Elektrosmog, Hormone und das metabolische Syndrom

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EMR exposure of humans

EMR exposure is a significant and frequent cause or contributor to the metabolic syndrome

4 sources of electromagnetic radiation (EMR)

- 1. household currents and appliances (creating both electric and magnetic fields)
- 2. Wireless technology (W-LAN)
- 3. Chordless phones (pulsed microwave)
- 4. incoming cell phone radiation (microwave)
Microwave-EMR

Saturday, 11 September 2010
EMR

Biochem J. 2007 Apr 25
Mechanism of a short-term ERK activation by electromagnetic fields at mobile phone frequency
Friedman J, Kraus S, Hauptman Y, Schiff Y, Seger R.

The exposure to non-thermal microwave electromagnetic field generated by mobile phones affects the expression of many proteins. This effect on transcription and protein stability can be mediated by the mitogen-activated protein kinase (MAPK) cascades, which serve as central signaling pathways, and govern essentially all stimulated cellular processes. Indeed, a long-term exposure of cells to mobile phone irradiation results in the activation of p38MAPKs as well as the ERK/MAPKs. Here we studied the immediate effect of irradiation on the MAPK cascades, and found that ERKs, but not stress related MAPKs are rapidly activated in response to various frequencies and intensities. Using signaling inhibitors we delineated the mechanism that is involved in this activation. We found that the first step is mediated in the plasma membrane by NADH oxidase, which rapidly generates reactive oxygen species (ROS). These ROS then directly stimulate matrix metalloproteinases and allow them to cleave and release heparin binding-EGF. This secreted factor, activates EGF receptor, which in turn further activates the ERK cascade. Thus, this study demonstrates for the first time a detailed molecular mechanism by which electromagnetic irradiation by mobile phones induces the activation of the ERK cascade and thereby induces transcription and other cellular...
Melatonin

- **Melatonin:**
- Melatonin is an amino acid hormone synthesized by the pineal gland in the brain. Melatonin controls the sleep-wake cycle of the body. Melatonin also controls the “Aging Clock” in our bodies. Melatonin increases sexual drive and energy, promotes heart health, improves immune system functioning and is a very powerful antioxidant.
Electro smog reduces melatonin production in the pineal gland. Why is this bad?

1. Melatonin induces sleep. We only heal and detoxify in deep non-rem sleep. Without melatonin no regeneration and no detoxification.

2. Melatonin is the most effective and potent neuroprotective chemical in the CNS and prevents damage from mercury, lead, aluminum, chemicals, mycotoxins, viruses, cigarette smoke, bacterial and parasitic endo-and exotoxins (Lyme, clostridia, ascaris) outgasing of carpets and new car plastics, etc.

Sener, G.et al: “Melatonin protects against mercury induced oxidative tissue damage”. Basic and Clinical Pharmacology & Toxicology Vol 93, Dec 2003, pp 290-296
Diagram of a cerebral capillary enclosed in astrocyte end-feet. Characteristics of the blood-brain barrier are indicated: (1) tight junctions that seal the pathway between the capillary (endothelial) cells; (2) the lipid nature of the cell membranes of the capillary wall which makes it a barrier to water-soluble molecules; (3), (4), and (5) represent some of the carriers and ion channels; (6) the enzymatic barrier that removes molecules from the blood; (7) the efflux pumps which extrude fat-soluble molecules that have crossed into the cells.
Cell phone use greater than 25 minutes per day for 13 days leads to decreased melatonin production

Melatonin metabolite excretion among cellular telephone users

- Burch JB, Reif JS, Noonan CW, Ichinose T, Bachand AM, Koleber TL, Yost MG.

- The relationship between cellular telephone use and excretion of the melatonin metabolite 6-hydroxymelatonin sulfate (6-OHMS) was evaluated in two populations of male electric utility workers (Study 1, n=149; Study 2, n=77).

MATERIALS AND METHODS: Participants collected urine samples and recorded cellular telephone use over 3 consecutive workdays. Personal 60-Hz magnetic field (MF) and ambient light exposures were characterized on the same days using EMDEX II meters. A repeated measures analysis was used to assess the effects of cellular telephone use, alone and combined with MF exposures, after adjustment for age, participation month and light exposure. RESULTS: No change in 6-OHMS excretion was observed among those with daily cellular telephone use >25 min in Study 1 (5 worker-days). Study 2 workers with >25 min cellular telephone use per day (13 worker-days) had lower creatinine-adjusted mean nocturnal 6-OHMS concentrations (p=0.05) and overnight 6-OHMS excretion (p=0.03) compared with those without cellular telephone use. There was also a linear trend of decreasing mean nocturnal 6-OHMS/creatinine concentrations (p=0.02) and overnight 6-OHMS excretion (p=0.08) across categories of increasing cellular telephone use. A combined effect of cellular telephone use and occupational 60-Hz MF exposure in reducing 6-OHMS excretion was also observed in Study 2.

