Hormone, Elektrosmog und die Matrix

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www.Klinghardt.org

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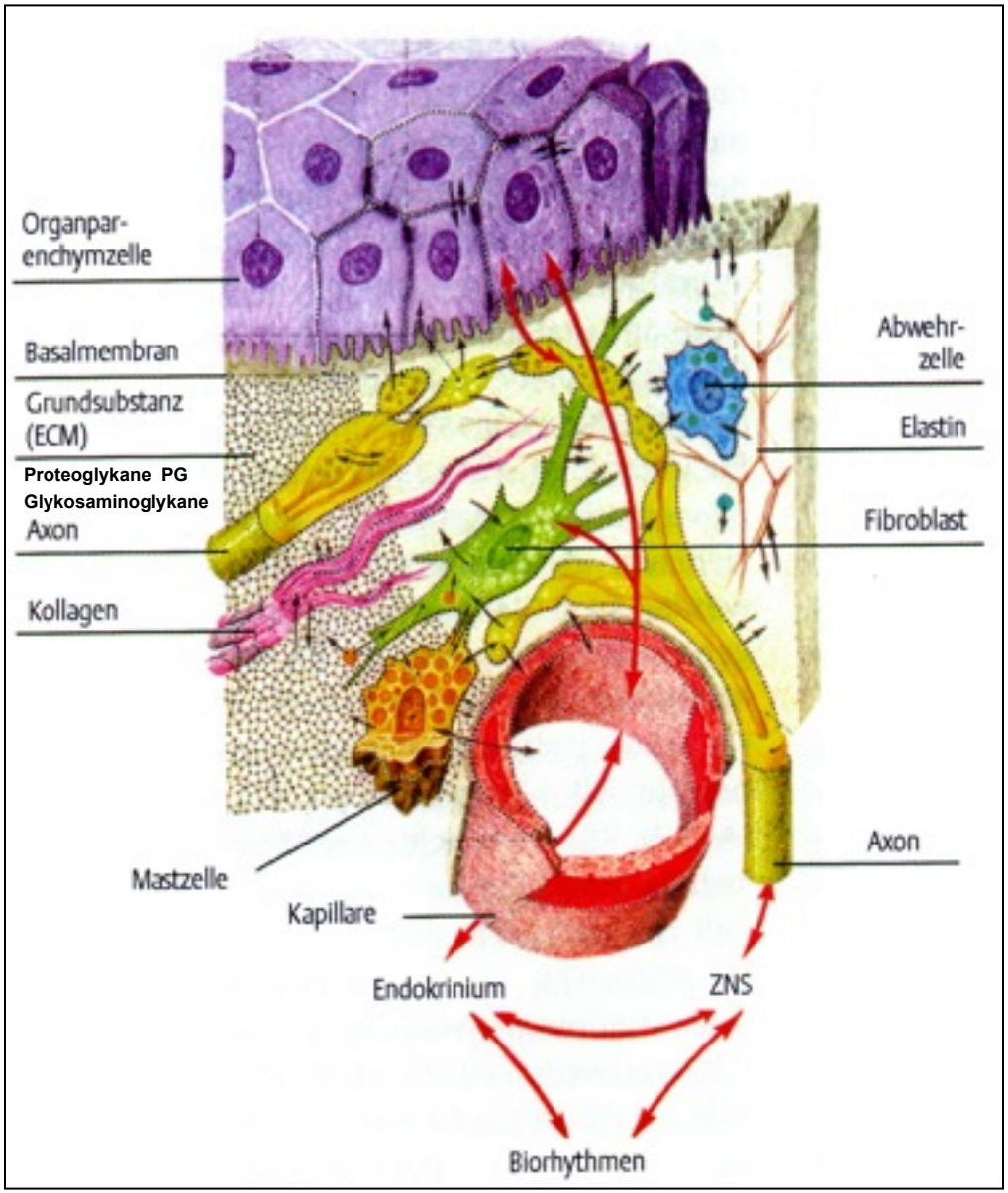
Hormone und Matrix

- Die zentrale Institution der extrazellulaeren Matrix ist der Fibroblast. Er “erzeut” die Matrix Grundsubstanz: GAGs, Heparin, Hyaluronsaeure, Kollagen, usw.
- Fibroblasten haben Hormon Rezeptoren, fuer alle Hormone, insbesondere hGH, Testosteron, Oestrogen (aber auch Rezeptoren fuer Neuropeptide)
- Hormone stimulieren entweder ueber Mechanismen an der Zellwand oder direkte Aufnahme in den Zellkern (T3) die Transskription von Genen


Organpar-
enchymzelle
Basalmembran
Grundsubstanz
(ECM)
Proteoglykane PG
Glykosaminoglykane
Axon
Kollagen
Abwehr-
zelle
Elastin
Fibroblast
Mastzelle
Kapillare
Axon
Endokrinium
ZNS
Biorhythmen

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 during deep sleep. HGH improves the action of the immune system and it also promotes healing.

Testosteron

- **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

*EMF exposure in rats leads to **decreased testosterone production***

Saudi Med J. 2005 Mar;26(3):405-10

- **Biological and morphological effects on the reproductive organ of rats after exposure to electromagnetic field.**
  - Ozguner M,
  - Koyu A,
  - Cesur G,
  - Ural M,
  - Ozguner F,
  - Gokcimen A,
  - Delibas N.
- Department of Histology and Embryology, Faculty of Medicine, Suleyman Demirel University, Isparta, Turkey. mozguner@hotmail.com
- OBJECTIVE: 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.

Oestrogene

- **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*

Epidemiology. 1998 Jul;9(4):392-7

- **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 > or = 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 > or = 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).

Progesteron

- **Progesterone:**
- Progesterone is primarily a female hormone but small amounts are found in males. Progesterone is produced in the corpus luteum, adrenal glands and placenta (during pregnancy). Progesterone assists in regulating the female menstrual cycle. Progesterone affects mood, assists the thyroid gland, builds bones, enhances sexual drive and helps in the utilization of fat for energy.

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*

Toxicol Lett. 2005 Jul 4;157(3):257-62. Epub 2005 Apr 11

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.

– Department of Physiology, Suleyman Demirel University, School of Medicine, 32260 Isparta, Turkey. ahmetkoyu@tnn.net

– In this study, the effects of exposure to a 900 megahertz (MHz) electromagnetic field (EMF) on serum thyroid stimulating hormone (TSH) and triiodothronine-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.**

Electrosmog




Pregnenolon

- **Pregnenolone:**
- Pregnenolone is known as the "grandmother" hormone of the body because it is a precursor to DHEA. Pregnenolone is derived from cholesterol, as are all of the steroidal hormones. Pregnenolone enhances all of our mental functions and it has been used to treat the pain of arthritis.

DHEA

- **DHEA:**
- Referred to as the “mother” hormone of the body, DHEA is produced in the brain and the adrenal glands. DHEA, a steroidal hormone, is the major precursor to the sex hormones. DHEA assists in increasing testosterone, progesterone and estrogen levels. DHEA is important in weight control, muscle gain, optimal brain functioning, heart health and immune system enhancement.

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

Cell phone use greater than 25 minutes per day for 13 days lead to decreased melatonin production

Int J Radiat Biol. 2002 Nov;78(11):1029-36

Melatonin metabolite excretion among cellular telephone users

- Burch JB, Reif JS, Noonan CW, Ichinose T, Bachand AM, Koleber TL, Yost MG.
- Department of Environmental and Radiological Health Sciences, Colorado State University, Fort Collins, CO 80523, USA. james.burch@colostate.edu

- PURPOSE: 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

- **1:** Mol Cell Biochem. 2005 Aug;276(1-2):31-7.

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

- Ozguner F, Oktem F, Armagan A, Yilmaz R, Koyu A, Demirel R, Vural H, Uz E.

– Department of Physiology, School of Medicine, Suleyman Demirel University, P. K. 13 32100 Isparta, Turkey. drmfehmi@yahoo.com

– **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).**

Melatonin and caffeic acid phenyl ester reduce retinal oxidative stress after long-term expsure 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.

- Department of Physiology, School of Medicine, Suleyman Demirel University, P. K. 13, 32100 Isparta, Turkey. drmfehmi@yahoo.com

- 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.

Mobile phone radiation decreases pre-bedtime melatonin level

- **1:** Int J Radiat Biol. 2006 Feb;82(2):69-76
  - **Does evening exposure to mobile phone radiation affect subsequent melatonin production?**
    - Wood AW,
    - Loughran SP,
    - Stough C.
  - Brain Sciences Institute, Swinburne University of Technology, Hawthorn, Victoria, Australia. awood@swin.edu.au
  - 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.

Serum melatonin in rats decreased by electric field exposure

Bioelectromagnetics. 1994;15(5):427-37

- **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-methyl transferase (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

