"The toxic metal contaminated terrain: breeding place for pathogens and parasites"

Symptoms of the 2013 Patient

- Daytime episodes of inappropriate physical and **mental fatigue** and **lack of zest**
- **Difficulty sleeping**, waking unrefreshed
- Difficulty with **short term memory**, concentration, word finding, mood swings, decreased assimilation of new information
- strange neurological symptoms: vibration, sounds, tingling, numbness, cranial nerve dysfunction (vagus, vertigo, tinnitus, blurred vision)
- General feeling of **weakness**, with shortness of breath after brief exertion, increased heart rate
- Body **aches** (FMS-like or not), joint **pains, exercise intolerance**
- **Sinus** problems, sore throats, light sensitivity, “floaters”, cough
- Difficulty regulating body temperature, sometimes with night sweats and general **coldness**
- Increased **thirst**, but “water runs straight through”, increased urination, light urine color

The 3 leading symptoms of chronic neurological conditions

1. Mercury toxicity: daytime fatigue, sleep problems (insomnia), brain fog (cognitive impairment)
2. Lead toxicity: fatigue, insomnia, brain fog
3. Mold illness: fatigue, insomnia, brain fog
4. Lyme disease: fatigue, insomnia, brain fog
5. Viral illness: EBV, HHV-6, XMRV: fatigue, insomnia, brain fog
6. Electrosensitivity/Electroallergy: fatigue, insomnia, brain fog
7. Petrochemical/insecticide exposure:

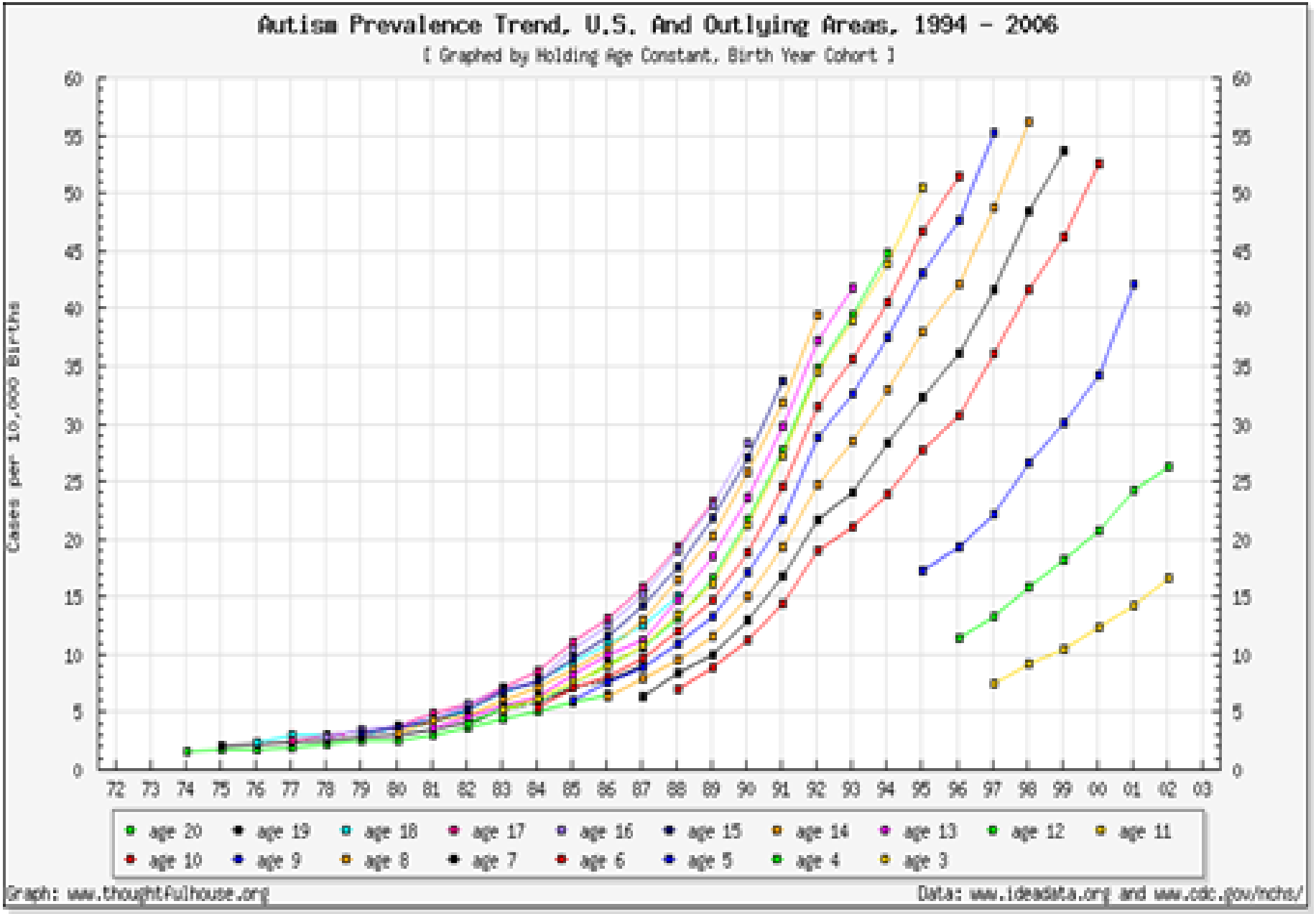
   a) early symptoms : fatigue, insomnia, brain fog.

   b) late symptoms: neuro-degenerative diseases and cancer

If this is true (and it is): How do you get from the symptom to the correct diagnosis and reasonable treatment?

Our Health Span has decreased significantly. Why?

- Cancer rates have been fairly stable over the last years, the rate of neurological illnesses is increasing exponentially. What is going on?
- Have our genes, gene switches or our epigenome suddenly changed?
- Is the increase caused by environmental toxins and influences?
- Electrosmog? Electrosensitivity?
- New emerging CNS infections? (Lyme, HSV-1, Borna virus, etc.)
- Burnout?
- Atmospheric Aerosol Spraying program? (Aluminum, Barium, Fluoride, nanonized plastic, etc.)


