoms by removal of dental amalgam from mercury poisoned patients", J Orthomol Med, 1993; 8: 145-148; & 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.html (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*, 1994; 74(1); & *Amer. J. Of Psychotherapy*, 1989; 58: 575-87; & *Poisoning and Toxicology compendium*, Leikin & Palouchek, Lexi-Comp, 1998, p705
- (108) M. Henningsson et al "Defensive characteristics in individuals with amalgam illness", *Acta Odont Scand*, 1996; 54(3): 176-181
- (109) Y. X. Liang et al "Psychological effects of low exposure to mercury vapor" *Environmental Med Research*, 1993; 60(2): 320-327; & T. Kampe et al, "Personality traits of adolescents with intact and repaired dentitions" *Acta Odont Scand*, 1986; 44: 95-99; & R. Kishi et al, "Residual neurobehavioral effects of chronic exposure to mercury vapor", *Occupat Envir. Med.*, 1994, 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 R L, Schatz J R, Hurst R, Kagen R. Toxic heavy metal ions inhibit reduction of disulfide bonds. *J Biol Chem* 1991; 266(19): 12695-702; Boot J H. Effects of SH-blocking compounds on the energy metabolism in isolated rat hepatocytes. *Cell Struct Funct* 1995; 20(3): 233-8; & Baauweegers H G, 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, 3(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*, 1992; 49: 233-240; & (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) . Tirado V, Garcia MA et al; [Neuropsychological disorders after occupational exposure to mercury vapors in El Bagre (Antioquia, Colombia)] *Rev Neurol*. 2000 Oct 16-31; 31(8): 712-6; & (f) Haut M W, Morrow L A et al. Neurobehavioral effects of acute exposure to inorganic mercury vapor. *Appl Neuropsychol*. 1999; 6(4): 193-200 & (g) Kopal Grum D, Kopal AB et al. Personality traits in miners with past occupational elemental mercury exposure. *Environ Health Perspect*, Feb 2006; 114(2): 290-6
- (122) B. Ono et al, "Reduced tyrosine uptake in strains sensitive to inorganic mercury", *Genet*, 1987, 11(5): 399-403
- (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*, 1993; 68(3): 307-310 & 69(3): 305
- (142) Ariza M E; Bijur G N; Williams M V. Lead and mercury mutagenesis: Role of H₂O₂, superoxide dismutase, and xanthine oxidase. *Environ Mol Mutagen* 1998; 31(4): 352-61
- (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

(181) P. W. Mathieson, "Mercury: god of TH2 cells" *Clinical Exp Immunol*, 1995; 102(2): 229-30

(196) Gowdy & Demes, 1978, in B. Wolfe and P. Wolfe, "Fillings, Mercury, and You" *Mothering* magazine, Summer, 1987.

(198) Fabbri E, Caselli F, Piano A, Sartor G, Capuzzo A. Cd²⁺ and Hg²⁺ affect glucose release and cAMP-dependent transduction pathway in isolated eel hepatocytes. *Aquat Toxicol*. 2003 Jan 10; 62(1): 55-65; & Bapu C, Purohit RC, Sood PP; & E.S. West et al. Fluctuation of trace elements during methylmercury toxication and chelation therapy. *Hum Exp Toxicol*. 1994 Dec; 13(12): 815-23, *Textbook of Biochemistry*, MacMillan Co, 1957, p853

(199) Dr. P. Kraub & M. Deyhle, Universitat Tubingen- 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, Tubingen Univ. Gynecological Clinic, Heidelberg, 1996

(207) Pendergrass J C, Haley B E, Univ. Of Kentucky Dept. Of Chemistry "The Toxic Effects of Mercury on CNS Proteins: Similarity to Observations in Alzheimer'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: 461-64

(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. Sibley, 86 Colorado patients, Dr. A. V. Zamm, 22 patients)

(222) M. Dauderer, "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)

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

(233) Sven Langworth et al, "Amalgamnews and Amalgamkadebunden, 1997 and Svenska Dogbladet, 1997 (286 cases); & F. Berglund et al. "Improved health after removal of dental amalgam fillings", *Swedish Assoc. Of Dental Mercury Patients*, 1998. (www.tf.nu) (over 1,000 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, 25(3/4): 230-33