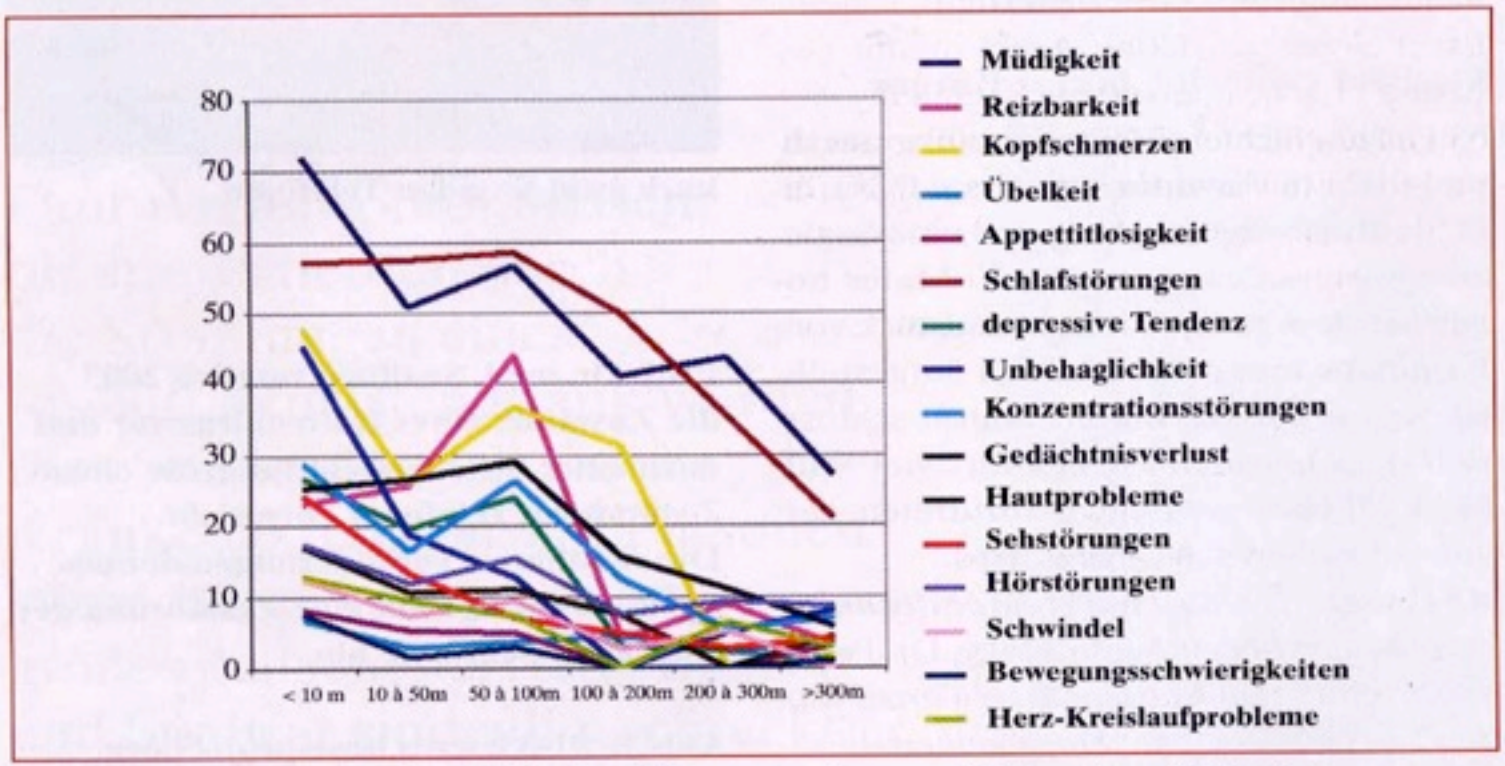
Neurosci Lett. 1999 May 14;266(3):209-12

- **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.

Thymus Peptide

- **Thymic Hormones:**
- The thymus gland produces the thymic hormones. The thymic hormones are known as the “T-killer cells” in the body, which help fight disease. As we age, the thymus gland shrinks significantly, thereby producing less “T-killer cells”. The thymic hormones improve the functioning of the endocrine, central nervous, and cardiovascular systems.

Electrosmog



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

Sleep Sanctuary

High frequency device to measure incoming cell phone

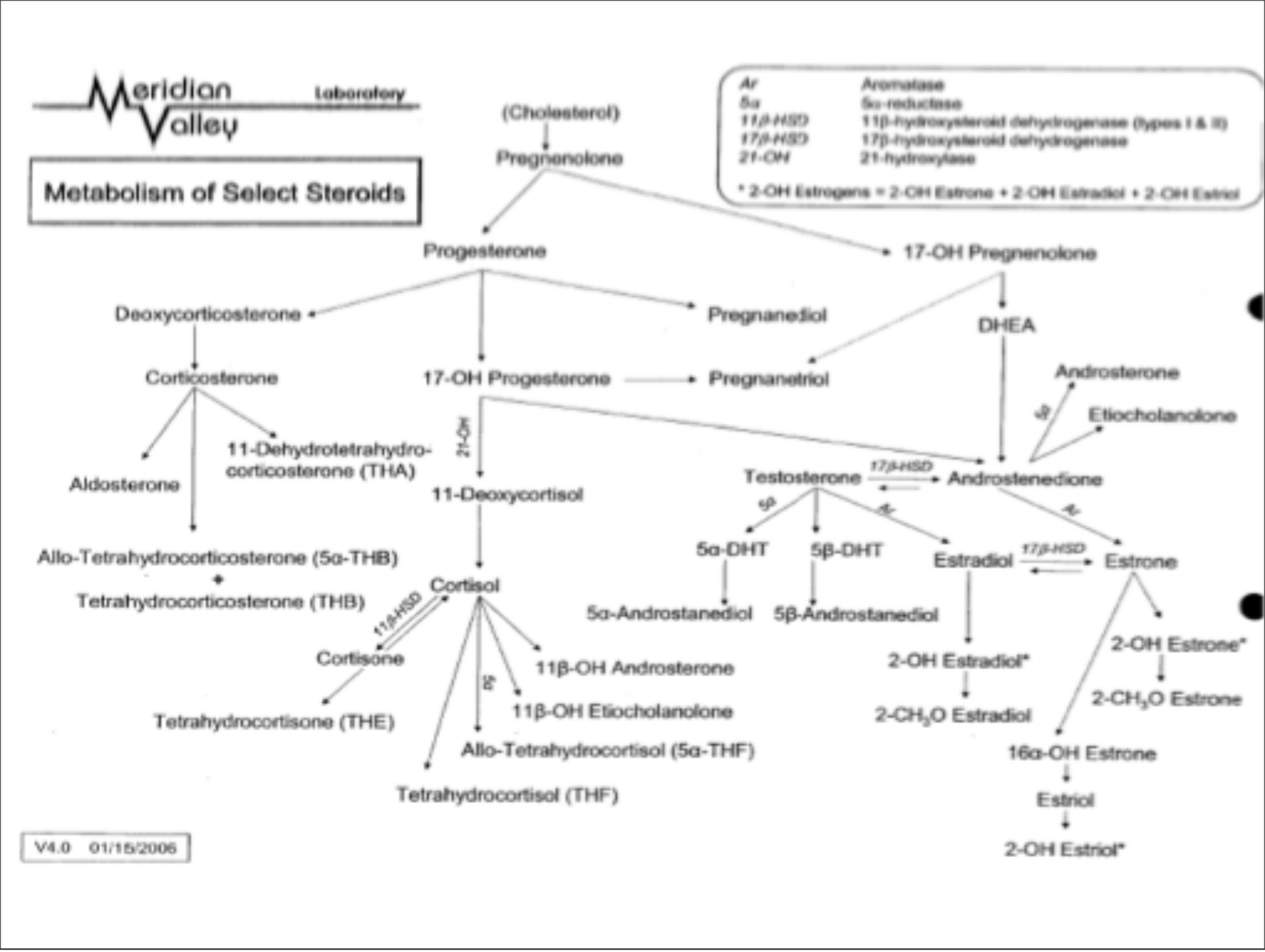
- sul

The 2-step EMF Solution for the autistic child

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
   (Best: the sleep sanctuary from www.BioToolsfor Wellness.com)

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


Meridian Valley Laboratory
Metabolism of Select Steroids
Ar Aromatase
5α 5α-reductase
11β-HSD 11β-hydroxysteroid dehydrogenase (types I & II)
17β-HSD 17β-hydroxysteroid dehydrogenase
21-OH 21-hydroxylase
* 2-OH Estrogens = 2-OH Estrone + 2-OH Estradiol + 2-OH Estriol
(Cholesterol)
Pregnenolone
Progesterone
17-OH Pregnenolone
Deoxycorticosterone
Pregnanediol
DHEA
Corticosterone
17-OH Progesterone
Pregnanetriol
Androsterone
Etiocholanolone
5β
21-OH
11-Dehydrotetrahydro-corticosterone (THA)
Aldosterone
11-Deoxycortisol
Testosterone
17β-HSD
Androstenedione
5β
Ar
Ar
5α-DHT
5β-DHT
Estradiol
17β-HSD
Estrone
Allo-Tetrahydrocorticosterone (5α-THB)
Tetrahydrocorticosterone (THB)
Cortisol
5α-Androstanediol
5β-Androstanediol
11β-HSD
Cortisone
11β-OH Androsterone
2-OH Estradiol*
2-OH Estrone*
5β
11β-OH Etiocholanolone
2-CH3O Estradiol
2-CH3O Estrone
Tetrahydrocortisone (THE)
Allo-Tetrahydrocortisol (5α-THF)
16α-OH Estrone
Tetrahydrocortisol (THF)
Estriol
2-OH Estriol*
V4.0 01/15/2006

Diagnos-Techs, Inc.

Clinical & Research Laboratory
PO BOX 389662, Tukwila, WA 98138-0662
Tel: (425) 251-0596
CLIA License # 50D0630141

Accession # 03-16208

Received : 04/16/2003
Completed: 04/17/2003
Reported : 04/17/2003

COMPREHENSIVE MEDICAL CENTER

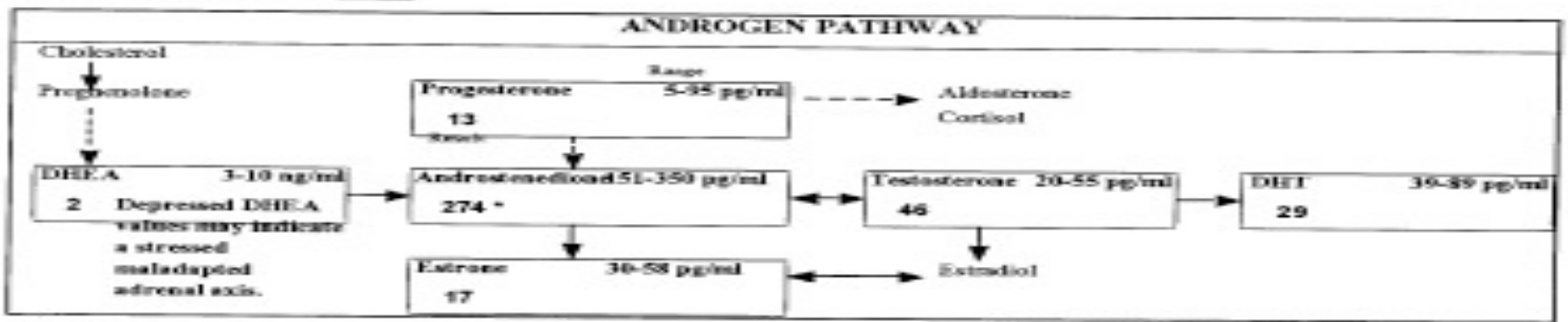
BELLEVUE WA 98004
Tel: (425)688-8818 Fax: (425)453-7015

Age: 66 Gender: Male

Specimen Collected: 04/15/2003

MHP **Male Hormone Panel**

ANDROGEN PATHWAY



* Beginning May 24th 2001, Androstenedione results were reported using an updated reference range due to an improved procedure. Previous results should be interpreted with the previous reference range(s):
Male 20 - 72 pg/ml.
Female 12 - 62 pg/ml.