Autism Prevalence Trend, U.S. And Outlying Areas, 1994 - 2006
[Graphed by Holding Age Constant, Birth Year Cohort]
Cases per 10,000 Births
0
5
10
15
20
25
30
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72 73 74 75 76 77 78 79 80 81 82 83 84 85 86 87 88 89 90 91 92 93 94 95 96 97 98 99 00 01 02 03
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age 17
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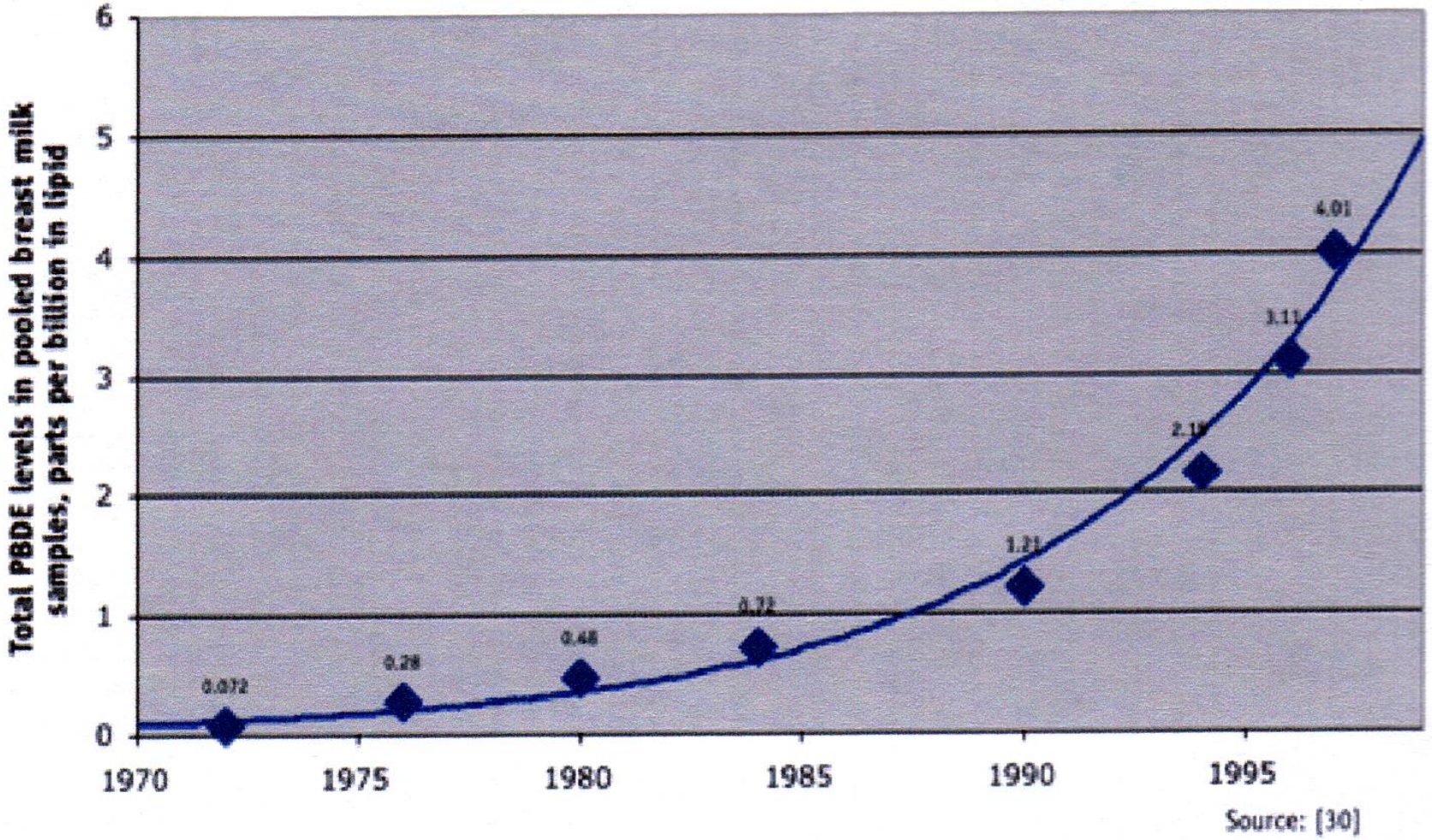
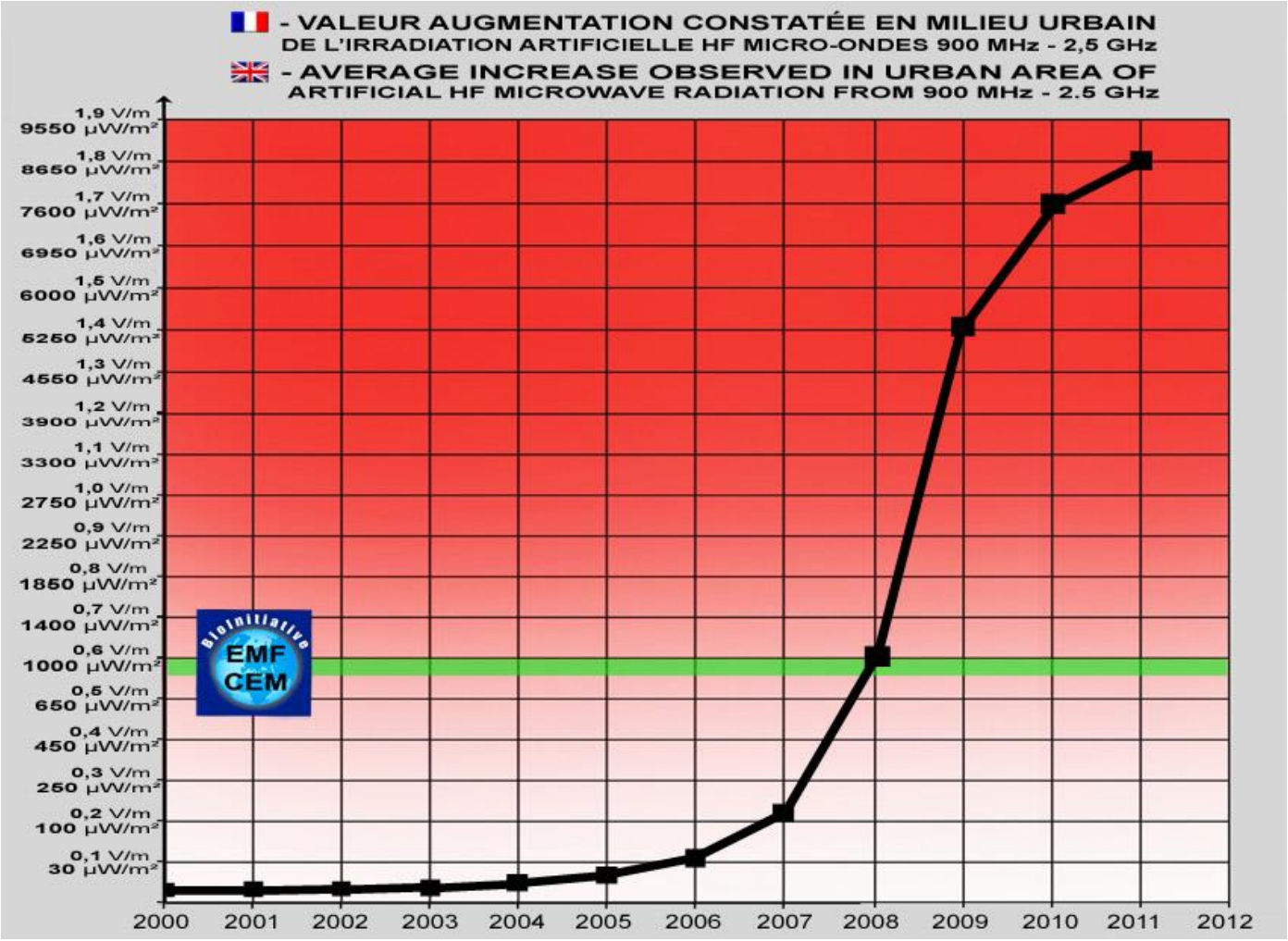
age 6
age 5
age 4
age 3
Graph: www.thoughtfulhouse.org
Data: www.ideadata.org and www.cdc.gov/nchs/

Dramatic increase in levels of fire retardants in Swedish women's bodies, 1972 to 1997



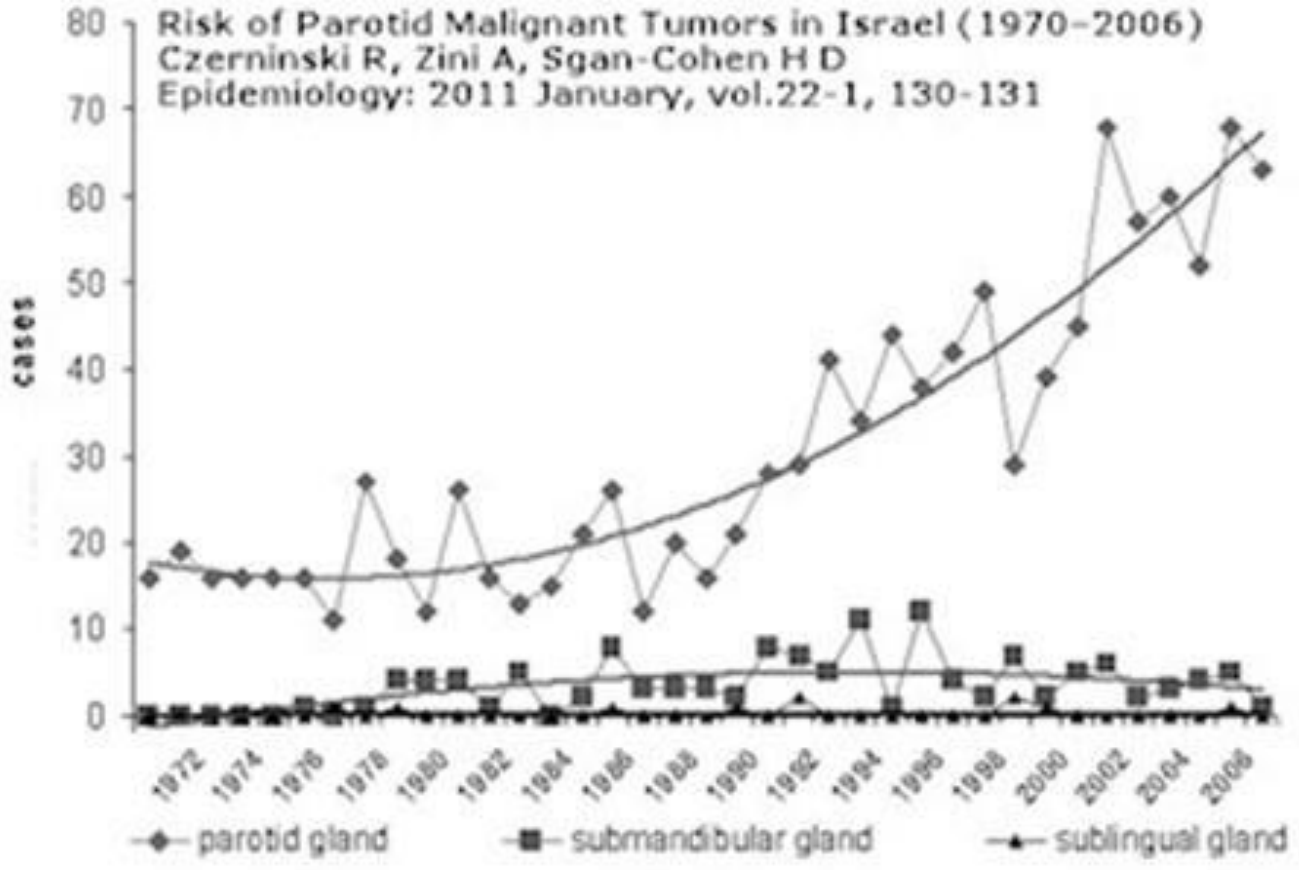
Source: [30]