(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* 1986; 62(724): 113-123; & A. D. Kay et al, "Cerebrospinal fluid biopterin is decreased in Alzheimer's disease" *Arch Neurol*, Oct 1986; 43(10): 996-9; & T. Yamiguchi et al, "Effects of tyrosine administration on serum biopterin In patients with Parkinson's Disease and normal controls", *Science*, Jan 1983; 219(4580): 75-77; & T. Nagatsu et al, "Catecholamine-related enzymes and the biopterin 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*, 1993; 40(2-3): p235-40; & "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*, 3 Mar 1998, 95(5): 2642-2647

(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*, 1972, 28: 1455-1456

(285) R. C. Perlingeiro et al, "Polymorphonuclear phagocytosis in workers exposed to mercury vapor", *Int J Immunopharmacology*", 1994; 16(12): 1011-7; & Mathieson P. W. 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*. Feb 1990; 47(2): 105-9. Soleo L, Urbano M L, 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 C S. 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 S J, Reynolds J N, Racz W J. 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 A M; Cherian M G. Effects of heavy metal cations, sulfhydryl reagents and other chemical agents on striatal D2 dopamine receptors. Biochem Pharmacol Oct 1985; 34(19): 3405-13; Lewis R N; 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 B M, 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.htm
- (295) Cecil Textbook of Medicine, 20th Ed., Bennett & Plum, W.B. Saunders and Company, Philadelphia, 1996, p 69; & Comprehensive Psychiatry, 1977; 18(6); p595-598, & Poisoning & Toxicology Compendium, Leikin and Palouchek, 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 disease", J. Neural Transm, 1998, 105(1): 59-68.
- (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 C C, Syed N I, Lorscheider F L. 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; 16(1); 31-40
- (317) S. Zinecker, "Amalgam: Quecksilberdamfe 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 amalgamentfernug", Separatdruck aus Schweiz. Monatsschr Zahnm. 1998, 108(8) (75 cases amalgam removal) <http://soho.globalpoint.ch/paul-engel>
- (330) B. Windham, ALS: The mercury connection, www.flcv.com/als.html; over 100 peer-reviewed medical study references
- (331) C. Gordon et al, "Abnormal sulphur oxidation in systemic lupus erythematosus (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 G B, et al; Xenobiotic metabolism in motor neuron disease, *Neurology* 1990, 40: 1095-98.

(333) A. J. Freitas et al, "Effects of Hg^{2+} and CH_3Hg^+ on Ca^{2+} 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, 16(4): 325-30; & E. Chavez et al, "Mitochondrial calcium release by Hg^{2+} ", *J Biol Chem*, 1988, 263: 8, 3582-86; 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 A D et al, Modifications of Ca^{2+} 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 T F 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*. 1977, 13: 470- 477; & (d) Boot J H. 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 V D M 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 V D; The beneficial effect of amalgam replacement on health in patients with autoimmunity. *Neuro Endocrinol Lett*. 2004 Jun; 25(3): 211-8 www.melisa.org

(372) Atchison W D. 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 G S, Chandra S V, Effect of interaction of Mn^{2+} with Zn^{2+} , Hg^{2+} , and Cd^{2+} 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 S N et al, Tryptophan depletion causes a rapid lowering of mood in normal males. *Psychopharmacology*, 1985, 87(2): 173-77; & Smith K A et al, Relapse of depression after depletion of tryptophan, *Lancet* 1997, 349(9056): 915-19; & Delgado P L et al, Serotonin function, depletion of plasma tryptophan, and the mechanism of antidepressant action. *Arch Gen Psychiatry* 1990, 47(5): 411-18.