FAXED 4/15/03 to patient

Hormone	Range	Age
Testosterone (Male)	70 - 135 60 - 110 50 - 80 40 - 70 35 - 65 20 - 55 15 - 45	< 20 yrs 20 - 30 yrs 31 - 40 yrs 41 - 50 yrs 51 - 60 yrs 61 - 70 yrs > 70 yrs
Dihydrotestosterone (Male)	22 - 72 52 - 123 51 - 107 39 - 89	30 - 39 yrs 40 - 49 yrs 50 - 59 yrs > 60 yrs
Androstenedione (Male > 15 years)	100 - 150 151 - 350 351 - 450	Borderline Low Normal Borderline High
Androstenedione (Female > 15 years)	75 - 124 125 - 274 275 - 400	Borderline Low Normal Borderline High
Estrone (Female)	58 - 68 26 - 64 35 - 65	40 - 49 yrs 50 - 59 yrs > 60 yrs

Meridian Valley Laboratory

[redacted]

Comprehensive Hormone Profile

[redacted]

pregnenolone intake can result in increased pregnenolol level

[redacted]

Dietrich K Klinghardt
Comprehensive Medical Center
12000 11(9th Ave NE Ste A-100
Bellevue, WA 98004
Fax: (425) 453-7015
Phone: 4255688888[illegible]

	Amount Excreted in 24hrs	Adult Reference Range
CREATININE	1.2 gm/24hr	0.5-2.0 gm/24hr
TOTAL VOLUME	1700 mL	

STEROID	Amount Excreted in ug/24hr		Male ug/24hr
ESTRONE	2.1	LOW	3 – 8
ESTRADIOL	0.7		0 – 23
ESTRIOL	7.7		2.0 – 28
TOTAL ESTROGENS	10.5		5 – 57

COMPREHENSIVE HORMONE PROFILE

Patient Name:
Accession #: 900185 *Test ID:* 142903 *Test Code:* 4001

STEROID	Amount Excreted in µg/24hr		Adult Reference Range Male µg/24hr
PREGNANEDIOL (progesterone metabolite)	735		0 - 1900
DHEA	13	LOW	100 - 2000
TESTOSTERONE	46.1		20.0 - 200.0
ANDROSTERONE	431	LOW	2000 - 5000
ETIOCHOLANOLONE	541	LOW	1400 - 5000
PREGNANETRIOL	499		200 - 1500
CORTISONE	104		31-209
CORTISOL	76		30-170
TETRAHYDROCORTISONE	1439	LOW	2100-7400
ALLO-TETRAHYDROCORTISOL	415	LOW	700-3800
TETRAHYDROCORTISOL	708	LOW	1200-4500
ALDOSTERONE	15		Normal Diet: 6-25 Low Salt: 17-44 High Salt: 0-6
ALLO-TETRAHYDROCORTICOSTERONE	107	LOW	130-600
TETRAHYDROCORTICOSTERONE	60		30-240

See our Interpretative Guide at: www.meridianvalleylab.com > Tests > Steroids > Steroid Hormone Profiles > 24 Hour Comprehensive Steroid Hormone Profile Interpretation

Patient Name:

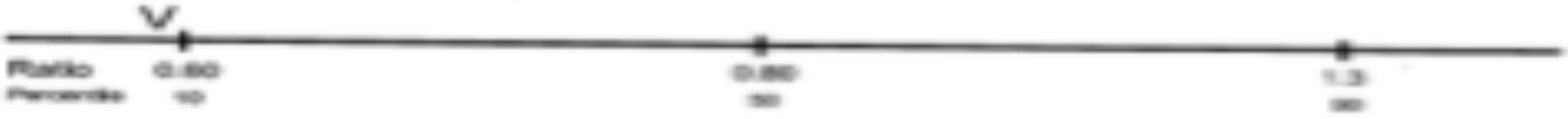
Specimen #: [illegible] Test ID: 142665 Test Code: 4001

ENZYME ACTIVITY PHENOTYPE ASSESSMENT

5-α -REDUCTASE

Androsterone/Etiocholanolone Ratio: 0.80

Allo-tetrahydrocortisol/tetrahydrocortisol Ratio: 0.59



Elevated 5α-reductase activity is associated with polycystic ovary syndrome and hirsutism in women, benign prostate hypertrophy and premature baldness in men, and obesity and insulin resistance in both genders. Low 5α-reductase activity may result in reduced conversion of testosterone to DHT and undervirilization in males.

11-β-hydroxysteroid dehydrogenase I &II

Cortisol/Cortisone Ratio: 0.73

Tetrahydrocortisol+allo-tetrahydrocortisol/Tetrahydrocortisone Ratio: 0.78

Low ratios are associated with obesity and insulin resistance

Elevated ratios are associated with low-renin hypertension, high dose licorice, and cortisol administration.

COMPREHENSIVE MEDICAL
11650 98TH AVE NE
KIRKLAND, WA 98034

at EVERGREEN

	PATIENT ID	SEX	AGE	PT PHONE NO	DOB
	10009758 / 0008	F	43		
PHYSICIAN	COLLECT DATE & TIME	PT LAB NO		REQUISITION NO	STATUS/ABNORMALITY
KLINGHARDT, DIETRICH ND	8/5/08 21:58	E1134		808119992	Final

RESULTS

Diagnostic Procedure		Abnormal	Reference Range	Units	Comments	Site Code	Status
DHEAS	19	L	27-199	ug/dL	1	16462	F
Progesterone	.80			ng/mL	2	16462	F
Testosterone Free							
Testosterone, Fr	<0.2	L	0.2-2.0	pg/mL	3	16462	F
Estrogens Fractionated							
Estrone Lvl	20			pg/mL	4	97	F
Estrogen Tot	52			pg/mL	5	16462	F
Estradiol (E2)	32			pg/mL	6	16462	F
DHEA	1.7	L	1.9-7.8	ng/mL	7	13	F
Misc Send-PAML							
PAML Result	Sex Hormone Binding Globulin					16462	F

TEST RESULT/FLAG REFERENCE RANGE
Sex Hormone Binding Globulin
13 nmol/L 30-135
Test Performed by ARUP, 500 Chipeta Way, Salt Lake City, UT 84108

PAML Test	SHBG					16462	F

COMMENTS

1 - DHEAS
Test Performed by Sacred Heart Medical Center, 101 W 8th, Spokane, WA 99204
2 - Progesterone
Female Reference Range for Progesterone:

Follicular	<0.89 - 1.40 ng/mL
Luteal	3.34 - 25.56 ng/mL
Mid-Luteal	8.46 - 28.03 ng/mL
Post-Menopausal	< 0.20 - 0.73 ng/mL
Pregnancy	
1st trimester	11.22 - 90.00 ng/mL
2nd trimester	25.55 - 89.40 ng/mL
3rd trimester	49.40 - 422.50 ng/mL

Women using oral contraceptives have suppressed progesterone levels. Minimum detectable concentration is 0.20 ng/mL.
3 - Testosterone, Fr
Test Performed by Sacred Heart Medical Center, 101 W 8th, Spokane, WA 99204
4 - Estrone Lvl

Early Follicular	0 to 150 pg/mL
Late Follicular	109 to 250 pg/mL
Luteal	0 to 200 pg/mL
Post Menopausal	15 to 103 pg/mL

5 - Estrogen Tot

Early Follicular	0 to 310 pg/mL
Late Follicular	134 to 600 pg/mL
Luteal	27 to 446 pg/mL
Post Menopausal	15 to 136 pg/mL

Test Performed by Sacred Heart Medical Center, 101 W 8th, Spokane, WA 99204
6 - Estradiol (E2)

Early Follicular	0 to 140 pg/mL
Late Follicular	34 to 400 pg/mL
Luteal	27 to 246 pg/mL
Post menopausal	0 to 35 pg/mL

7 - DHEA

COMPREHENSIVE MEDICAL
11555 98TH AVE NE
KIRKLAND, WA 98034

[redacted] at EVERGREEN

PATIENT NAME	PATIENT ID	SEX	AGE	PT. PHONE NO.	
	10697758 / 0660	F	40		
PHYSICIAN	COLLECT DATE & TIME	PT. LOC NO.			
KLINGHARDT, DIETRICH MD	8/4/08 15:39	E1134			Final

RESULTS

Diagnostic Procedure		Abnormal	Reference Range	Units	Comments	Site Code	Status
Fax Results							
Fax Results	Yes					1542	F
Phone#	4258238817					1542	F
TSH	0.92		0.40-5.00	uIU/mL	1	1542	F
T4 Free	0.8		0.8-1.8	ng/dL		1542	F
T3 Free	3.2		2.3-4.2	pg/mL	2	79	F
FSH	101.0			mIU/mL	3	1542	F
LH	44.7			mIU/mL	4	1542	F
Thyroid Peroxidase Auto A							
Thyroid Peroxidase Ab	<10.0		0.0-35.0	IU/mL	5	79	F
MiscSend-PAML							
PAML Result	Reverse T3					1542	F
TEST	RESULT/FLAG		REFERENCE RANGE				
Reverse T3	193	pg/mL	90-350				

(NOTE)
INTERPRETIVE DATA: Triiodothyronine, Reverse
This test uses a kit developed by the manufacturer and
"for research use, not for clinical use." The performance
characteristics of this test were validated by ARUP
Laboratories, Inc. The U.S. Food and Drug Administration
(FDA) has not approved this test. The results are not
intended to be used as the sole means for clinical
diagnosis or patient management decisions. ARUP is
authorized under Clinical Laboratory Improvement
Amendments (CLIA) and by all states to perform high-
complexity testing.
Test Performed by ARUP, 500 Chipeta Way, Salt Lake City, UT 84108