Growth in Exposure to Microwave Radiation 2000-2010



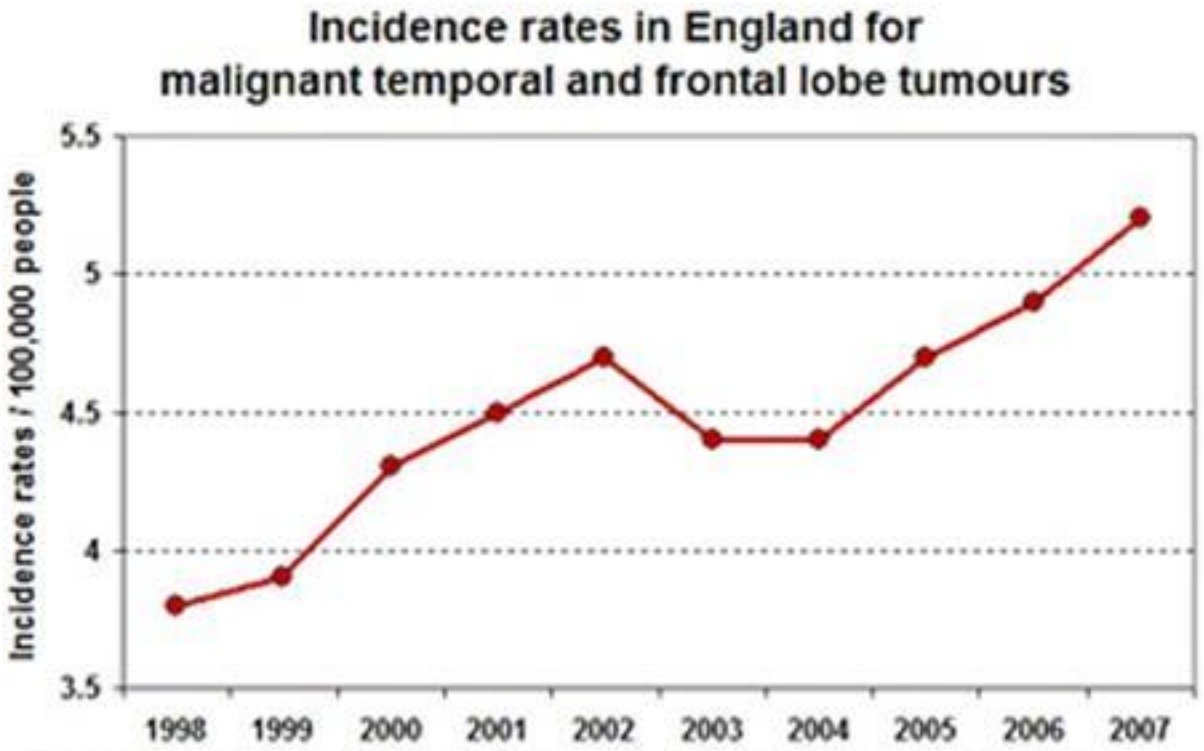
Next-Up Organisation

Parotid Gland Cancer-Israel 1970-2006



Tumors rose 4-fold on the side of head where phone was used.

Malignant Temporal and Frontal Lobe Tumors UK - 1998-2007



Data extracted from Table 1 in de Vocht F, Burstyn I, Cherrie JW. Time trends in brain cancer incidence rates in relation to mobile phone use in England. PMID:21280060 Bioelectromagnetics 2011 Jul;32(5):334-9.

The healing power of Minerals, special nutrients and trace elements
Paul Bergner, 1997 ,Prima Publishing

Mineral Famine:

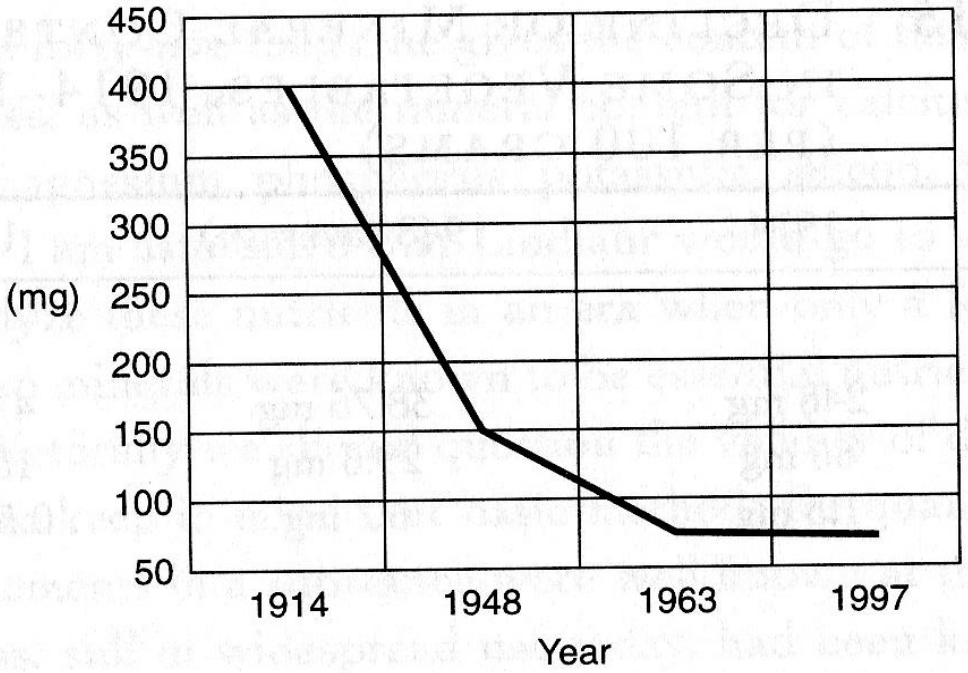


Figure 3.1 Average mineral content in selected vegetables, 1914–1997. Sums of averages of calcium, magnesium, and iron in cabbage, lettuce, tomatoes, and spinach. (Sources: Lindlahr, 1914; Hamaker, 1982; and U.S. Department of Agriculture, 1963 and 1997)

11 overlooked issues that can block our toxin elimination enzymes and turn us – over time - into a neurological patient

1. **Parasites (**the elephant in the living room):anti-parasitic drugs
2. **EMR**: exposure to microwave, electric- and magnetic fields: shield!
3. **Metal toxicity**: diagnose (or assume) and detox
4. **Mold** exposure (provocation/neutralization,anti-fungals, CSM)
5. **Dental issues**: a. toxicity from root filled teeth, amalgam fillings or cavitations b. electro-galvanism and microwave antenna function of dental metal restorations, allergy/toxicity from dental materials
6. **Methylation** block: caused by early psycho-emotional trauma, intra-uterine events or transgenerational(traumata of parents or grandparents)- B12, folate, Zn, B6, Mg. Psychological Trauma tx
7. **HPU**: Hemo-Pyrrol Lactam Uria (most often caused by stressful childhood events or accident in early years. Also transgenerational): Core, trauma tx, Prolotherapy c-spine
8. **Vascular inflammation and CCSVI**: stenoses of the anterior neck veins: balloon dilatation
9. Desynchronized **brain waves**: CES from www.littleTreeGroup.com,
10. Chronic **tonsillitis**: ozone injections, www.kryoPraxis.de and **sinusitis**: MARCoNS, neural therapy, intranasal flora (symbioflor 1 /2)
11. Decreased **“regulatory neuropeptides”**: MSH, OXT, ADH, VIP and melatonin (Homeo-K Harmony)

Parasites

Most commonly diagnosed:

- Whipworm (might be beneficial to suppress auto-immunity in intestines)
- Roundworms (ascaris, volvulus, looks like earthworms. Larvae migrate at night through the lungs)
- Schistosomiasis (Bilharzia, often lives in bladder,now found in lakes in US, Europe. Most common parasite after malaria. Competes for this position with amoebas)
- Strongyloides (looks like matted fiber in stool)

Rarely diagnosed:

- Varestrongylus Klapowi (lungworm. Pos.in over 80 % of clients with fatiguing symptoms. Tx: inhalation with ETOH, iodine or ASEA
- Ropeworm (lines small and large intestine. Often misdiagnosed as “false lining”, mucus or biofilm)

Worms affect the brain – you become stupid.
Would it not be good, to diagnose and treat?

Psychological Bulletin
1997, Vol. 121, No. 2. 171-191

0033-2909/97/$3.00

'Stupidity or Worms": Do Intestinal Worms Impair Mental Performance?

William E. Watkins and Ernesto Pollitt
University of California at Davis

The title of a 1930s article asked the question, "Stupidity or Hookworm?" In this article, the authors discuss research that attempts to answer the question of whether intestinal worms namely, hookworm, whipworm, and **roundworm**—harm the mental performance of their hosts. After Introducing the biology and epidemiology of intestinal worms, the authors present the Historical background to the problem. They review research from the 1910s through the 1990s; there is evidence that high intensities of worms can affect mental performance, but not all dewormed children show Improved performance. They discuss the mechanisms of how worms might affect the mind.

Nuclear Weapons and Neglected Diseases: The "Ten-Thousand-to-One Gap". **PLoS;** Negl Trop Dis 4(4): e680; Hotez PJ (2010)

Abstract: **Each of the 11 nuclear weapons states** also suffer from high rates of neglected tropical diseases (and related neglected infections of poverty), defined as chronic and **debilitating parasitic** and other infectious **diseases** that occur in association with extreme poverty. In addition to their health effects, the neglected tropical diseases also cause poverty through their ability to **impair child physical and intellectual development, pregnancy outcomes, and worker productivity**, while simultaneously promoting conflict and war through their agriculturally and socially destabilizing effects.