CONCLUSIONS: Exposure-related reductions in 6-OHMS excretion were observed in Study 2, where daily cellular telephone use of >25 min was more prevalent. Prolonged use of cellular telephones may lead to reduced melatonin production, and elevated 60-Hz MF exposures may potentiate the effect.
Melatonin and caffeic acid phenethyl ester exert protective effects on mobile phone induced renal impairment in rats


Comparative analysis of the protective effects of melatonin and caffeic acid phenethyl ester (CAPE) on mobile phone-induced renal impairment in rat.


- Melatonin and caffeic acid phenethyl ester (CAPE), a component of honeybee propolis, were recently found to be potent free radical scavengers and antioxidants. There are a number of reports on the effects induced by electromagnetic radiation (EMR) in various cellular systems. Mechanisms of adverse effects of EMR indicate that reactive oxygen species may play a role in the biological effects of this radiation. The present study was carried out to compare the protective effects of melatonin and CAPE against 900 MHz EMR emitted mobile phone-induced renal tubular injury. Melatonin was administered whereas CAPE was given for 10 days before the exposure. Urinary N-acetyl-beta-D-glucosaminidase (NAG, a marker of renal tubular injury) and malondialdehyde (MDA, an index of lipid peroxidation), were used as markers of oxidative stress-induced renal impairment in rats exposed to EMR. Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) activities were studied to evaluate the changes of antioxidant status in renal tissue. Urinary NAG and renal MDA were increased in EMR exposed rats while both melatonin and CAPE caused a significant reduction in the levels of these parameters. Likewise, renal SOD and GSH-Px activities were decreased in EMR exposed animals while melatonin caused a significant increase in the activities of these antioxidant enzymes but CAPE did not. Melatonin caused a significant decrease in urinary NAG activity and MDA levels which were increased because of EMR exposure. CAPE also reduced elevated MDA levels in EMR exposed renal tissue, but the effect of melatonin was more potent than that of CAPE. Furthermore, treatment of EMR exposed rats with melatonin increased activities of SOD and GSH-Px to higher levels than those of control rats. In conclusion, melatonin and CAPE prevent renal tubular injury by reducing oxidative stress and protect the kidney from oxidative damage induced by 900 MHz mobile phone.

- Nevertheless, melatonin seems to be a more potent antioxidant compared with CAPE in kidney. (Mol Cell Biochem 276: 31-37, 2005).
Mobile phone radiation decreases pre-bedtime melatonin level


- Does evening exposure to mobile phone radiation affect subsequent melatonin production?
  - **Wood AW, Loughran SP, Stough C.**
- PURPOSE: To test whether exposure to the emissions from a digital mobile phone handset prior to sleep alters the secretion of melatonin. MATERIALS AND METHODS: In a double-blind cross-over design, 55 adult volunteers were both actively exposed or sham-exposed (in random order on successive Sunday nights) to mobile phone emissions for 30 min (0.25 W average power). Urine collection occurred immediately prior to retiring to bed and on rising the next morning. Melatonin output was estimated from principal metabolite concentrations (6-sulphatoxymelatonin (aMT6s) via radioimmunoassay), urine volumes and creatinine concentrations.
- RESULTS: Total melatonin metabolite output (concentration x urine volume) was unchanged between the two exposure conditions (active 14.1+-1.1 microg; sham 14.6+-1.3 microg). The pre- and post-bedtime outputs considered separately were also not significantly different, although the pre-bedtime value was less for active versus sham exposure. When melatonin metabolite output was estimated from the ratio of aMT6s to creatinine concentrations, the pre-bedtime value was significantly less ($p = 0.037$) for active compared to sham. Examination of individual responses is suggestive of a small group of 'responders'. CONCLUSIONS: Total nighttime melatonin output is unchanged by mobile phone handset emissions, but there could be an effect on melatonin onset time.
Melatonin and caffeic acid phenyl ester reduce retinal oxidative stress after long-term exposure to 900 MHZ emitting cell phone

Mol Cell Biochem. 2006 Jan;282(1-2):83-8

Protective effects of melatonin and caffeic acid phenethyl ester against retinal oxidative stress in long-term use of mobile phone: a comparative study.

- Ozguner F, Bardak Y, Comlekci S.

There are numerous reports on the effects of electromagnetic radiation (EMR) in various cellular systems. Melatonin and caffeic acid phenethyl ester (CAPE), a component of honeybee propolis, were recently found to be potent free radical scavengers and antioxidants. Mechanisms of adverse effects of EMR indicate that reactive oxygen species may play a role in the biological effects of this radiation. The present study was carried out to compare the efficacy of the protective effects of melatonin and CAPE against retinal oxidative stress due to long-term exposure to 900 MHz EMR emitting mobile phones. Melatonin and CAPE were administered daily for 60 days to the rats prior to their EMR exposure during our study. Nitric oxide (NO, an oxidant product) levels and malondialdehyde (MDA, an index of lipid peroxidation), were used as markers of retinal oxidative stress in rats following to use of EMR. Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) activities were studied to evaluate the changes of antioxidant status in retinal tissue.