PAML Test	REVT3					1542	F

Other Orders in Requisition	Current Status	Order Priority
FSH	Order was canceled	Routine
LH	Order was canceled	Routine

COMMENTS

2 - TSH
Testing performed by a highly sensitive, solid-phase
methodology. Only highly sensitive TSH assays have sufficient
clinical sensitivity to detect the minor degrees of TSH excess or
deficiency associated with early, subclinical phases of hyper-or
hypothyroidism.
2 - T3 Free
Test Performed by Pathology Associates Medical Lab, 110 W. Cliff Dr., Spokane, WA 99204
3 - FSH
Reference Range for FSH:
Females:
Follicular 4.0 - 13.0 mIU/mL
Midcycle 5.0 - 22.0 mIU/mL
Luteal 2.0 - 13.0 mIU/mL
Post-Menopausal 20.0 -138.0 mIU/mL

Males 1.0 - 9.0 mIU/mL
4 - LH
Reference Range for LH:
Females:

(509) 926-2400 • (800) 541-7994 • FAX (509) 926-0857
CLIENT SERVICES (509) 927-6299 • FAX (509) 924-1127

THOMAS J. ALLERDING MD
MEDICAL DIRECTOR

FINAL REPORT

PATIENT	SEX/AGE	DOCTOR	DATE RECEIVED	LABORATORY#
	M 70	DIETRICH ELLENHARDT MD	04/18/06	602369
	DOB:		DATE REPORTED 04/19/06	

REQUEST: [illegible]OL,OL

COMMENT: NONFASTING

COLLECTED 04/18/06 NOT GIVEN

TEST	NORM	ABNORM	UNITS	REFERENCE RANGE	LT = Less Than, GT = Greater Than	REF LAB
ESTRADIOL	30.4		PG/ML			45
REFERENCE RANGE:						
MENSTRUATING FEMALES:						
FOLLICULAR PHASE: 18.9-246.7 PG/ML						
MIDCYCLE: 35.5-570.8 PG/ML						
LUTEAL PHASE: 22.4-256.0 PG/ML						
POST-MENOPAUSAL FEMALES: NOT DETECTABLE - 44.5 PG/ML						
MALES: 11.6-41.2 PG/ML						
TOTAL PSA		6.07 H	ng/mL	0.00-5.60		01
FREE PSA	0.98		ng/mL			01
FREE/TOTAL PSA RATIO	16.1		%			01
Ratios >20% suggest benign.						
Ratios between 10% and 20% show substantial overlap in benign and malignant conditions.						
Ratios <10% suggest carcinoma.						
The ratio is most clinically useful in the total PSA range of 4 to 10 ng/mL.						
This is now an FDA approved procedure.						
For purposes of calculating the free PSA ratio, the total PSA and the free PSA were measured by the same analytical method (Roche Diagnostics). This procedure will ensure the most accurate free PSA ratio. PAML's routine total PSA method is from Bayer Diagnostics and those results may show slight differences from results obtained with the Roche method.						
The free PSA ratio is useful in differentiating between benign prostatic hypertrophy and prostatic carcinoma. Serial monitoring of patients should be done with total PSA measurements performed with the routine Bayer method.						
TESTOSTERONE, FREE	13.4		pg/mL	5-25		17

**** REF LAB ****
01 PAML - Spokane, WA 99204
17 SNMC - Spokane, WA 99204
45 Overlake Hospital Medical Ctr - Bellevue, WA 98004

AT 335 04/18/06 135 1500 BIA

PK und Biologie

3 grosse biologische Systeme bestimmen unsere Gesundheit:

1. das **Genom** (unsere Gene)

Beispiel: Gen fuer den Bauplan der Glutathion S Transferase M1

2. Das **Proteom** (die Gesamtheit der Proteinbestaqndteile der metabolischen Enzyme)

Beispiel: Glutathion S Transferase

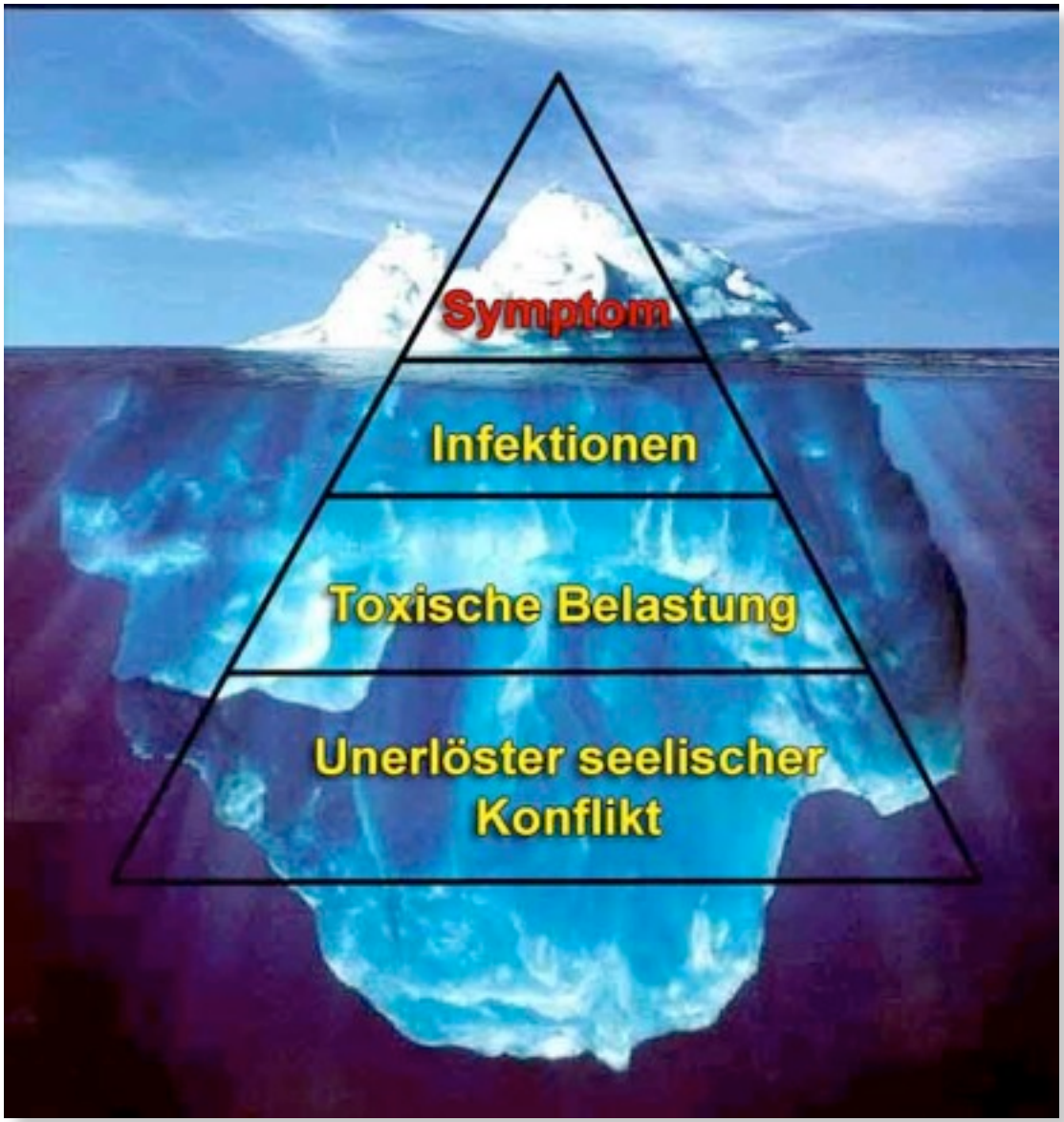
3. Das **Metabolom** (die metabolische Leistung der Enzyme)

Beispiel: der extrazellulaere (=Serum Spiegel) und intrazellulaere Gehalt von reduziertem Glutathion, der die Entgiftungsleistung der Zelle bestimmt

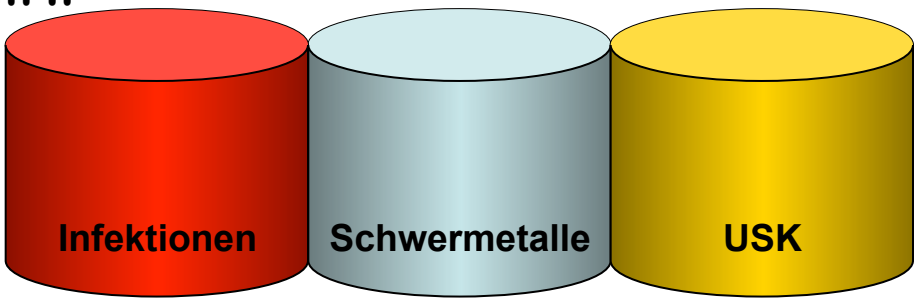
Die Gene und Proteom werden gesteuert durch 2 wesentliche Einfluesse:

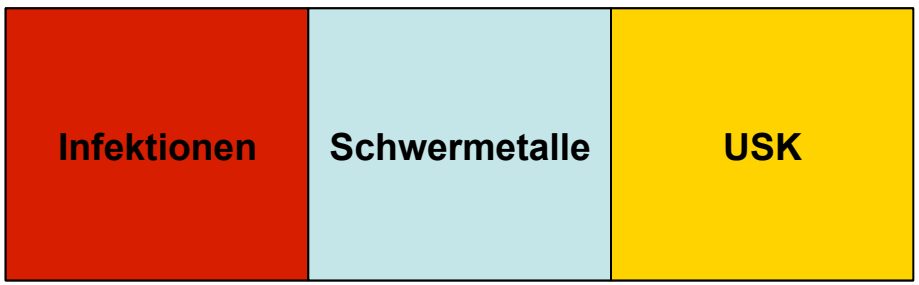
a. die Umwelt (Nahrung, Vitamine, Giftstoffe, usw) und