Although it is common to think of neglected diseases as confined to low-income countries in sub-Saharan Africa, Southeast Asia, and Latin America, as shown in these infections also exhibit a high prevalence in middle-income countries such as China, India, Pakistan, North Korea, Iran, and Syria, as well as **in selected areas** of poverty found **in the US**, Russia, and Eastern Europe. Indeed high neglected disease burdens are present in all of the nuclear weapons states**, particularly the helminth infections, leishmaniasis and Chagas disease, toxoplasmosis, and trachoma.**

N Engl J Med. 1992 Sep 3;327(10):692-5.

Neurocysticercosis in an Orthodox Jewish community in New York City.

Schantz PM, Moore AC, Muñoz JL, Hartman BJ, Schaefer JA, Aron AM, Persaud D, Sarti E, Wilson M, Flisser A.
Division of Parasitic Diseases, Centers for Disease Control, Atlanta 30333.

Abstract

BACKGROUND AND METHODS:

From June 1990 through July 1991, intracerebral infection with the larval stage of the pork tapeworm Taenia solium was diagnosed in four unrelated persons in an Orthodox Jewish community in New York City. None of the patients had eaten pork, and only one had traveled to a country in which T. solium infection was endemic. We investigated this outbreak, screened serum samples from family members and household contacts for antibodies to cysticercosis, and examined stool specimens from household employees for eggs of taenia species.

RESULTS:

The four patients had **recurrent seizures and brain lesions** that were radiologically consistent with the presence of cysticerci. The diagnosis was confirmed in two patients by a brain biopsy, and in two by immunoblot assays for cysticercus antibodies. **Of 17 immediate family members screened serologically, 7 from two families had cysticercus antibodies**. Magnetic resonance imaging of the brain showed cystic lesions in two of the seropositive family members, one of whom had had a seizure. **Examinations of six domestic employees from all four households revealed an active infection with taenia species in one and a positive serologic test in another.** Since these women had recently emigrated from Latin American countries where T. solium infection is endemic, they were the most likely sources of infection in the members of these households.

CONCLUSIONS:

A diagnosis of neurocysticercosis should be considered in patients with seizures and radiologic evidence of cystic brain lesions, even in those who do not eat pork and who have not traveled to a country in which T. solium infection is endemic. **Recent emigrants from countries in which T. solium infection is endemic should be screened for tapeworm infection in their stools before they are employed as housekeepers or food handlers.**

Parasites in the Lung?

J Clin Pathol. 2005 Apr;58(4):420-2.

Strongyloides stercolaris infection mimicking a malignant tumour in a non immunocompromised patient. Diagnosis by bronchoalveolar cytology.

Mayayo E, Gomez-Aracil V, Azua-Blanco J, Azua-Romeo J, Capilla J, Mayayo R.

Source

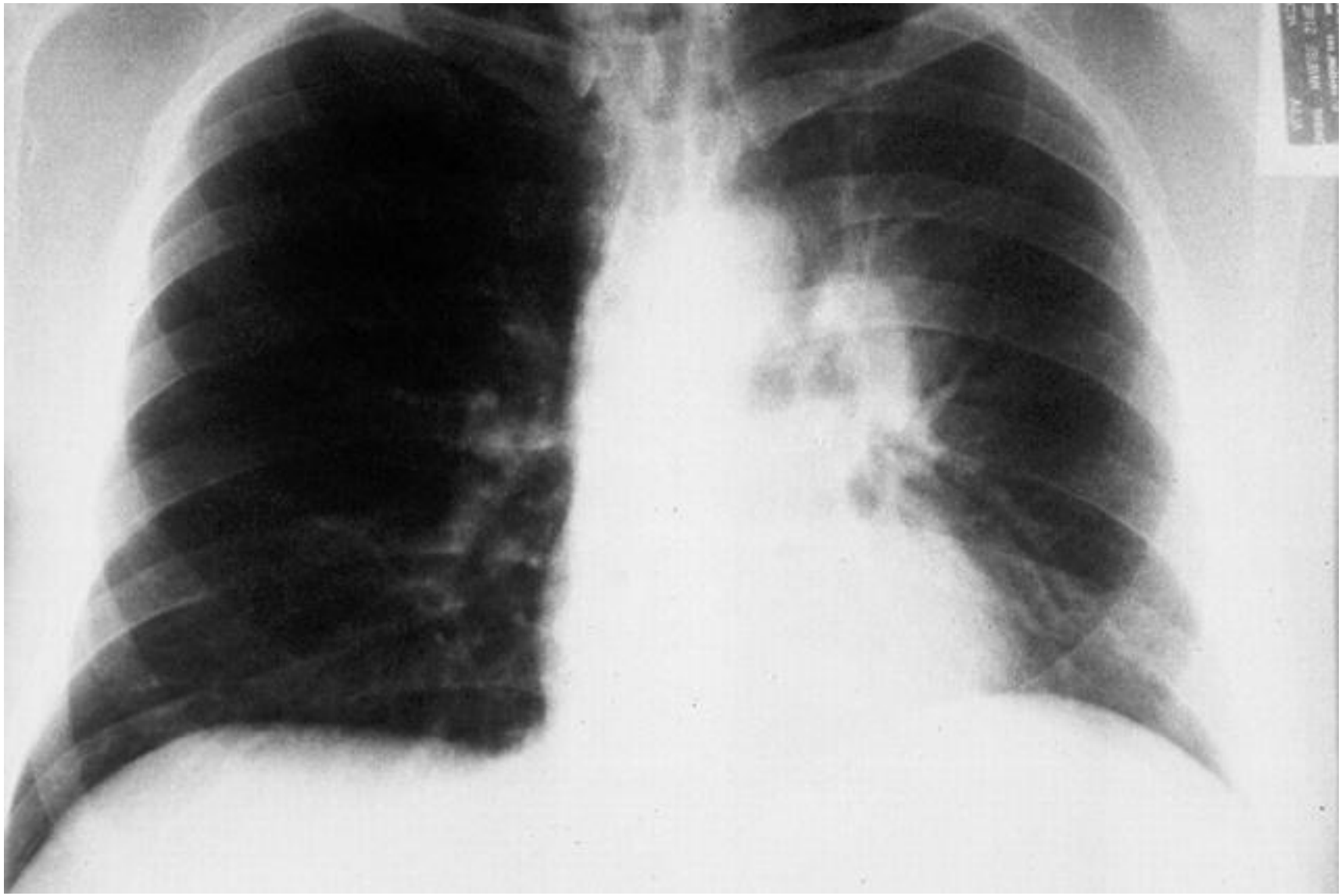
Service of Pathology, Hospital Universitario de Tarragona Juan XXIII and Department of Ciéncias Médicas Básicas, Facultad de Medicina, Universidad Rovira y Virgili, 43201 Tarragona, Spain. ema@fmcs.urv.es

Abstract

Autoinfective strongyloidiasis is often fatal in immunosuppressed patients or in immunocomprised hosts. An interesting case of Strongyloides stercolaris hyperinfection was seen in an immunocompetent patient. This report describes a case of fatal strogyloidiasis in a 79 year old man, who had suffered gastrointestinal discomfort for years, and who presented because of respiratory illness. A chest radiograph showed an irregular mass close to the mediastinum and interstitial infiltrates, but blood eosinophilia was not observed. Cytological examination of the samples obtained from bronchial aspiration and brushing identified several filariform larvae. Thus, cytology was essential for the correct diagnosis in this patient and is a very reliable method to diagnose lung parasitosis.