Retinal levels of NO and MDA increased in EMR exposed rats while both melatonin and CAPE caused a significant reduction in the levels of NO and MDA. Likewise, retinal SOD, GSH-Px and CAT activities decreased in EMR exposed animals while melatonin and CAPE caused a significant increase in the activities of these antioxidant enzymes. Treatment of EMR exposed rats with melatonin or CAPE increased the activities of SOD, GSH-Px and CAT to higher levels than those of control rats. In conclusion, melatonin and CAPE reduce retinal oxidative stress after long-term exposure to 900 MHz emitting mobile phone. Nevertheless, there was no statistically significant difference between the efficacies of these two antioxidants against to EMR induced oxidative stress in rat retina. The difference was in only GSH-Px activity in rat retina. Melatonin stimulated the retinal GSH-Px activity more efficiently than CAPE did.
Serum melatonin in rats decreased by electric field exposure


- Electric field exposure alters serum melatonin but not pineal melatonin synthesis in male rats.
  - Grota LJ, Reiter RJ, Keng P, Michaelson S.
- Department of Psychiatry, University of Rochester School of Medicine and Dentistry, New York.
- Sprague-Dawley male rats, maintained in a 14:10 h light:dark cycle were exposed for 30 days (starting at 56 days of age) to a 65 kV/m, 60 Hz electric field or to a sham field for 20 h/day beginning at dark onset. Pineal N-acetyltransferase (NAT), hydroxy-indole-o-methyltransferase (HIOMT), and melatonin as well as serum melatonin were assayed. Preliminary data on unexposed animals indicated that samples obtained 4 h into the dark period would reveal either a phase delay or depression in circadian melatonin synthesis and secretion. Exposure to electric fields for 30 days did not alter the expected nighttime increase in pineal NAT, HIOMT, or melatonin. Serum melatonin levels were also increased at night, but the electric field-exposed animals had lower levels than the sham-exposed animals. Concurrent exposure to red light and the electric field or exposure to the electric field at a different time of the day-night period did not reduce melatonin synthesis. These data do not support the hypothesis that chronic electric field exposure reduces pineal melatonin synthesis in young adult male rats.
- However, serum melatonin levels were reduced by electric field exposure, suggesting the possibility that degradation or tissue uptake of melatonin is stimulated by exposure to electric fields.
Increased geomagnetic activity in combination with 60Hz magnetic fields led to decreased melatonin production

Neurosci Lett. 1999 May 14;266(3):209-12

• Geomagnetic disturbances are associated with reduced nocturnal excretion of a melatonin metabolite in humans.
  • Burch JB, Reif JS, Yost MG.
• Department of Environmental Health, Colorado State University, Fort Collins 80523, USA.
• The effects of geomagnetic disturbances on urinary excretion of the melatonin metabolite, 6-hydroxymelatonin sulfate (6-OHMS), were studied in conjunction with 60 Hz magnetic field (MF) and ambient light exposure in 132 electric utility workers. Geomagnetic activity was assessed using a local (equivalent amplitude or A(K), Boulder, CO) and global (average antipodal or aa) index. Personal exposures to 60 Hz MFs and light were obtained using data-logging meters. The relationship between geomagnetic activity and 6-OHMS was assessed with adjustment for age, light exposure, and month of participation. Mean overnight 6-OHMS excretion was lower on days when the 36-h A(K) or aa values exceeded 30 nT. A greater reduction in 6-OHMS excretion was observed when increased geomagnetic activity was combined with elevated 60 Hz MF or reduced ambient light exposures.
Human Growth Hormone (HGH)

HGH is the “master hormone” in the body. HGH is a peptide hormone secreted by the pituitary gland that influences the growth and development of almost all tissues and organs in the body. Most of the HGH in the body is secreted at night only during deep delta-sleep. HGH improves the action of the immune system and it also promotes healing.
Testosterone:

Testosterone is the major male sex hormone and is primarily secreted by the testes in males. Small amounts of testosterone are found in females and are produced in the ovaries. Testosterone enhances a woman’s libido and nipple and clitoral sensitivity. Testosterone is responsible for male sexual development, erectile function, libido, energy levels, muscle mass, body fat and mood. In males, testosterone assists in keeping the heart healthy and it improves brain function. Low levels significantly worsen the BMI.
**EMF exposure in rats leads to decreased testosterone production**


- **Biological and morphological effects on the reproductive organ of rats after exposure to electromagnetic field.**

- The biological effect of electromagnetic field (EMF) emitted from mobile phones is a current debate and still a controversial issue. Therefore, little is known on the possible adverse effects on reproduction as mobile phone bio-effects are only a very recent concern. The aim of this experimental study was to determine the biological and morphological effects of 900 MHz radiofrequency (RF) EMF on rat testes. METHODS: The study was performed in the Physiology and Histology Research Laboratories of Suleyman Demirel University, Faculty of Medicine, Isparta, Turkey in May 2004. Twenty adult male Sprague-Dawley rats weighing 270-320 gm were randomized into 2 groups of 10 animals: Group I (control group) was not exposed to EMF and Group II (EMF group) was exposed to 30 minutes per day, 5 days a week for 4 weeks to 900 MHz EMF. Testes tissues were submitted for histologic and morphologic examination. Testicular biopsy score count and the percentage of interstitial tissue to the entire testicular tissue were registered. Serum testosterone, plasma luteinizing hormone (LH) and follicle stimulating hormone (FSH) levels were assayed biochemically. RESULTS: The weight of testes, testicular biopsy score count and the percentage of interstitial tissue to the entire testicular tissue were not significantly different in EMF group compared to the control group.