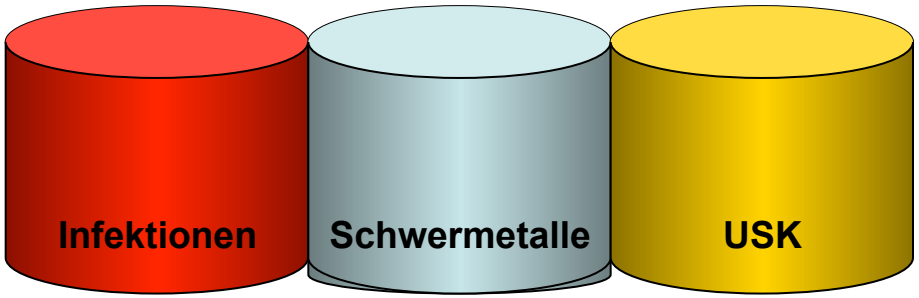
b. **die Psyche: mit PK haben wir Einfluss auf unsere gesamte Biologie, bis tief hinein in die Genetik**

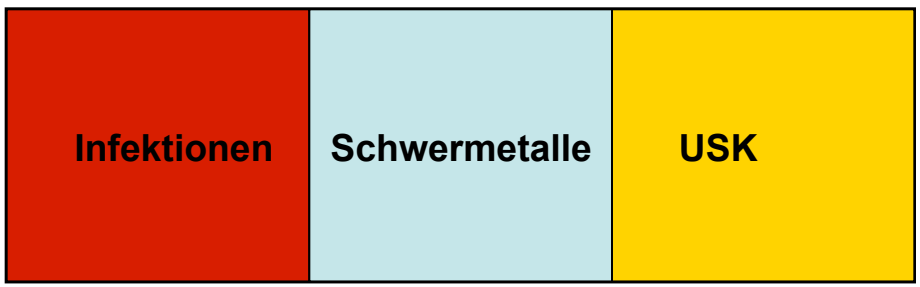

Symptom
Infektionen
Toxische Belastung
Unerlöster seelischer Konflikt

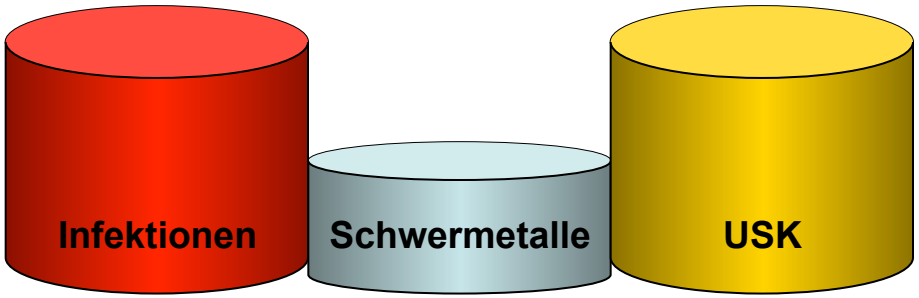
Klinghardt Axiom II

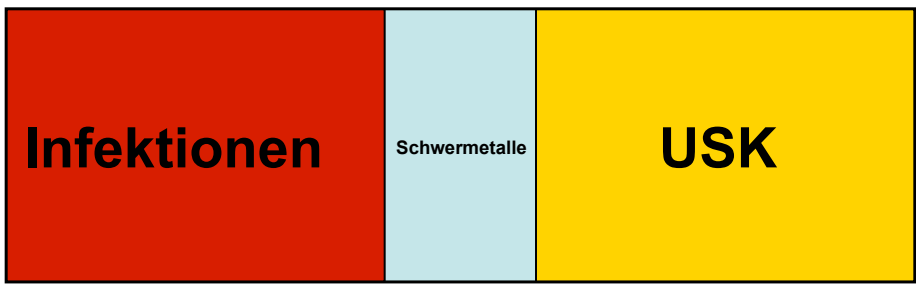


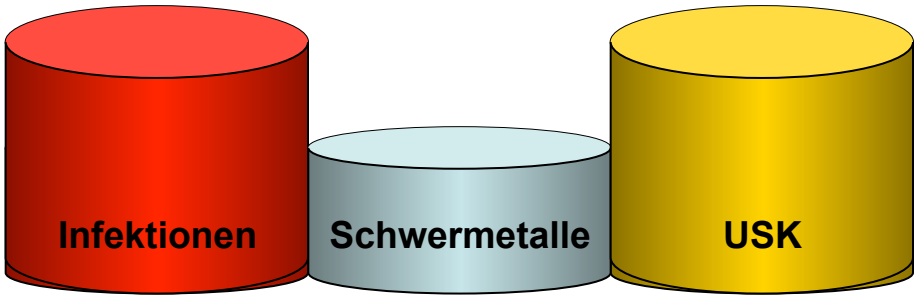


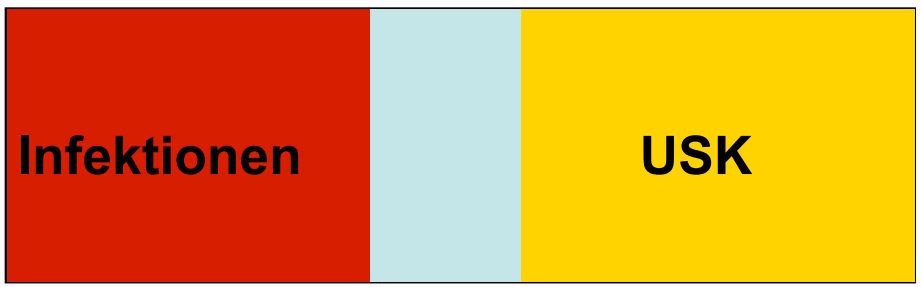

Infektionen
Schwermetalle
USK

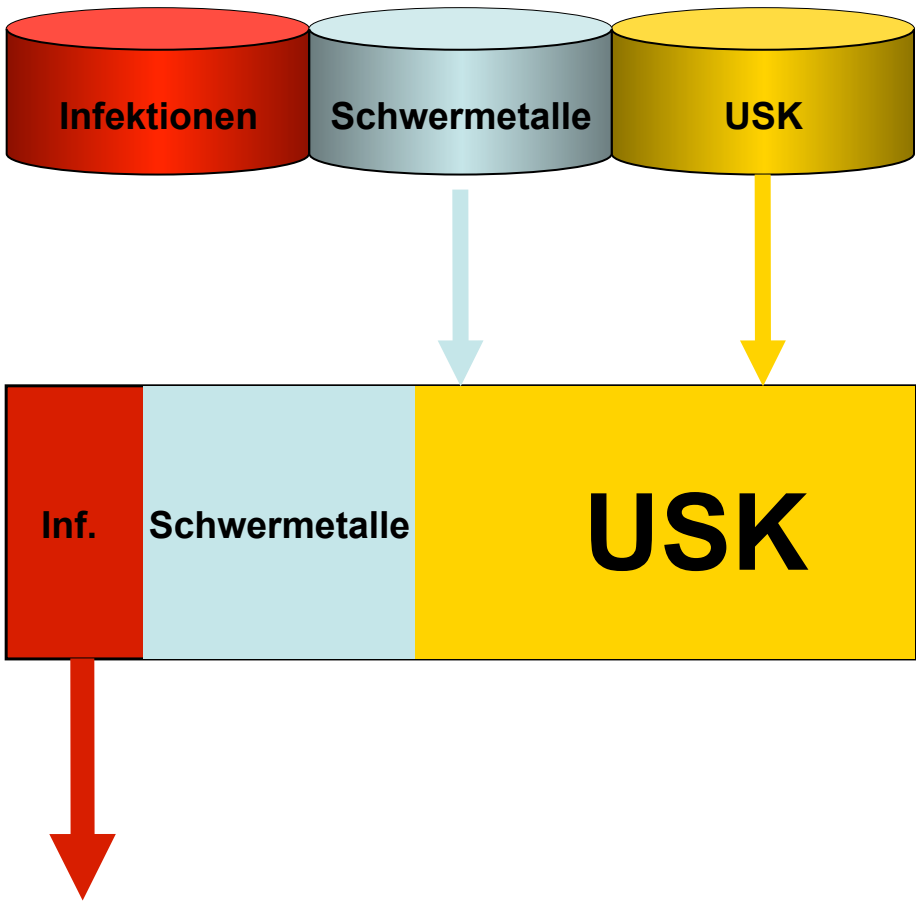

Infektionen
Schwermetalle
USK


Infektionen
Schwermetalle
USK


Infektionen
Schwermetalle
USK

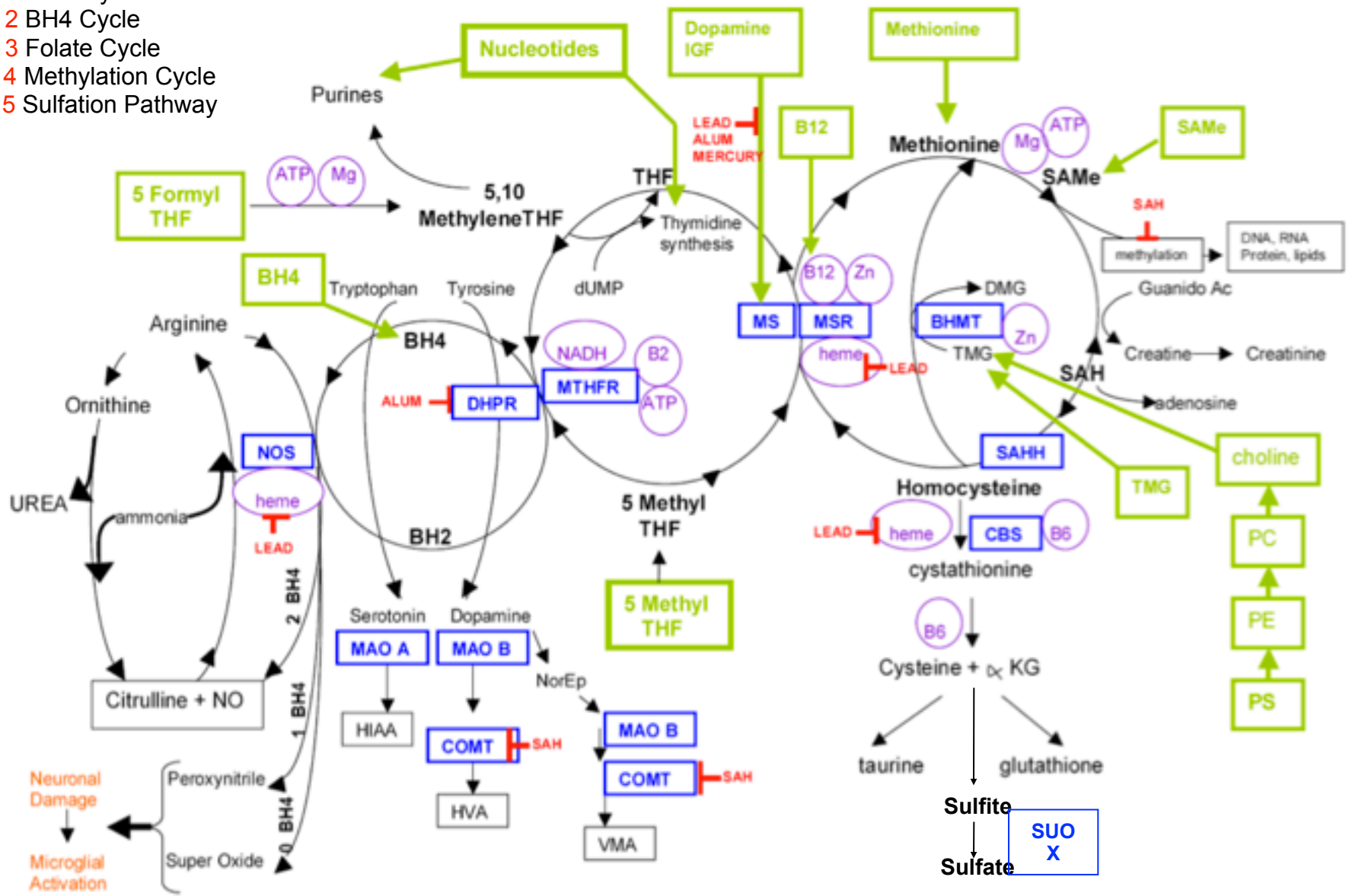

Infektionen
Schwermetalle
USK


Infektionen
USK


Infektionen
Schwermetalle
USK
Inf.
Schwermetalle
USK

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

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



Modified from Amy Yasko

Beilspiel: Pk und das Chronische Muedigkeitssyndrom (CFIDS)

- Haeufigste Ursache: HHV-6(a) – Human Herpes Virus Typ 6a (manchmal auch Borrelien, “Coagulase Negative Staph” oder EBV)