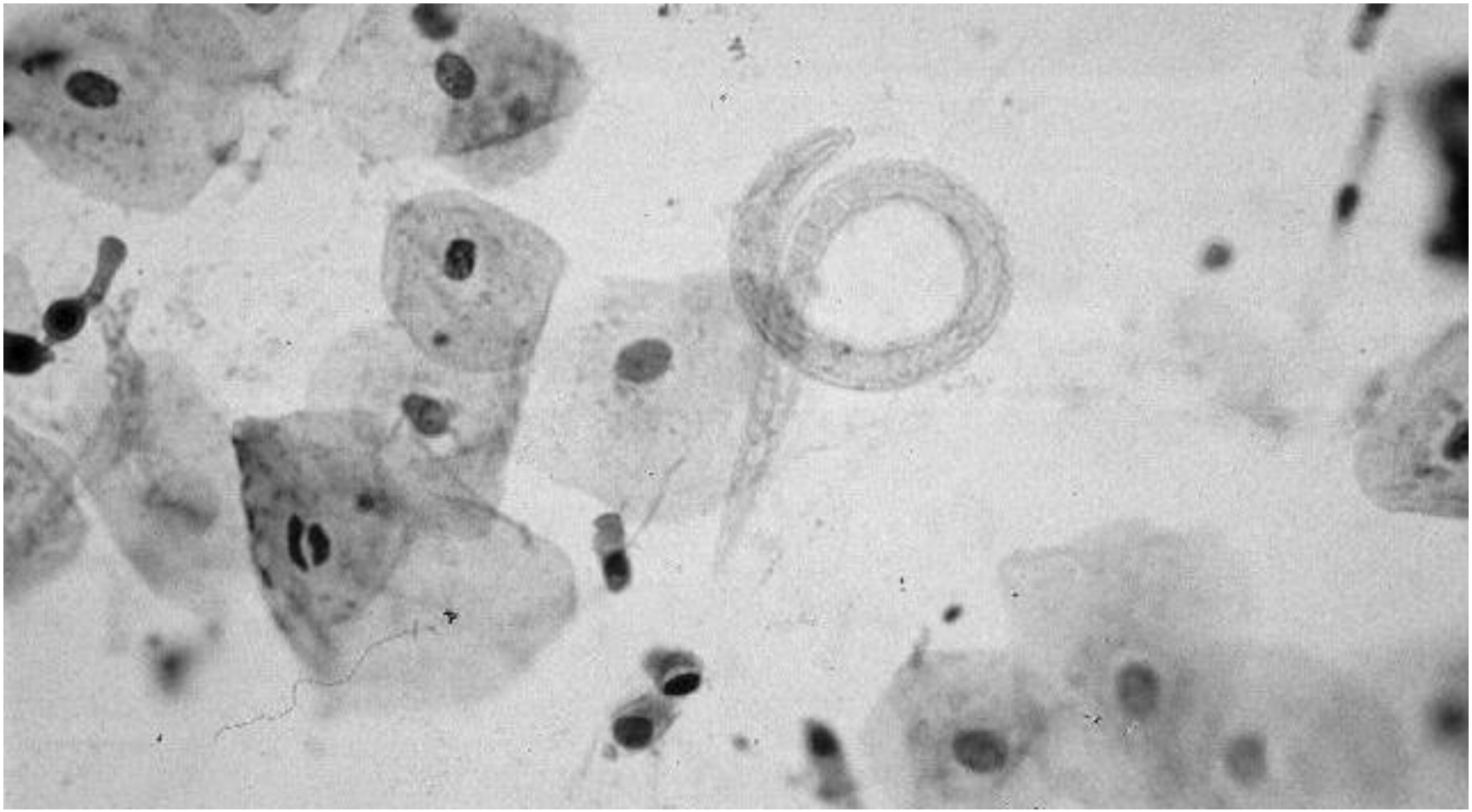
PMID: 15790710 [PubMed - indexed for MEDLINE] PMCID: PMC1770632

http://www.ncbi.nlm.nih.gov/pubmed/15790710



Squamous cells, some bronchial cells, and the presence of a filariform larva (Papanicolaou stain; original magnification, ×400)

J Clin Pathol. 2005 April; 58(4): 420–422.



Ivermectin and Propolis as Anti-Cancer Agents

Drug Discov Ther. 2009 ;3(6):243-246.

Ivermectin inactivates the kinase PAK1 and blocks the PAK1-dependent growth of human ovarian cancer and NF2 tumor cell lines.

Hashimoto H, Messerli SM, Sudo T, Maruta H

ABSTRACT: Ivermectin is an old anti-parasitic antibiotic which selectively kills nematodes at a very low dose (0.2 mg/kg) by inhibiting their GABA (gamma-aminobutyric acid) receptor, but not mammalian counterpart. Interestingly, several years ago it was reported by a Russian group that Ivermectin can suppress almost completely the growth of human melanoma and a few other cancer xenografts in mice at the much higher doses (3-5 mg/kg) without any adverse effect on mice. However, its anti-cancer mechanism still remained to be clarified at the molecular levels, that would determine the specific type of cancers susceptible to this drug. The first hint towards its anti-PAK1 potential was a recent finding that Ivermectin at its sublethal doses dramatically reduces the litter size (number of eggs laid) of the tiny nematode C. elegans. Interestingly, either a PAK1-deficiency (gene knock-out) or treatment with natural anti-PAK1 products such as CAPE (caffeic acid phenethyl ester) and ARC (artepillin C), the major anti-cancer ingredients in propolis, also causes the exactly same effect on this nematode, suggesting the possibility that the kinase PAK1 might be a new target of Ivermectin. This kinase is required for the growth of more than 70% of human cancers such as pancreatic, colon, breast and prostate cancers and NF (neurofibromatosis) tumors. Here we demonstrate for the first time that Ivermectin blocks the oncogenic kinase PAK1 in human ovarian cancer and NF2-deficient Schwannoma cell lines to suppress their PAK1-dependent growth in cell culture, with the IC50 between 5-20 μM depending on cell lines.

Key Words: Ivermectin, PAK1, ovarian cancer, neurofibromatosis type 2 (NF2), C. elegans

Chronic Schistosomiasis

Schistosomiasis is a disease that is caused by parasites (genus Schistosoma) that enter humans by attaching to the skin, penetrating it, and then **migrating through the venous system** to the portal veins where the parasites produce eggs and eventually, the symptoms of acute or chronic disease (for example, fever, abdominal discomfort, blood in stools). This disease is also known as Bilharziosis. **Schistosomiasis is the second most prevalent tropical disease in the world**; malaria is the first. The disease is found mainly in Africa**, Asia, South America, the Middle East, and the Caribbean**. About 207 million people in at least 74 countries are estimated to have the disease**. In the U.S., it is diagnosed in tourists who have visited** these developing countries and in visitors from these countries, or from lab accidents. The type of snail that is part of the parasite's life cycle (see below) is not endemic to U.S. freshwater sources, so the disease is not endemic in the U.S. Acute schistosomiasis may reach a death (mortality) rate of 25%, although most areas report lower rates.

- Schistosomiasis is a disease caused by Schistosoma spp. that can cause acute and chronic infection with many symptoms that frequently include fever, blood in stools or urine, and abdominal discomfort.
- The immune response and Schistosoma spp. egg migration through tissues and their deposition in body organs cause the disease.
- Schistosomiasis has an acute and chronic phase.
- Schistosomiasis is diagnosed by the identification of characteristic eggs in feces, urine, or biopsy samples; diagnosis may be aided with serologic (blood) tests.
- Schistosomiasis is most often effectively treated with the antiparasitic drug **praziquantel (Biltricide)**, especially in acute phase disease.
- Chronic schistosomiasis often produces complications in various organ systems (for example, gastrointestinal system, urinary system, heart, liver).
- Currently, there is no vaccine available for schistosomiasis.

- REFERENCES:
United States. Centers for Disease Control and Prevention. "Schistosomiasis." July 20, 2009. <http://www.dpd.cdc.gov/dpdx/HTML/Schistosomiasis.htm>.

The majority of people who develop **chronic schistosomiasis** have symptoms develop months or years after the initial exposure to the parasites. The following is a list of most symptoms associated with chronic schistosomiasis. Patients usually have a few of these symptoms:

- Abdominal pain
- Abdominal swelling (ascites)
- Bloody diarrhea or blood in the stools
- Blood in the urine and painful urination
- Shortness of breath and coughing
- Weakness
- Chest pain and palpitations
- Seizures
- Paralysis
- Mental status changes
- Lesions on the vulva or the perianal area

Diagnosis

The presumptive diagnosis of schistosomiasis is based on the medical caregiver's history and physical examination of the patient. Thick fecal smears and urine concentration tests are used to determine if any Schistosoma spp. eggs are present.

Blood tests and, more recently, polymerase chain reaction (PCR) tests can help confirm the diagnosis, but positive results may only indicate past exposure. However, these tests are not usually positive until the patient has been infected for about six to eight weeks because it takes time for the eggs to develop and stimulate the human immune response. The PCR test is available from the U.S. Centers for Disease Control and Prevention.

Many other tests and procedures may be necessary to establish the diagnosis, especially if no eggs are found in the feces or urine, which is often the situation in chronic schistosomiasis. Colonoscopy cystoscopy endoscopy and liver biopsy are all methods that can be used to obtain tissue biopsy material. In addition, ultrasound, chest X-rays, CT MRI and echocardiograms may be used to determine the extent of the infection in various organ systems.

Zur Chemie und Toxikologie der Ascariden
Archives of Pharmacology Volume 67, Numbers 4-5,
275-392 Ferdinand Flury
Ascaris is wide spread in Western countries. It produces significant biotoxins, that produce symptoms identical to those of Lyme disease

Ein Beitrag zur Physiologie und zum Wirt-Parasit-Verhältnis
von Graphidium strigosum (Trichostrongylidae, Nematoda**)**
Parasitology Research, Volume 10, Number 3, 386-414,
Karl Enigk
This parasite - like many others - takes over the physiology of the host by modulating the host's regulatory neuropeptides and several aspects of the host immunity

Emerging and Recurring Helminthiases and the Public Health of China
Perspectives Vol3, No3, July-Sept 1997, 303-310 Peter Hotez
Worms are common, difficult if not impossible to permanently eliminate from an infested system. Most helminths have been successfully exported by now to all western countries, including the US and Canada

Genes Dev. 2008 June 15; 22(12): 1636–1646.