- **However, the diameter of the seminiferous tubules and the mean height of the germinal epithelium were significantly decreased in EMF group (p<0.05). There was a significant decrease in serum total testosterone level in EMF group (p<0.05).**

- Therefore, there was an insignificant decrease in plasma LH and FSH levels in EMF group compared to the control group (p>0.05). CONCLUSION: The biological and morphological effects resulting from 900 MHz RF EMF exposure lends no support to suggestions of adverse effect on spermatogenesis, and on germinal epithelium. Therefore, testicular morphologic alterations may possibly be due to hormonal changes.
Estrogens:

Predominately a female sex hormone but small amounts are found in males. The three estrogens in the body are estrone, estradiol, and estriol. The “estrogens” are primarily produced in the ovaries in females. In females, estrogen plays a major role in sexual development and the female ovulation cycle. In women, estrogen decreases the risk of osteoporosis, heart disease and affects neurotransmitters in the brain. Too much estrogen in men, which is caused by low testosterone levels, is not healthy.
Residential magnetic field exposure greatly increases risk of breast cancer in women under the age of 50 who have receptors sensitive to estrogen


Magnetic fields and breast cancer in Swedish adults residing near high-voltage power lines

- Feychting M, Forssen U, Rutqvist LE, Ahlbom A.
- Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden.
- We conducted a case-control study to test the hypothesis that residential magnetic field exposures increase the incidence of breast cancer. The study was based on people who had lived within 300 m of 220- or 400-kV power lines in Sweden at any time between 1960 and 1985. We identified 699 cases of breast cancer in women and 9 cases in men. One matched control per female case and eight per male case were selected at random. Estrogen receptor information was available for a subset of female cases. We assessed magnetic field exposure through calculations of the magnetic fields generated by the power lines before diagnosis. For calculated magnetic field levels \( \geq 0.2 \) microtesla (microT) closest in times before diagnosis, we estimated the relative risk to be 1.0 [95% confidence interval (CI) = 0.7-1.5] for women and 2.1 (95% CI = 0.3-14.1) for men. Women younger than 50 years of age at diagnosis had a relative risk of 1.8 (95% CI = 0.7-4.3). For women with estrogen receptor-positive breast cancer, the relative risk was estimated at 1.6 (95% CI = 0.6-4.1), using the exposure cutoff point \( \geq 0.1 \) microT.

Among estrogen receptor-positive women younger than 50 years at diagnosis, the relative risk increased to 7.4 (95% CI = 1.0-178.1).
Schilddruesenhormone

- **Thyroid Hormones:**
- The thyroid hormones (T2, T3, T4) provide energy and fuel to the body and also regulate the body’s temperature by controlling the body’s metabolism. The thyroid hormones affect brain function, heart health, and they improve the function of the immune system.
Mobile phone radiation decreases TSH, T3, T4 in rats


Effects of 900 MHz electromagnetic field on TSH and thyroid hormones in rats

- Koyu A, Cesur G, Ozguner F, Akdogan M, Mollaoglu H, Ozen S.

In this study, the effects of exposure to a 900 megahertz (MHz) electromagnetic field (EMF) on serum thyroid stimulating hormone (TSH) and triiodothyronine-thyroxin (T3-T4) hormones levels of adult male Sprague-Dawley rats were studied. Thirty rats were used in three independent groups, 10 of which were control (without stress and EMF), 10 of which were exposed to 900 MHz EMF and 10 of which were sham-exposed. The exposures were performed 30 min/day, for 5 days/week for 4 weeks to 900 MHz EMF. Sham-exposed animals were kept under the same environmental conditions as the study groups except with no EMF exposure. The concentration of TSH and T3-T4 hormones in the rat serum was measured by using an immunoradiometric assay (IRMA) method for TSH and a radio-immunoassay (RIA) method for T3 and T4 hormones. TSH values and T3-T4 at the 900 MHz EMF group were significantly lower than the sham-exposed group (p<0.01). There were no statistically significant differences in serum TSH values and T3-T4 hormone concentrations between the control and the sham-exposed group (p>0.05).