(375) Stejskal V D M, 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 P, Prochazkova J, Bartova J, Reactions to metals in patients with chronic fatigue and autoimmune endocrinopathy. *Vnitřní Lek*, Sep 1999; 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*, 1993, 45(2): 75-77
- (386) Genova Diagnostics [click: Tests, Search by Disease, see Disease in question & Heavy Metal Toxicity], www.genovadiagnostics.com/; & 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, Imi.analyslab@swipnet.se <http://home.swipnet.se/misac/research11.html#biospectrons>
- (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 M A, Costa A J, 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 J D, Tremont G. *Minerva Endocrinol*. 2007 Mar; 32(1): 49-65.
- (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.* 1986, 1(4): pp261-5
- (407) Eedy D J, Burrows D, Dliford T, Fay A. Elevated T cell subpopulations in dental students. *J Prosthet Dent* 1990; 63(5): 593-6; & Yonk L J et al, CD+4 helper T-cell depression in autism. *Immunol Lett*, 1990, 25(4): 341-5; & Jaffe J S, Strober W, Sneller M C, Functional abnormalities of CD8+ T cells define a unique subset of patients with common variable immunodeficiency. *Blood* 1993, 82(1): 192-20.
- (408) Yonk L J et al, CD+4 helper T-cell depression in autism. *Immunol Lett*, 1990, 25(4): 341-5; & Jaffe J S, Strober W, Sneller M C, 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 911 : 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 N C, Pearson C M.

Dipeptyl Peptidases in human muscle disease. *Clin Chim Acta* 1978; 82(1-2): 185-92; & Seroussi K, *Autism and Pervasive Developmental Disorders*, 1998, p174.

(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 M L. Is infantile autism a hypoglutamatergic disorder? *J Neural Transm* 1998, 105(4-5): 525-35; & (b) Rolf L H, Haarman F Y, Grotemeyer K H, 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 S B, Cantor D S. Autism: Xenobiotic influences. *Toxicol Ind Health* 1998; 14(4): 553-63; & Liska, D J. The detoxification enzyme systems. *Altern Med Rev* 1998. 3(3): 187-98

(416) Kim P, Choi B H. "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 C K, Roy S, Packer L. Cellular and mitochondrial changes in glutamate-induced HT4 neuronal cell death *Neuroscience*. 2000; 97(3): 531-41.

(418) Srikantaiah M V; Radhakrishnan A N. 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 M R. Nutritional influences on metal toxicity. *Environ Health Perspect* 1979; 29: 95-104

(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 C S, 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*, 1985; 4(5-6): 325-332

(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, 27(7), 535-536; & Devinsky et al. Aggressive Behavior Following Exposure to Cholinesterase Inhibitors. *Journal of Neuropsychiatry*, Spring 1992; 4(2): 189-199.

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

(465) Walsh W J, Health Research Institute, *Biochemical Treatment of Mental Illness and Behavior*

Disorders, Minnesota Brain Bio Assoc, Nov 17, 1997; <http://www.hriptc.org/Minnesota.htm>; & William J. Walsh, Laura B. Glab, and Mary L. Haakenson; Pfeiffer Treatment Center, Biochemical Therapy and Behavior Outcomes; 2000, <http://www.hriptc.org/btbres.htm>

(480) Salzer H M, Relative hypoglycemia as a cause of neuropsychiatric illness, *J National Med Assoc*, 1996; 58(1): 12-17; & Heninger G R 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 M O; 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) Pranjic N, Sinanovic O, et al. 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.

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

(483) Thomas D E et al, Tryptophan and nutritional status in patients with senile dementia, *Psychological Med* 1986; 16: 297-305; & Yaryura-Tobias J A et al, Changes in serum tryptophan and glucose in psychotics and neurotics, *Nutrition*, 4557: 1132; & Carney M W P, *Brit Med J*, 1967, 4: 512-516.

(484) Urberg M, Zemel M B; 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 R A 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 M W; Morrow L A; Pool D; Callahan T S; Haut J S; Franzen M D. 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/depression_01.htm

(490) Fava M, Giannelli A, Rapisarda V, Patrاليا A, Guaraldi G P. Rapidity of onset of the antidepressant effect of parenteral S-adenosyl-L-methionine. *Psychiatry Res* 1995 Apr 28;56(3):295-7; & Rosenbaum J F, Fava M, Falk W E, Pollack M H, Cohen L S, Cohen B M, Zubenko G S. 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 K N, Levine J, Kupfer D J. Inositol as an add-on treatment for bipolar depression. *Bipolar Disord* 2000 Mar; 2(1): 47-55