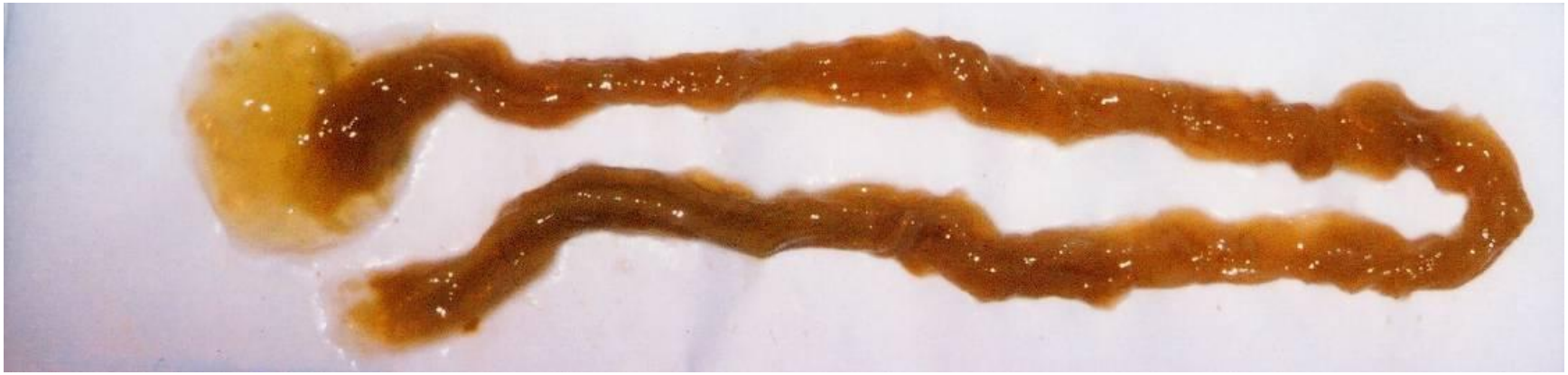
An *Entamoeba histolytica* rhomboid protease with atypical specificity cleaves a surface lectin involved in phagocytosis and immune evasion

Leigh A. Baxt,[1] Rosanna P. Baker,[2] Upinder Singh,[1,4] and Sinisa Urban[2,3]
Department of Molecular Biology and Genetics, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205, USA

Abstract

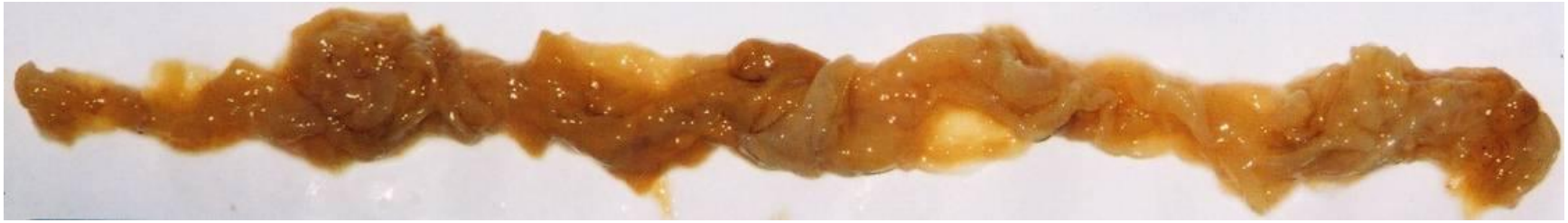
Rhomboid proteases are membrane-embedded enzymes conserved in all kingdoms of life, but their cellular functions across evolution are largely unknown. Prior work has uncovered a role for rhomboid enzymes in host cell invasion by malaria and related intracellular parasites, but this is unlikely to be a widespread function, even in pathogens, since rhomboid proteases are also conserved in unrelated protozoa that maintain an extracellular existence. We examined rhomboid function in ***Entamoeba histolytica*****,** an extracellular, parasitic ameba that is **second only to malaria in medical burden globally**. Despite its large genome, *E. histolytica* encodes only one rhomboid (EhROM1) with residues necessary for protease activity. EhROM1 displayed atypical substrate specificity, being able to cleave *Plasmodium* adhesins but not the canonical substrate *Drosophila* Spitz. We searched for substrates encoded in the ameba genome and found EhROM1 was able to cleave a cell surface lectin specifically. In *E. histolytica* trophozoites, EhROM1 changed localization to vesicles during phagocytosis and to the posterior cap structure during surface receptor shedding for immune evasion, in both cases colocalizing with lectins. Collectively these results implicate rhomboid proteases for the first time in immune evasion and suggest that a common function of rhomboid enzymes in widely divergent protozoan pathogens is to break down adhesion proteins.

The Rope Parasite




2 cm

US university lab: mucous, food residues, shed gut lining
DNA probing in German lab: Parasite. Taxonomy unknown
Russian parasitology, university level: “Rope Parasite”



Development stages of the "rope" human intestinal parasite

Submitted to arxiv.org on Jan. 13, 2013, Alex A. Volinsky, Ph.D a*, Nikolai V. Gubarev, Ph.D b, Galina M. Orlovskaya, RN-C c, Elena V. Marchenko, M.D., Ph.D , Department of Mechanical Engineering, University of South Florida, Tampa FL 33620, USA, Occupational Safety Ltd. (OOO "Bezopasnost Truda"), 32 ul. Koli Tomchaka, suite 14, St.Petersburg 196084, Russia, Department of Surgery, St. Petersburg City Hospital No. 15, 4 Avangard St., St. Petersburg 198205, Russia, Formerly research volunteer at H. Lee Moffitt Cancer Center and Research Institute, 12902 USF Magnolia Drive, Tampa FL 33612, USA, Corresponding author. Phone: +1 813 974 5658, Fax: +1 813 974 3539, Email: volinsky@usf.edu

Abstract

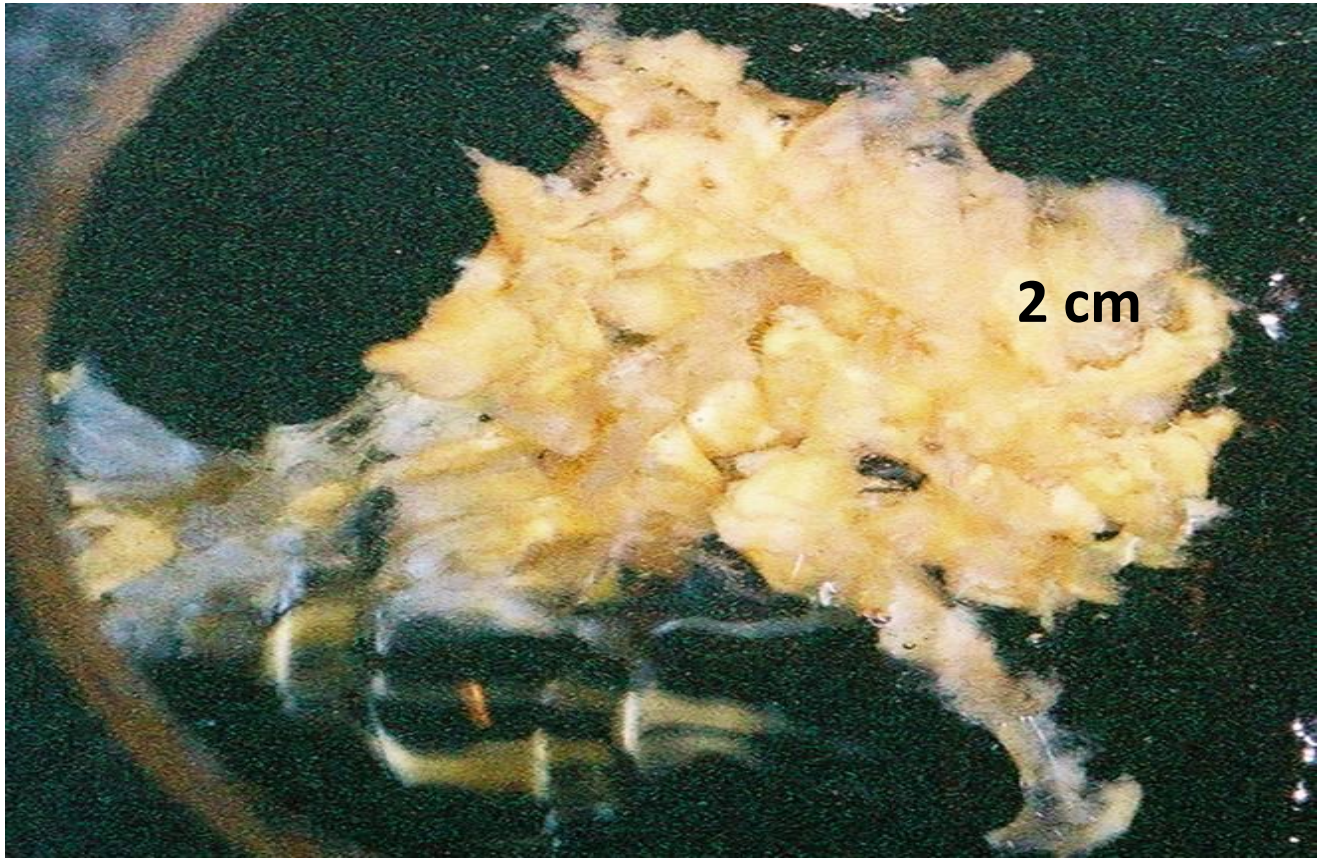
This paper describes the five development stages of the rope human parasite. Rope parasites have been discovered as a result of cleansing enemas. Parasite adult stages live in human gastrointestinal tract and are anaerobic. They move inside the body by releasing gas bubbles utilizing jet propulsion. Rope parasites look like a rope, and can be over a meter long. It takes tens of years for them to fully develop into mature species (fifth stage). The fourth stage looks similar, but the parasite is shorter and has softer slimier body. The third stage looks like branched jellyfish. The second stage is viscous snot, or mucus with visible gas bubbles that act as suction cups. The first stage is slimier mucus with fewer bubbles, which can reside almost anywhere in the body. Antihelminthic methods are also mentioned in the paper.