- These results indicate that 900 MHz EMF emitted by cellular telephones decrease serum TSH and T3-T4 levels.
Dr. R. Santini untersuchte 1999 den Zusammenhang zwischen dem Auftreten bestimmter Krankheiten und der Nähe zu Mobilfunk-Basisstationen in Frankreich. Anhand einer Befragung von 530 Personen kam er zu dem Ergebnis, dass sich innerhalb einer 300 m Zone folgende Symptome häufen: Müdigkeit, Schlafstörungen, Reizbarkeit, Kopfschmerzen, Gedächtnisverlust, Konzentrationschwierigkeiten etc.

www.funkenflug.de
Salford 2003: Ratbrain, 50 days post 2-hour exposure to cellphone
High frequency device to measure incoming cell phone
The 2-step EMF Solution

1. Switch off all fuses at bedtime.  
   (buy some flashlights or find electrician who can install a “demand switch”)

2. Create a Faraday cage around the bed
   the sleep sanctuary (www.INK-AG.de)

Results: instant improvement of sleep and mood. Other neurological improvements and increased responses to biomedical and neurosensory treatment modalities may take a few months but are dramatic
## COMPREHENSIVE HORMONE PROFILE

**Patient Name:**

Accession #: 900386  Test ID: 142903  Test Code: 4001

<table>
<thead>
<tr>
<th>STEROID</th>
<th>Amount Excreted in μg/24hr</th>
<th>Adult Reference Range</th>
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<tbody>
<tr>
<td>PREGNANEDIOL (progesterone metabolite)</td>
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<td>0 - 1200</td>
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<td>DHEA</td>
<td>13</td>
<td>LOW</td>
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<td>TESTOSTERONE</td>
<td>46.1</td>
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<td>ANDROSTERONE</td>
<td>431</td>
<td>2000 - 5000</td>
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<td>ETIOCHOLANOLONE</td>
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<td>PREGNANETRIOL</td>
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<td>CORTISONE</td>
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<td>CORTISOL</td>
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See our interpretative Guide at: www.meridianvalleylab.com > Tests > Steroids > Steroid Hormone Profiles > 24 Hour Comprehensive Steroid Hormone Profile Interpretation
## RESULTS

<table>
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<tr>
<th>Diagnostic Procedure</th>
<th>Abnormal</th>
<th>Reference Range</th>
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<th>Comments</th>
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## TESTS

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<td>Sex Hormone Binding Globulin</td>
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## COMMENTS

1 - DHEA
- Test performed by Sacred Heart Medical Center, 101 W 8th, Spokane, WA 99204

2 - Progesterone
   - Female Reference Ranges for Progesterone:
     - Full-luteal: 0.29 - 1.40 ng/dL
     - Post-menopausal: 0.20 - 0.70 ng/dL

3 - Testosterone, Fr
- Test performed by Sacred Heart Medical Center, 101 W 8th, Spokane, WA 99204

4 - Estradiol (E2)
- Early Follicular: 0 to 15 pg/mL
- Late Follicular: 125 to 225 pg/mL
- Mid-luteal: 200 pg/mL
- Post-Menopausal: 10 to 15 pg/mL

5 - Estrone (E1)
- Early Follicular: 0 to 150 pg/mL
- Late Follicular: 250 to 450 pg/mL
- Mid-luteal: 400 pg/mL
- Post-Menopausal: 5 to 15 pg/mL

6 - Estradiol (E2)
- Early Follicular: 0 to 100 pg/mL
- Late Follicular: 125 to 225 pg/mL
- Mid-luteal: 200 pg/mL
- Post-Menopausal: 0 to 25 pg/mL
Symptom

Infektionen

Toxische Belastung

Unerlöster seelischer Konflikt
Klinghardt Axiom II

Infektionen  Schwermetalle  USK

Infektionen  Schwermetalle  USK
Infektionen  Schwermetalle  USK
Infektionen

Schwermetalle

USK

Infektionen

Schwermetalle

USK
USK - Infektionen

Schwermetalle

USK

Infektionen

USK
Pathways With Enzymes, Cofactors, Supplements & Blocking Metals/SAH

1. Urea Cycle
2. BH4 Cycle
3. Folate Cycle
4. Methylation Cycle
5. Sulfation Pathway

5 Formyl THF

BH4

5 Methyl THF

NOS

COMT

MAO A

MAO B

Dopamine

Serotonin

THF

5,10 MethyleneTHF

Nucleotides

Dopamine IGF

Methionine

B12

SAMe

Sulfite

Sulfate

SUO

X

Saturday, 11 September 2010
Neuronal Tubulin, the Most Abundant Brain Protein, Is Especially Vulnerable to Mercury
Visualization Of Mercury Emitting From A Dental Amalgam

This is Mercury escaping from an amalgam filling. The filling is 50 years old. The tooth was extracted 15 years ago.