- Vorbedingung: das Enzym Methionin Synthase (MS) wurde durch Toxin Einfluesse umgeschaltet, um die Glutathion Versorgung zu verbessern. Die durch HHV-6 infizierten Gene werden de-methyliert und der Virus wird aktiv
- PK –Behandlung:
  a) Ausleitungsmittel austesten und verordnen
  b) RD Testung der mit dem Methylierungszuklus assoziierten Substanzen: Methyl B12, methylierte Folsaeure, SAM-e usw. und Erstellen der Liste von Substanzen die Stress verursachen und derer, die gebraucht werden
  c) die mit den Stoffen verbundenen USKs loesen. Meist liegt die seelische Ursache bei einem ungeloesten Trauma in der Familiengeschichte der Mutter (Schaedigung der epigenetischen Organisation der Methylierung)

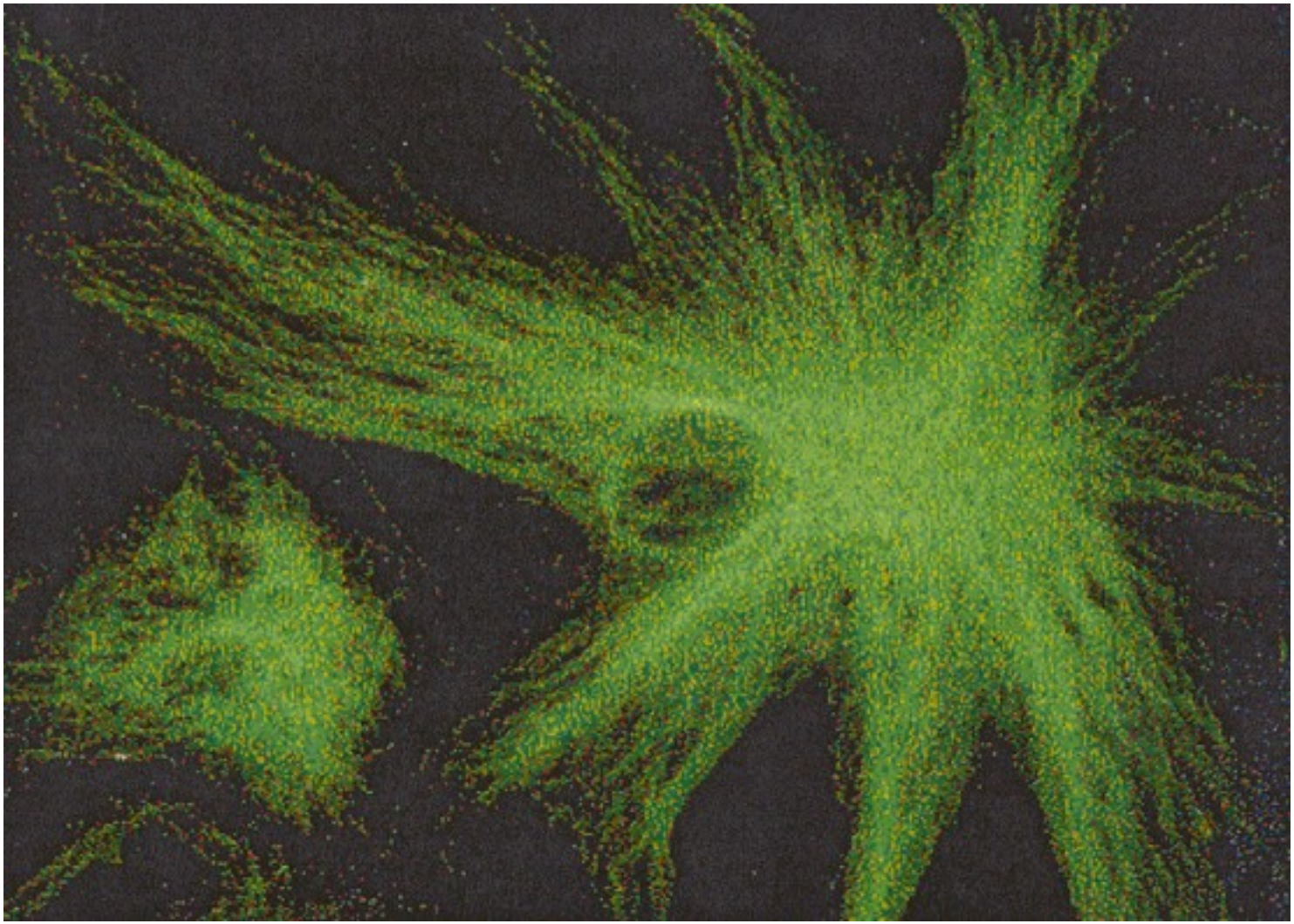
Klinghardt Axiom I

- Wenn eine Erkrankung aus der Sicht der Schulmedizin als “physisch bedingt” interpretiert wird, liegt die Ursache meist im seelischen Bereich und kann erfolgreich mit PK geloest werden

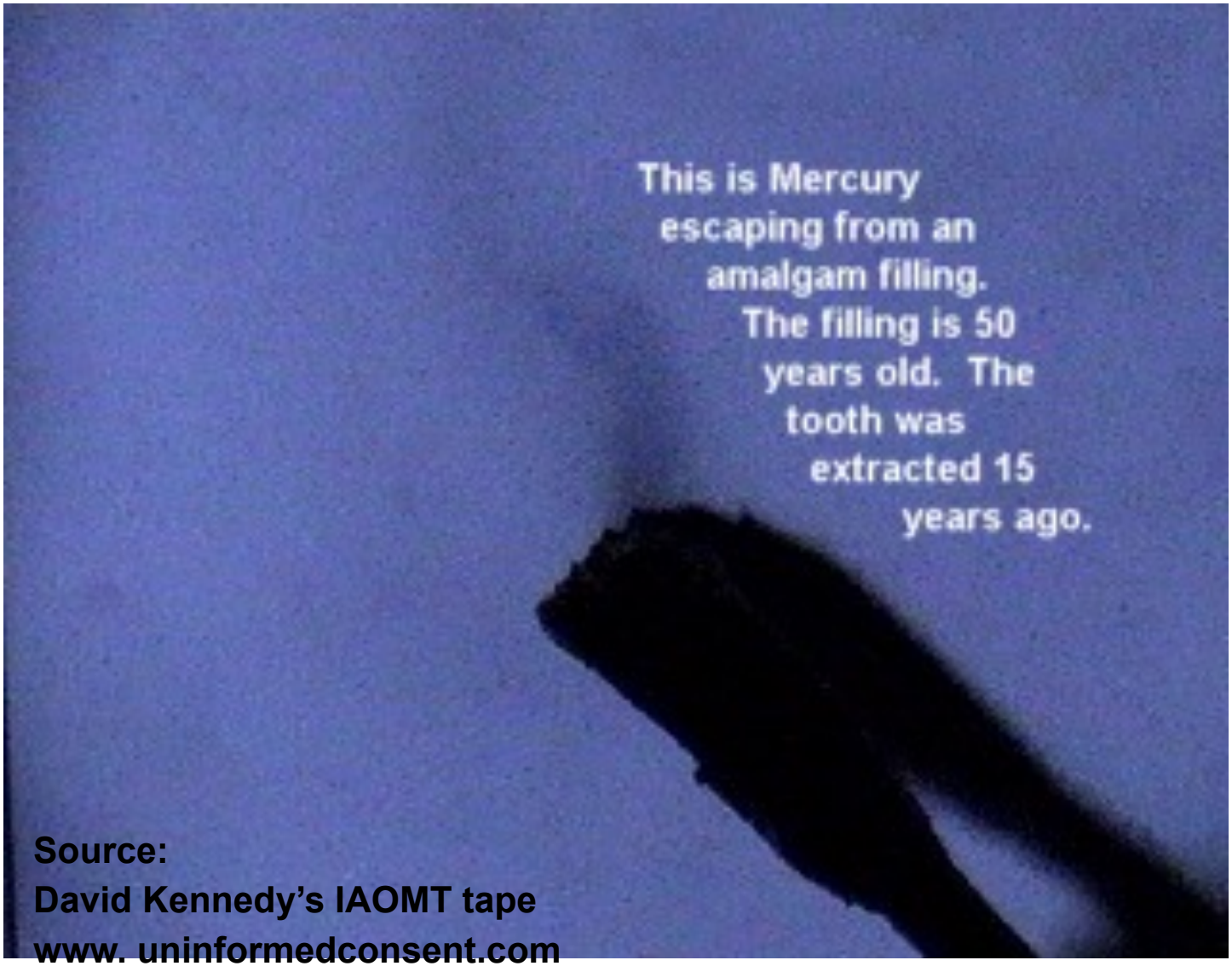
- Wird ein Erkrankung als “psychiatrisch” interpretiert, besteht meist eine Ursache im physischen Bereich (Infektion, Toxinbelastung) und die PK Behandlung hat nur eine sekundaere Bedeutung