- (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-Talarmin 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 K F, Atkinson A, Khan A T. 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.
- (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-Talarmin Y M. Inhibition of acetylcholine synthesis and tyrosine nitration induced by peroxynitrite are differentially prevented by antioxidants. *Mol Pharmacol* 2001 Oct; 60(4): 838-46
- (534) Tirado V, Garcia M A, Franco A., Neuropsychological disorders after occupational exposure to mercury vapors, *Rev Neurol* 2000 Oct 16-31; 31(8): 712-6; & Powell T J. Chronic neurobehavioural effects of mercury poisoning on a group of chemical workers. *Brain Inj* 2000 Sep; 14(9): 797-814
- (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.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 H Y, 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 H H, Mortensen P B, Jensen I W. Depression and magnesium deficiency. *Int J Psychiatry Med* 1989; 19(1): 57-63.
- (563) Naylor G J, Smith A H, Bryce-Smith D, Ward N I. Elevated vanadium content of hair and mania. *Biol Psychiatry*1984; 19(5): 759-764.
- (567) Kim C Y, 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 J C, Seegal R F; 2000, PCBs and methylmercury alter intracellular calcium concentrations in rat cerebellar granule cells. *Neurotoxicology*, 21(6): 1123-1134.
- (569) Baccarelli A, Pesatori A C, Bertazzi P A. 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 S M, Costa L G, 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, <http://www.life-enhancement.com/>

(581) Starck G, Carlsson M L, et al, 1H magnetic resonance spectroscopy study in adults with obsessive compulsive disorder: Relationship between metabolite concentrations and symptom severity, *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 M L, *Biol Psychiatry.* 2001 Jan; 25(1): 5-26

(582) Vitamin Research News, weekly journal (several editions), 2003-2009, www.vrp.com

(583) Dr. J. Teitelbaum, Health & Wellness Update, Issue 198, Jan 2009.

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

(590) Fava M, Giannelli A, Rapisarda V, Patralia A, Guaraldi G P. Rapidity of onset of the antidepressant effect of parenteral S-adenosyl-L-methionine. *Psychiatry Res* 1995 Apr 28;56(3):295-7; & Rosenbaum J F, Fava M, Falk W E, Pollack M H, Cohen L S, Cohen B M, Zubenko G S. The antidepressant potential of oral S-adenosyl-L-methionine. *Acta Psychiatr Scand* 1990 May; 81(5): 432-6

(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 K N, Levine J, Kupfer D J. Inositol as an add-on treatment for bipolar depression. *Bipolar Disord* 2000 Mar; 2(1): 47-55

(592) McIntyre R S, Soczynska J K, Kennedy S H et al; Should Depressive Syndromes Be Reclassified as "Metabolic Syndrome Type II"? *Ann Clin Psychiatry.* 2007 Oct-Dec; 19(4): 257-64. Inflammation, depression and dementia: are they connected? *Neurochem Res.* 2007 Oct; 32(10): 1749-56. Epub 2007 Aug 20 Leonard B E.

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

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

(597) Barnes D M, Kircher E A; Effects of mercuric chloride on glucose transport in 3T3-L1 adipocytes. *Toxicol In Vitro.* 2005 Mar; 19(2): 207-14; & Barnes D M, Hanlon P R, Kircher E A; Effects of inorganic HgCl₂ on adipogenesis. *Toxicol Sci.* 2003 Oct; 75(2): 368-77. Epub 2003 Jul 25; & (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; & Klip A, Grinstein S, Biber J, Semenza G. Interaction of the sugar carrier of intestinal brush-border membranes with HgCl₂. *Biochim Biophys Acta.* 1980 May 8; 598(1): 100-14,

(598) Overcoming Depression, Dr. Russell Blaylock, The Blaylock Wellness Report, March 2008; 5(3); & Food Additives, What you eat can kill you, 4(10); <http://www.blaylockreport.com/>

(599) Rayssiguier Y, Gueux E, et al; 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; & (b) Bo S, Durazzo M, Pagano G. et al; 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 & (c) Guerrero-Romero F, Rodríguez-Morán. Hypomagnesemia, oxidative stress, inflammation, and metabolic syndrome. *Diabetes Metab Res Rev.* 2006 Nov-Dec; 22(6): 471-6.

(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 doctor or dentist. www.flcv.com/amalg6.html

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

(602) Mercury exposure levels from dental amalgam: Review, B Windham (Ed), www.flcv.com/damspr1.html

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

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

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