Keywords: New taxa; rope parasite; *funis parasitus; helminths; human intestinal parasite;* development stages.

Introduction

Human parasitic worms are classified as nematodes (roundworms), cestodes (tapeworms), trematodes (flukes) and monogeneans (Grove, 1990). It is estimated that every fourth human is hosting intestinal parasites (Watkins and Pollitt, 1997, World Development Report, 1993), meaning that even more people carry parasite intermediate stages. Humans can also carry intermediate stages of animal parasites, such as cat ascaris worms. Parasitic worms have different life cycles, sometimes using humans as permanent or temporary hosts. What if there is a parasite that does not have intermediate stages outside the human body, lives and dies with the human? Such specie, called rope parasite, or ***funis parasitus in*** *Latin, has been recently discovered and described (Gubarev, 2009,* Volinsky et. al. 2013). It does not fall under a single known parasite category. Based on its attributes, this pre-nematode may be older than other parasites.

Branched jellyfish 3rd stage of the rope parasite development.




GLACIER BAY

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Gubarev N.V., Gubarev A.V., Lebedev S.A., Orlovskaya L.P., Orlovskaya G.M., Pakulina O.N. 2007. Method of Human dehilminthation/Sposob izgnaniya gelmintov iz organizma cheloveka, Russian Federation Patent RU2270688.

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First Class: Special Literature, 2009. -109 p. : ill. – ISBN 978-5-903984-08-4.

Parasites: an uneasy alliance

Parasitology / Volume 126 / Issue 07 / March 2003, pp S53-S60

- DOI: http://dx.doi.org/10.1017/S003118200300372X (About DOI), Published online: 10 November 2003

- **Accumulation of heavy metals by intestinal helminths in fish: an overview and perspective**

- **B. SURES** [a1][c1]
  [a1] Zoologisches Institut – Ökologie/Parasitologie, Geb. 07.01, Universität Karlsruhe, Kornblumenstr. 13, 76128 Karlsruhe, Germany

- **Abstract**
- Intestinal helminths of fish are of increasing interest as potential bioindicators for heavy metal contamination in aquatic habitats. **Among these parasites cestodes and acanthocephalans in particular have an enormous heavy metal accumulation capacity exceeding that of established free living sentinels.** Metal concentrations several thousand times higher in acanthocephalans than in host tissues were described from field and laboratory studies. Whereas larval stages inside their intermediate hosts are not able to take up high quantities of metals, young worms begin to take up metals immediately after infection of the final host. After four to five weeks of exposure, the parasites reach a steady-state concentration orders of magnitude higher than the ambient water level. Thus, acanthocephalans are not only very effective in taking up metals, but they can also respond very rapidly to changes in environmental exposure. The mechanism which enable acanthocephalans to take up metals from the intestinal lumen of the host appears to be based on the presence of bile acids, which form organo-metallic complexes that are easily absorbed by the worms due to their lipophilicity. Investigations of the environmental conditions affecting metal uptake have shown that the parasites are more consistent and reliable indicators for metal pollution than the host tissues as metal levels of the latter are much more dependent on the water chemistry. Thus, after some years of research on the uptake of metals by acanthocephalans and on the factors affecting metal accumulation in intestinal parasites it should be asked if acanthocephalans meet the criteria commonly accepted for sentinels. If parasites can be considered as promising sentinels, we need reasons for the establishment of 'new' indicators. Therefore, this review summarises the present knowledge about parasites as bioindicators and compares the accumulation properties of parasites and established free living indicators. Finally, this review presents possible answers to the question why it could be advantageous to have new and even more sensitive indicators for environmental monitoring purposes.

Parasites: an uneasy alliance

Parasit Vectors. 2013 Jan 18;6:21. doi: 10.1186/1756-3305-6-21.

Comparison of the metal accumulation capacity between the acanthocephalan Pomphorhynchus laevis and larval nematodes of the genus Eustrongylides sp. infecting barbel (Barbus barbus).

Nachev M, Schertzinger G, Sures B.

Source

Aquatische Ökologie and Zentrum für Wasser-und Umweltforschung, Universität Duisburg-Essen, Universitätsstraße 5, D 45141, Essen, Germany. Milen.Nachev@uni-due.de

Abstract

BACKGROUND:

Metal uptake and accumulation in fish parasites largely depends on the parasite group with acanthocephalans showingthe highest accumulation rates. Additionally, developmental stage (larvae or adult) as well as parasite location in the host are suggested to be decisive factors for metal bioconcentration in parasites. By using barbel (Barbus barbus) simultaneously infected with nematode larvae in the body cavity and adult acanthocephalans in the intestine, the relative importance of all of these factors was compared in the same host.

METHODS:

Eleven elements Arsenic (As), Cadmium (Cd), Cobalt (Co), Copper (Cu), Iron (Fe), Manganese (Mn), Lead (Pb), Selenium(Se), Tin (Sn), Vanadium (V) and Zinc (Zn) were analyzed in barbel tissues (muscle, intestine, liver) as well as in their acanthocephalan parasites Pomphorhynchus laevis and the larval nematode Eustrongylides sp. (L4) using inductively coupled plasma mass spectrometry (ICP-MS).

RESULTS:

Nine elements were detected in significantly higher levels in the parasites compared to host tissues. The element composition among parasites was found to be strongly dependent on parasite taxa/developmental stage and localization within the host. Intestinal acanthocephalans accumulated mainly toxic elements (As, Cd, Pb), whereas the intraperitoneal nematodes bioconcentrated essential elements (Co, Cu, Fe, Se, Zn).

CONCLUSION:

Our results suggest that in addition to acanthocephalans, **nematodes** such as Eustrongylides sp**. can also be applied as bioindicators for metal pollution**. Using both parasite taxa simultaneously levels of a wide variety of elements (essential and non essential) can easily be obtained. Therefore this host-parasite system can be suggested as an appropriate tool for future metal monitoring studies, if double infected fish hosts are available.

Parasites: an uneasy alliance

Parasitologia. 2007 Sep;49(3):173-6.

Host-parasite interactions from an ecotoxicological perspective.

Sures B.

Applied Zoology/Hydrobiology, University of Duisburg-Essen, D-45117 Essen, Germany.

Bernd.Sures@uni-due.de

Abstract

- In recent years there has been an increasing number of papers showing how parasitism and pollution can interact with each other in aquatic organisms. Apart from parasitological aspects these interactions are also important in terms of ecotoxicological research. The current presentation aims at identifying three promising directions for future research in the interdisciplinary field of parasitology and ecotoxicology. 1. **Parasites as sinks for pollutants within their hosts: Some parasites are able to reduce pollutant levels in the tissues of their host.** The reduction of pollutants is an interesting implication since parasites are beneficial to their hosts from this perspective. In other cases free-living accumulation indicators may erroneously indicate low levels of pollution if they are infected with parasites. 2. Parasites as a diagnostic tool to test bioavailability of substances. In order to take up and accumulate pollutants the substances have to be metabolized by the host first. Accordingly, the detection of substances within endoparasites is a sign for the biological availability of pollutants. 3. Changes of biomarker responses of the host against pollutants. Parasites can alter physiological reactions of their hosts against pollutants in different ways. Therefore, in ecotoxicological studies, examining the question whether exposure to certain chemicals affects the physiological homeostasis of a test organism, it is important to use organisms that are known to be uninfected.
- PMID: 18410076 [PubMed - indexed for MEDLINE]

Parasites: an uneasy alliance

- Adv Parasitol. 1991;30:201-38.
- **Influence of pollution on parasites of aquatic animals.**
- Khan RA, Thulin J.
- **Source**
- Department of Biology and Ocean Sciences Centre, Memorial University of Newfoundland, St John's, Canada.
- **Abstract**
- We have tried to draw attention to an increasing body of evidence (from several publications) that **parasites of fish might be useful indicators of pollution**. Several types of pollutants, including domestic sewage, pesticides, polychlorinated biphenyls, heavy metals, pulp and paper effluents, petroleum aromatic hydrocarbons, acid rain, and others, are known to affect aquatic animals. Many of the latter are parasitized and, under natural environmental conditions, most fish parasites are believed to cause little or no harm. However, chronic exposure to pollutants over a period of time causes biochemical, physiological and behavioural host changes that ultimately can influence the prevalence and intensity of parasitism. Some of these changes include host nutrition, growth and reproduction. Macroscopic lesions might not always be apparent, but subtle disorders in several specific tissues and organs might occur. Pollutants might promote increased parasitism in aquatic animals, especially fish, by impairing the host's immune response or favouring the survival and reproduction of the intermediate hosts. Alternatively, decreased parasitism might ensue through toxicity of the pollutant to free-living stages and intermediate hosts or by alteration of the host's physiology. Experimental studies indicate that the numbers of ectoparasites such as trichodinid ciliates and monogeneans increase significantly on the gills following exposure to a pollutant, and this is supported by field data on other ciliates and monogeneans where evidence of pollution has been clearly demonstrated. There is also evidence that endoparasitic protozoons, such as myxozoons, microsporans and haematozoons, all of which are capable of proliferating in their hosts, increase substantially in prevalence and intensity when interacting with pollutants. The period of patency might also be prolonged in haematozoan infections. Most reports of pollution effects on endoparasites suggest increased parasitism in fish hosts. This also applies to fish living in areas which receive thermal effluents. Parasites might in turn enhance their hosts' susceptibility to pollutants, and information in support of this view is accumulating. Finally, immunosuppression represents one of the underlying mechanisms influencing increased parasitism. Thus, while published information suggests more than a casual connection between fish parasites and pollution, further research is needed to establish the cause-and-effect relationship and at the same time take cognizance of histopathological effects of the toxic agents and their concentrations in water. Areas for future research are recommended.
-

Parasites: an uneasy alliance

- Parasit Vectors. 2013 Jan 18;6:21. doi: 10.1186/1756-3305-6-21.