Source:
David Kennedy’s IAOMT tape
www.uninformedconsent.com
<table>
<thead>
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<th>Tissue</th>
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<td>Whole blood</td>
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<td>Kidney</td>
<td>3053.5</td>
</tr>
<tr>
<td>Liver</td>
<td>133.1</td>
</tr>
<tr>
<td>Spleen</td>
<td>15.6</td>
</tr>
<tr>
<td>Frontal cortex</td>
<td>7.2</td>
</tr>
<tr>
<td>Occipital cortex</td>
<td>12.6</td>
</tr>
<tr>
<td>Thalamus</td>
<td>9.9</td>
</tr>
<tr>
<td>Sciatic nerve</td>
<td>0.0</td>
</tr>
<tr>
<td>Spinal cord</td>
<td>0.0</td>
</tr>
<tr>
<td>Cerebrospinal fluid</td>
<td>1.9</td>
</tr>
<tr>
<td>Pituitary</td>
<td>83.6</td>
</tr>
<tr>
<td>Thyroid</td>
<td>4.1</td>
</tr>
<tr>
<td>Adrenal</td>
<td>31.3</td>
</tr>
<tr>
<td>Pancreas</td>
<td>15.6</td>
</tr>
<tr>
<td>Testes</td>
<td>12.7</td>
</tr>
</tbody>
</table>
GENOVATIONS

PHASE II Detoxification: Conjugation of Toxins and Elimination

In Phase II detoxification, large water-soluble molecules are added to toxins, usually at the reactive site formed by Phase I reactions. After Phase II modifications, the body is able to eliminate the transformed toxins in the urine or the feces (through the bile).

**Methylation**

<table>
<thead>
<tr>
<th>Result</th>
<th>Gene</th>
<th>Location</th>
<th>Internet Information</th>
<th>Affects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>genovations.com/gdv158m</td>
<td>Liver/Gut</td>
</tr>
</tbody>
</table>

(NAT SNP) both slow and rapid acetylators are at increased risk for developing lung, colon, bladder, or head & neck cancer.

**Acetylation (N-acetyl transferase)**

**SLOW METABOLIZER POLYMORPHISM**

<table>
<thead>
<tr>
<th>Result</th>
<th>Gene</th>
<th>Location</th>
<th>Internet Information</th>
<th>Affects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>genovations.com/gdr64w</td>
<td>All Cells</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>genovations.com/gdr187q</td>
<td>Liver/Gut</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>genovations.com/gdi114t</td>
<td>Liver/Gut</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>genovations.com/gdr197q</td>
<td>Liver/Gut</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>genovations.com/gdr286e</td>
<td>Liver/Gut</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>genovations.com/gdr64q</td>
<td>Liver/Gut</td>
</tr>
</tbody>
</table>

(FAST METABOLIZER POLYMORPHISM)

<table>
<thead>
<tr>
<th>Result</th>
<th>Gene</th>
<th>Location</th>
<th>Internet Information</th>
<th>Affects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>genovations.com/gdk268r</td>
<td>Liver/Gut</td>
</tr>
</tbody>
</table>

(GST SNP) The GST isoforms (M1, P1, and T1) are more or less prevalent in various tissues; all catalyze the conjugation of electrophilic compounds with glutathione. Defects in GST activity can contribute to fatigue syndromes, and to various cancers throughout the body.

**Glutathione Conjugation (Glutathione S-transferase)**

**NULL GSTM1**

<table>
<thead>
<tr>
<th>Result</th>
<th>Gene</th>
<th>Location</th>
<th>Internet Information</th>
<th>Affects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>genovations.com/gdstm1</td>
<td>Liver/Kidney</td>
</tr>
<tr>
<td>+</td>
<td>GSTP1</td>
<td>I104V</td>
<td>genovations.com/gdstp1</td>
<td>Brain/Skin</td>
</tr>
<tr>
<td>+</td>
<td>GSTP1</td>
<td>A113V</td>
<td>genovations.com/gda113v</td>
<td>Brain/Skin</td>
</tr>
</tbody>
</table>

**Oxidative Protection**

<table>
<thead>
<tr>
<th>Result</th>
<th>Gene</th>
<th>Location</th>
<th>Internet Information</th>
<th>Affects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SOD1</td>
<td>G93A</td>
<td>genovations.com/gdg93a</td>
<td>Cytosol</td>
</tr>
<tr>
<td>+</td>
<td>SOD1</td>
<td>A4V</td>
<td>genovations.com/gdg4v</td>
<td>Cytosol</td>
</tr>
<tr>
<td>+</td>
<td>SOD2</td>
<td>A16V</td>
<td>genovations.com/gda16v</td>
<td>Mitochondria</td>
</tr>
</tbody>
</table>

**Key**

- Neither chromosome carries the genetic variation.
- One chromosome (of two) carries the genetic variation.
- Both chromosomes carry the genetic variation.

**Your Results:** Catechol-O-methyl transferase is the enzyme primarily responsible for breaking down the neurotransmitters dopamine, epinephrine, and norepinephrine.

**Your Results:** N-acetyl Transferase detoxifies many environmental toxins, including tobacco smoke and exhaust fumes. Polymorphisms can result in slower than normal or faster than normal addition of an acetyl group to these toxins. Slow acetylators have a build up of toxins in the system and rapid acetylators add acetyl groups so rapidly that they make mistakes in the process. Both slow and rapid acetylators are at increased risk for toxic overload if they are exposed to environmental toxins. If the toxin exposure is reduced, the risk is reduced.