- Psychologische Probleme liegen oft in der Mitte zwischen den beiden

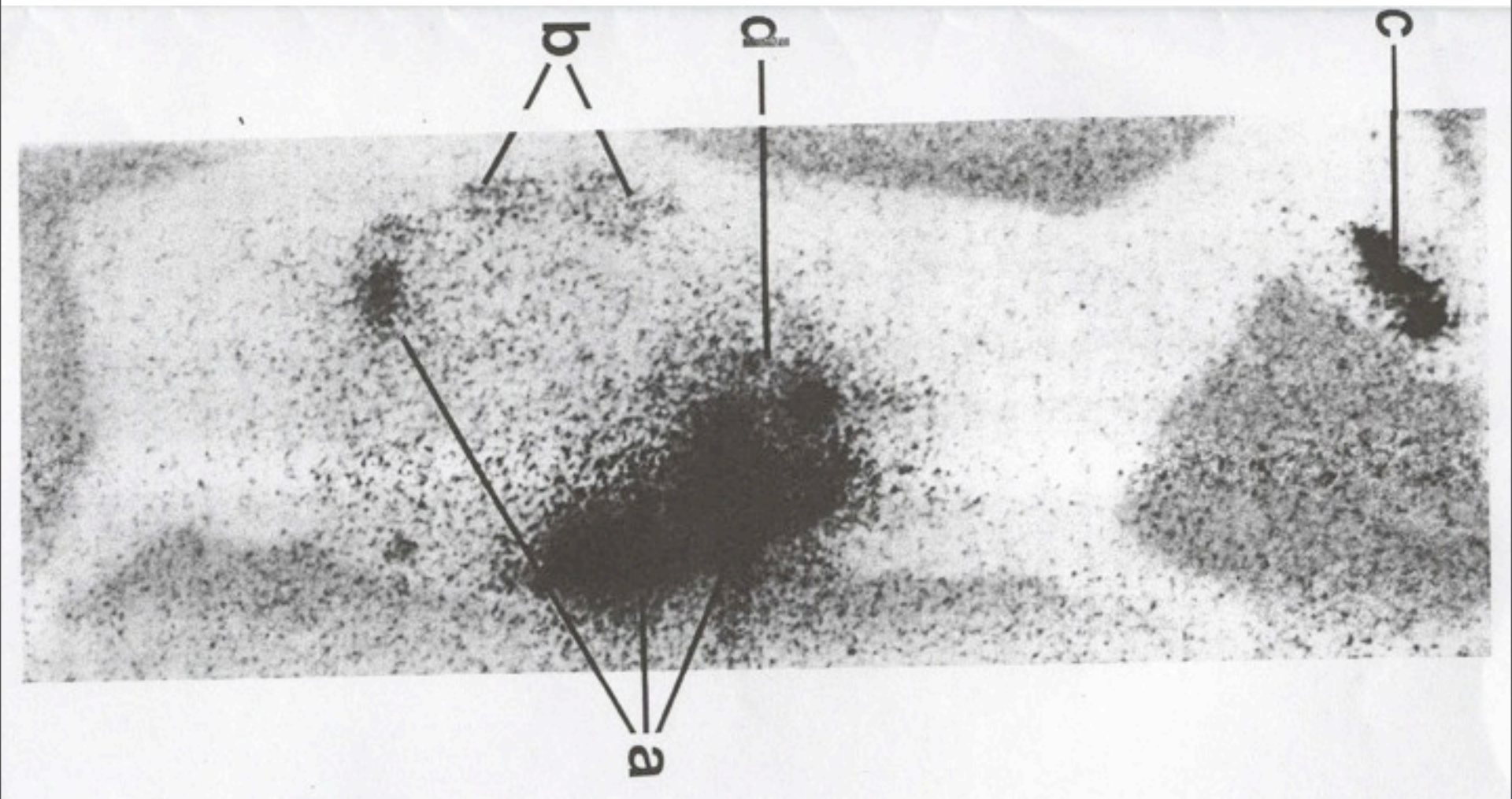
Neuronal Tubulin, the Most Abundant Brain Protein, Is Especially Vulnerable to Mercury



Visualization Of Mercury Emitting From A Dental Amalgam



Source:
David Kennedy's IAOMT tape
www. uninformedconsent.com


b
d
c
a


Prof. Murray

TABLE 1. *Concentration of amalgam Hg in monkey tissues 28 days a[fter] placement of dental amalgam tooth fillings*

Tissue	ng Hg/g
Whole blood	5.8
Urine	17.7
Synovial membrane (knee joint)	31.6
Skeletal muscle (gluteus)	1.9
Fat (mesentery)	0.0
Tooth alveolar bone	7756.1
Oral mucosa	86.6
Gingivae	4190.4
Tongue	253.3
Parotid gland	1.6
Stomach	18.4
Small intestine	68.9
Large intestine	983.1
Colon	482.7
Bile	243.1
Feces	3490.2
Heart (ventricle)	6.6
Lung	15.0
Trachea	12.6
Kidney	3053.5
Liver	133.1
Spleen	15.6
Frontal cortex	7.2
Occipital cortex	12.6
Thalamus	9.9
Sciatic nerve	0.0
Spinal cord	0.0
Cerebrospinal fluid	1.9
Pituitary	83.6
Thyroid	4.1
Adrenal	31.3
Pancreas	15.6
Testes	12.7

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

(COMT SNP) higher risk for depression, bipolar disorder, ADHD and alcoholism.

Result	Gene	SNP Location	Internet Information	Affects
- -	COMT	V158M	www.genovations.com/gdv158m	Liver/Gut

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

Acetylation (N-acetyl transferase)

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

SLOW METABOLIZER POLYMORPHISM

Result	Gene	SNP Location	Internet Information	Affects
- -	NAT1	R64W	www.genovations.com/gdr64w	All Cells
- -	NAT1	R187Q	www.genovations.com/gdr187q	Liver/Gut
- -	NAT2	I114T	www.genovations.com/gdi114t	Liver/Gut
+ -	NAT2	R197Q	www.genovations.com/gdr197q	Liver/Gut
- -	NAT2	G286E	www.genovations.com/gdg286e	Liver/Gut
- -	NAT2	R64Q	www.genovations.com/gdr64q	Liver/Gut

FAST METABOLIZER POLYMORPHISM

- -	NAT2	K268R	www.genovations.com/gdk268r	Liver/Gut

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.

Glutathione Conjugation (Glutathione s-transferase)

(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.

Result	Gene	Location	Internet Information	Affects
NULL	GSTM1	1p13.3	www.genovations.com/gdgstm1	Liver/Kidney
+ -	GSTP1	I104V	www.genovations.com/gdgstp1	Brain/Skin
- -	GSTP1	A113V	www.genovations.com/gda113v	Brain/Skin

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 toxic burden and increase oxidative stress.

Oxidative Protection

(SOD SNP) SOD1 is present in the cytosol; SOD2 is present in the mitochondria. Changes in the SOD enzyme are associated with changes in risk for neurodegenerative disorders like ALS.

Result	Gene	SNP Location	Internet Information	Affects
- -	SOD1	G93A	www.genovations.com/gdg93a	Cytosol
- -	SOD1	A4V	www.genovations.com/gda4v	Cytosol
+ -	SOD2	A16V	www.genovations.com/gda16v	Mitochondria

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.