**Your Results:** Glutathione-S-transferase detoxifies many water-soluble environmental toxins, including many solvents, herbicides, fungicides, lipid peroxides, and heavy metals (e.g., mercury, cadmium, and lead). The various forms of GST work together to eliminate toxins. Decreased glutathione conjugation capacity may increase toxin burden and increase oxidative stress.

**Your Results:** Superoxide Dismutase is an enzyme that protects cells from increased oxidative stress and free radical damage to cell structures like membranes, mitochondria, DNA, and proteins.
Mercury Birth Hair Levels Vs. Amalgam Fillings In Autistic And Control Groups

<table>
<thead>
<tr>
<th>Number of amalgams:</th>
<th>0-3</th>
<th>4-5</th>
<th>6-7</th>
<th>8-9</th>
<th>≥10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control: autistic ratio:</td>
<td>2.64</td>
<td>6.93</td>
<td>6.70</td>
<td>6.32</td>
<td>17.91</td>
</tr>
<tr>
<td>N:</td>
<td>15</td>
<td>22</td>
<td>29</td>
<td>30</td>
<td>43</td>
</tr>
</tbody>
</table>

Data from A. Holmes, M. Blaxill & B. Haley, Int. J. of Toxicology v22, in press, 2003

Saturday, 11 September 2010
Mercury Toxicity and Systemic Elimination Agents

JOSEPH MERCOLA DO1 AND DIETRICH KLINGHARDT MD PhD2

1Optimal Wellness Center, 1443 W. Schaumburg, Schaumburg, IL 60194, USA; 2American Academy of Neural Therapy, 2802 E. Madison#147, Seattle, WA 98112, USA

Abstract
This paper reviews the published evidence supporting amalgam toxicity and clinical techniques that facilitate mercury elimination. A literature review is provided which documents effective mercury elimination strategies to improve mercury toxicity syndromes. Considering the weight of evidence supporting mercury toxicity, it would seem prudent to select alternative dental restoration materials and consider effective mercury elimination strategies if mercury toxicity is present.

Keywords: amalgam and mercury toxicity, DMPS, DMSA, chlorella, cilantro.

MERCURY EXPOSURE AND TOXICITY IS A PREVALENT AND SIGNIFICANT PUBLIC HEALTH THREAT

Chronic mercury exposure from occupational, environmental, dental amalgam and contaminated food exposure is a significant threat to public health [1]. Those with amalgam fillings exceed all occupational exposure allowances of mercury exposure of all European and North American countries. Adults with four or more amalgams run a significant risk from them, while in children as few as two amalgams will contribute to health problems [2]. In most children, the largest source of mercury is that received from immunizations [3–6] or that transferred to them in utero from their mothers [7, 8].

DENTAL AMALGAMS ARE A MAJOR SOURCE OF MERCURY TOXICITY

A single dental amalgam filling with a surface area of only 0.4 cm² is estimated to release as much as 15 μg Hg day⁻¹ primarily through mechanical wear and evaporation [1, 9–11]. The average individual has eight amalgam fillings and could absorb up to 120 μg Hg day⁻¹ from their amalgams. These levels are consistent with reports of 60 μg Hg day⁻¹ collected in human feces [12]. By way of contrast, estimates of the daily absorption of all forms of mercury from fish and seafood is 2.3 μg and from all other foods, air and water is 0.3 μg per day [13]. Currently, Germany, Sweden and Denmark severely restrict the use of amalgams [1].

A “silver” filling, or dental amalgam, is not a true alloy. Amalgams are made up of 50% mercury. The amalgam also consists of 35% silver, 9% tin, 6% copper and a trace of zinc [6]. More than 100 million mercury fillings are placed each year in the US as over 90% of dentists use them for restoring posterior teeth [14]. The mercury vapor from the amalgams is lipid soluble and passes readily through cell membranes and across the blood–brain
Symptoms of Chronic Mercury Toxicity

Immune System

- Repeated infections
  - Viral and fungal
  - Mycobacterial
  - Candida and other yeast infections

- Cancer

- Autoimmune disorders
  - Arthritis
  - Lupus erythematosus (SLE)
  - Multiple sclerosis (MS)
  - Scleroderma
  - Amyolateral sclerosis (ALS)
  - Hypothyroidism
Protective effects of Chlorella vulgaris extract (CVE°) in lead-exposed mice infected with Listeria monocytogenes

Queiroz ML, Rodrigues AP, Bincoletto C, Figueiredo CA, Malacrida S.
Departamento de Farmacologia/Hemocentro, Faculdade de Ciencias Medicas,
Universidade Estadual de Campinas (UNICAMP), C.P. 6111, CEP 13083-970, SP,
Campinas, Brazil. mlsq@fcm.unicamp.br Int Immunopharmacol. 2003 Jun;3(6):889-900