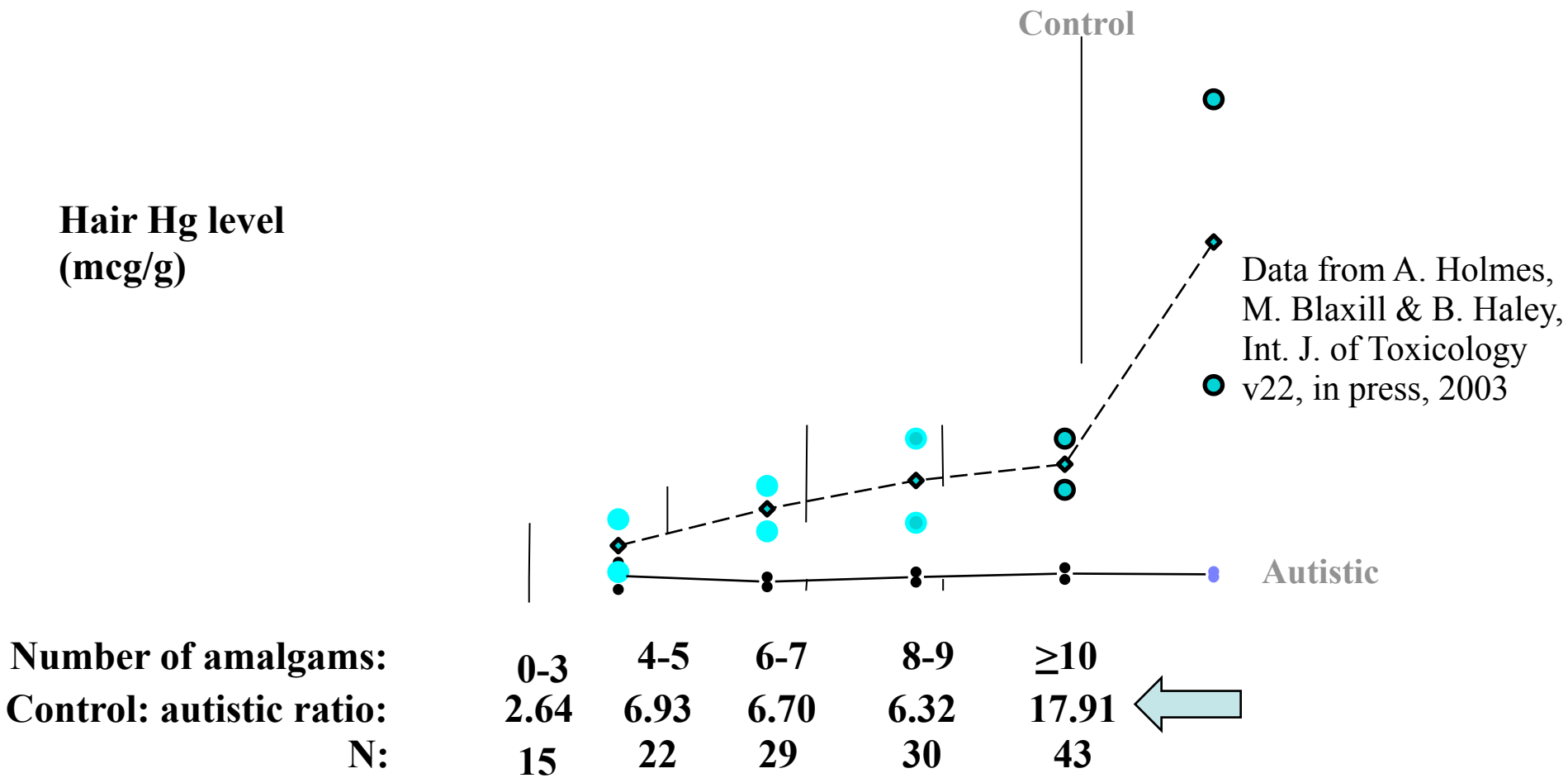
Key

- -	Neither chromosome carries the genetic variation.	Homozygous negative or wild type
+ -	One chromosome (of two) carries the genetic variation.	Heterozygous positive
++	Both chromosomes carry the genetic variation.	Homozygous positive

NR / NULL / IND See commentary

(You inherit one chromosome from each parent)

Mercury Birth Hair Levels Vs. Amalgam Fillings In Autistic And Control Groups



Mol Psychiatry 2004 Sep.;9(9): 833-45
Neurotoxic Effects of Postnatal thimerosal are mouse strain dependent,
Horning M, Chian D, Lipkin WI., Jerome L. and Dawn Greene Infectious Disease Laboratory, Dep. Of Epidemiology, Mailman School of Public Health, Columbia University, New York

- Autoimmune propensity influences outcomes in Mice following thimerosal challenges that mimic routine childhood immunizations
- Mice show growth delay
- Reduced locomotion
- Exaggerated response to novelty
- Densely packed hippocampal neurons with altered glutamate receptors and transporters

Other resent findings:

- After the Am. College of Pediatrics recommended a vaccine schedule in 1989 considered by many insane, a sharp raise in new autism cases resulted across the US, not in other western countries that did not follow the US lead. After the college recommended to reduce the amount of thimerosal in the vaccines in 1999, a sharp drop in new autism cases was observed
- There is no autism in the Amish population. There are no vaccinations in the Amish. The only rare cases of autism in the Amish were found in members of the few families that did vaccinate

Urine Toxic Elements Post DMPS Challenge

C.N.: 35 year old male Dx: CFIDS, FMS

Date	mcg Hg/24 hrs	ppb (post DMPS 3 mg/kg i.v push)
4/23/93	27.8	27.8
6/24/93	99.0	99.0
9/21/93	49.4	49.4
12/23/93	2.1	2.1
4/94-8/94	four treatments with neuraltherapy	
8/24/94	**1514.4**	**1954.0**

A.H.: 46 year old Dx: severe depression, multiple neurological symptoms (muscle weakness, numbness, whole body pain)

Date	mcg Hg /24 hrs	mcg Hg/g creatinine (post DMPS)
11/97-4/98	treatment with APN/MFT	
1/24/1998	2100	2700
2/3/1998		**2900**
4/3/1998	1500	930
4/18/1998		370

Journal of Nutritional & Environmental Medicine (2001) 11, 53–62

REVIEW

Mercury Toxicity and Systemic Elimination Agents

JOSEPH MERCOLA DO[1] AND DIETRICH KLINGHARDT MD PhD[2]

[1]Optimal Wellness Center, 1443 W. Schaumburg, Schaumburg, IL 60194, USA;
[2]American 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 cm^{-2} 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

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Symptoms of Chronic Mercury Toxicity Immune System

Amalgam/Mercury Detox as a Treatment for Chronic Viral, Bacterial, and Fungal Illnesses

Editorial Note: The following article is a transcription of a lecture presented by the author at the Annual Meeting of the International and American Academy of Clinical Nutrition, San Diego, CA, September 1996.

On the Amalgam "Controversy"

From a scientific point of view there is no more "controversy" about the ill health effects of the metals contained in and released by the typical dental amalgam fillings. The sheep and monkey studies conducted at the University of Calgary, Canada—under the guidance of Dr. Murray Vimy, DDS—showed that radioactively labeled mercury released from freshly and correctly placed amalgam fillings (in a monkey study)[1] appeared quickly in the kidneys, brain and wall of the intestines. Through its affinity for sulfhydryl-groups, mercury bonds very firmly to structures in the nervous system. Other studies showed that mercury is taken up in the periphery by all nerve endings (i.e., the hypoglossal nerve of the tongue,[2] the autonomic nerves of the lung or intestinal wall and connective tissue) and actively transported inside the axon of the nerves (axonal transport) to the spinal chord and brainstem.[3] On its way from the periphery to the brain, mercury immobilizes the enzyme that is essential for "making" tubulin.[4] Tubulin forms tubular structures within each nerve, along which the nerve-cell transports metabolic waste from the nerve cell into the periphery and along which the nutrients required by the nerve cell are transported from the periphery to the cell. Once mercury has traveled up the axon, the nerve cell is impaired in its ability to detoxify itself and in its ability to nourish itself. The cell becomes toxic and dies—or lives in a state of chronic malnutrition. The mercury that has entered the nerve cell can no longer be excreted in the normal axonal transport routes (some can exit through the Ca^{++} and Na^{+} channels) and begins to exert its more well-known ill-effects on the mitochondria, nucleus and other organelles of the cell. A multitude of illnesses, usually associated with neurological symptoms, results.

Mercury and Chronic Infections

Practitioners have long observed that patients diagnosed with chronic viral illnesses (EBV, CMV, HIV, herpes zoster and genital herpes, CFIDS, etc.) chronic fungal illnesses (Candidiasis and others) and recurrent episodes of bacterial infections (chronic sinusitis, tonsillitis, bronchitis, bladder/prostate infections, HIV related infections) often have dramatic recoveries following an aggressive mercury/amalgam detoxification program.

The fact that the presence of mercury in the tissues represents the immune system has long been known and is supported by the literature.[5,6,7,8] This would explain a general immune enhancing effect of any metal mercury detoxification program. It has also been shown that the presence of amalgam fillings conveys immunity to antibiotics to various bacteria and also impairs the body's own defense system.[9] Mercury is therefore the only substance ever shown that induces antibiotic resistance in bacteria, other than an antibiotic itself. It is known that periodontal disease is caused by bacteria and that the removal of amalgam fillings can often be curative.[10] No studies have tested the mercury hypothesis in other infections, even though the clinical evidence is overwhelming.

In chronic fungal syndromes, the scientific literature gives only circumstantial evidence that mercury fosters those infections. The most valuable clinical pearls I found in a book written for the mining industry: "Biosorption of Heavy Metals."[11] To increase the yield of precious metals in old mines, so-called "biosensors" are sprayed into the mine shaft, washed out with water, and collected on ion exchange membranes. A biomass is a sludge of membranes from usually mono-cellular organisms that have a tendency to accumulate metals in their outer cell wall that they are exposed to.

The list of organisms that have the highest affinity for toxic metals reads like a "who's who" of our typical infectious diseases: fungi of the candida species, streptococci, staphylococci, amoeben, etc., etc. The list is topped by two algae: *Chlorella pyrenoidosa* and *Chlorella vulgaris* (not spirulina or super blue green algae!). The list prompted me to state what in Germany is now refered to as the "Klinghardt Axiom": Most—if not all—chronic infectious diseases are not caused by a failure of the immune system, but are a conscious adaptation of the immune system to an otherwise lethal heavy metal environment. Mercury suffocates the intracellular respiratory mechanism and can cause cell death. So, the immune system makes a deal: it cultivates fungi and bacteria that can bind large amounts of toxic metals. The gain: the cells can breathe. The cost: the system has to provide nutrition for the microorganisms and has to deal with their metabolic products ("toxins"). That does not imply that the tolerated guest cannot grow out of control, as it sometimes clearly does. Therefore, there is still a limited place for antifungal/antibacterial treatment—but only for the acute phase of the disease. A so-called "die-off effect" (the sometimes severe crisis or even lethal reaction a patient can have in the initial stages of aggressive pharmaceutical antifungal or antibacterial treatment) is often nothing else but acute heavy metal toxicity—metals released from the cell walls of dying microorganisms as suggested by my own correlation of clinical syndromes and urinalysis for metals. Colleagues in Germany are working on a study at this time. Preliminary results show a dramatic improvement in clinical and scientific parameters in chronic Candidiasis using the Klinghardt protocol for heavy metal detoxification.

When it comes to chronic viral conditions, as evidence is even more circumstantial. There are several articles in the cell-cell literature showing remarkable

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- 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

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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. Extramedullar 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.

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'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 susbstance 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*