Chlorella vulgaris extract (CVE) was examined for its chelating effects on the myelosuppression induced by lead in Listeria monocytogenes-infected mice. The reduction in the number of bone marrow granulocyte-macrophage progenitors (CFU-GM) observed after the infection was more severe in the groups previously exposed to lead. Extramedullary hematopoiesis, which was drastically increased after the infection, was not altered by the presence of lead. Treatment with CVE, given simultaneously or following lead exposure, restored to control values the myelosuppression observed in infected/lead-exposed mice and produced a significant increase in serum colony-stimulating activity. The benefits of the CVE treatment were also evident in the recovery of thymus weight, since the reduction produced by the infection was further potentiated by lead exposure. The efficacy of CVE was evident when infected and infected/lead-exposed mice were challenged with a lethal dose of L. monocytogenes after a 10-day treatment with 50 mg/kg CVE/day, given simultaneously to the exposure to 1300 ppm lead acetate in drinking water. Survival rates of 30% for the infected group and of 20% for the infected/lead-exposed groups were observed. Evidence that these protective effects of CVE are partly due to its chelating effect was given by the changes observed in blood lead levels. We have observed in the group receiving the CVE/lead simultaneous exposure a dramatic reduction of 66.03% in blood lead levels, when compared to lead-exposed nontreated control. On the other hand, CVE treatment following lead exposure produced a much less effective chelating effect. CVE treatments for 3 or 10 days, starting 24 h following lead exposure, produced a reduction in blood lead levels of 13.5% and 17%, respectively, compared to lead-exposed nontreated controls. The significantly better response observed with the simultaneous CVE/lead administration indicates that the immunomodulation effect of CVE plays an important role in the ability of this algae to reduce blood lead levels. In this regard, additional experiments with gene knockout C57BL/6 mice lacking a functional IFN-gamma gene demonstrated that this cytokine is of paramount importance in the protection afforded by CVE. The antibacterial evaluation measured by the rate of survival demonstrated that, in face of a 100% survival in the control group composed of normal C57BL/6 mice, which are resistant to L. monocytogenes, we observed no protection whatsoever in the IFN-gamma knockout C57BL/6 mice treated with CVE and inoculated with L. monocytogenes.

PMID: 12781705 [PubMed - in process]
‘Chlorella Accelerates Dioxin Excretion in Rats’
Morita, K; Matsueda T; Iida, T; Hasegawa, T:
Journal of Nutrition. 1999 Sept; 129 (9): pps 1731-6

Abstract:
We investigated the effects of Chlorella on the fecal excretion of polychlorinated dibenzo-p-dioxin (PCDD) congeners and polychlorinated dibenzofuran (PCDF) congeners in Wistar rats administered the rice oil that caused Yusho disease, as a substitute for purified dioxin. The rats were fed 4g of a control diet or a 10%Chlorella diet containing 0.2 mL of the rice oil once during the 5-d experimental period. The amounts of PCDD and PCDF congeners excreted in feces from d 1 to 5 in the group fed 10% Chlorella were 0.2-11.3 and 0.3-12.8 times greater (P < 0.05), respectively, than those of the control group. We then investigated the fecal excretion of PCDD and PCDF congeners from d 8 to 35 in rats administered 0.5 mL of the rice oil. Rats consumed the basal diet for 1 wk. After 1 wk, they consumed either the basal diet or the 10% Chlorella diet. The fecal excretions of PCDD and PCDF congeners in the group fed 10% Chlorella were 0.3-3.4 and 0.5-2.5 times greater (most, P <0.05), respectively, than those of the control group. Thus the fecal excretions of PCDD and PCDF congeners were greater in rats fed Chlorella. These findings suggest that the administration of Chlorella may be useful in preventing gastrointestinal absorption and for promoting the excretion of dioxin already absorbed into tissues. Moreover, these findings suggest that Chlorella might be useful in the treatment of humans exposed to dioxin.

This report unfortunately does not reveal which strain of chlorella was used in the study (vulgaris and pyrenoidosa are the two most frequently used for medical research purposes). It is safe to assume, however, that if the vulgaris strain was used, then the researchers might well have obtained higher dioxin excretion rates by testing with the pyrenoidosa strain.

Some of the early research into chlorella’s ability to remove chemical toxins was conducted here in the US at The University of West Virginia School of Medicine.

That study showed that rats fed a diet containing chlordecone (a chlorinated hydrocarbon insecticide), that were subsequently given chlorella pyrenoidosa, effectively decreased the half-life of the circulating toxin from 40 days to 19 days. When the experiment was repeated with chlorella vulgaris, the reduction in half-life was not nearly so pronounced (40 days to almost 33 days.)

This difference between chlorellas has been attributed to the presence of a carotene-like substance known as ‘sporopollenin’ that is unique to the pyrenoidosa strain. (1)

If you and your family fall into any of the ‘high-risk for dioxin exposure’ groups, - including consuming a diet containing foods that potentially harbor high levels of dioxin, two of the smartest things you can do are obviously to reduce your intake of the foods that are on the high-risk list, and ensure that Nature’s Balance Pure Premium Grade Chlorella pyrenoidosa features in your daily supplement regimen!

1. Pore, R.S.: Detoxification of chlordecone poisoned rats with chlorella and chlorella-derived sporopollenin. Drug-Chem-
Toxicol. 1984, 7(1), 57-71