- **Comparison of the metal accumulation capacity between the acanthocephalan Pomphorhynchus laevis and larval nematodes of the genus Eustrongylides sp. infecting barbel (Barbus barbus).**

- Nachev M, Schertzinger G, Sures B.
- **Source**
- Aquatische Ökologie and Zentrum für Wasser-und Umweltforschung, Universität Duisburg-Essen, Universitätsstraße 5, D-45141, Essen, Germany. Milen.Nachev@uni-due.de

- **Abstract**
- ***BACKGROUND:***
- Metal uptake and accumulation in fish parasites largely depends on the parasite group with acanthocephalans showing the highest accumulation rates. Additionally, developmental stage (larvae or adult) as well as parasite location in the host are suggested to be decisive factors for metal bioconcentration in parasites. By using barbel (Barbus barbus) simultaneously infected with nematode larvae in the body cavity and adult acanthocephalans in the intestine, the relative importance of all of these factors was compared in the same host.
- ***METHODS:***
- Eleven elements Arsenic (As), Cadmium (Cd), Cobalt (Co), Copper (Cu), Iron (Fe), Manganese (Mn), Lead (Pb), Selenium (Se), Tin (Sn), Vanadium (V) and Zinc (Zn) were analyzed in barbel tissues (muscle, intestine, liver) as well as in their acanthocephalan parasites Pomphorhynchus laevis and the larval nematode Eustrongylides sp. (L4) using inductively coupled plasma mass spectrometry (ICP-MS).
- ***RESULTS:***
- Nine elements were detected in significantly higher levels in the parasites compared to host tissues. The element composition among parasites was found to be strongly dependent on parasite taxa/developmental stage and localization within the host. **Intestinal acanthocephalans accumulated mainly toxic elements (As, Cd, Pb),** whereas the intraperitoneal nematodes bioconcentrated essential elements (Co, Cu, Fe, Se, Zn).
- ***CONCLUSION:***
- Our results suggest that in addition to acanthocephalans, **nematodes** such as Eustrongylides sp**. can also be applied as bioindicators for metal pollution**. Using both parasite taxa simultaneously levels of a wide variety of elements (essential and non essential) can easily be obtained. Therefore this host-parasite system can be suggested as an appropriate tool for future metal monitoring studies, if double infected fish hosts are available.

Parasites: an uneasy alliance

Environmental Pollution
Volume 157, Issues 8–9, August–September 2009, Pages 2584–2586

Influence of parasitism on the use of small terrestrial rodents in environmental pollution monitoring

Ivana Jankovská[a,*], Daniela Miholová[b], Iva Langrová[a], Vladimír Bejček[c], Jaroslav Vadlejch[a], Dana Kolihová[b], Miloslav Šulc[b]

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[b] Department of Chemistry, Faculty of Agrobiology, Food and Natural Resources, Czech University of Life Sciences, Kamycka 129, 165 21 Prague 6 – Suchdol, Czech Republic

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Abstract

- Bioaccumulation of cadmium, chromium, copper, manganese, nickel, lead and zinc in small terrestrial rodents – voles and their cestode parasite *Paranoplocephala dentata* was studied. Contents of Pb, Mn, Ni and Zn in the parasite were found to be higher than in the kidney and liver of the parasitized animals. **Lead** level in the cestode was **37 fold higher** than in the liver of the infected rodents. Bioaccumulation factors of zinc, nickel and manganese in the cestode are mostly in the range from 2 to 4.5. Considering the different contents of manganese and zinc in livers of non-parasitized and parasitized rodents, kidney tissue was found to be more reliable than liver as an indicator of environmental pollution by manganese and zinc; the kidneys of parasitized animals showed no significant change in the concentrations of those elements that are accumulated in the cestode.

Parasites: an uneasy alliance

Helminthologia 2000 Vol. 37 No. 1 pp. 15-18

Concentrations of some heavy metals in *Ligula intestinalis* plerocercoids (Cestoda) and *Philometra ovata* (Nematoda) compared to some of their hosts (Osteichthyes).

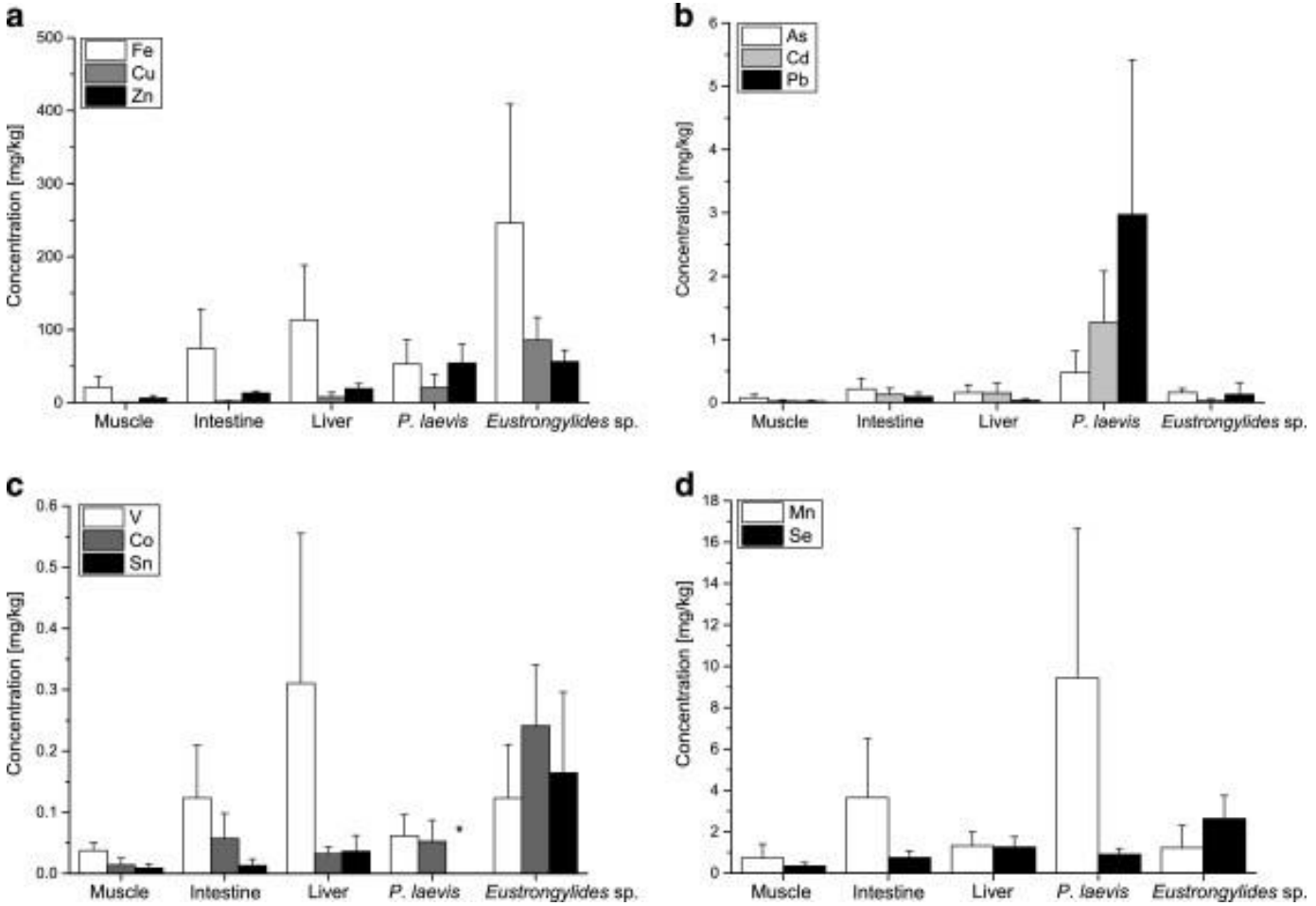
Tenora, F.; Baruš, V.; Kráčmar, S.; Dvořáček, J.

Abstract

Ligula intestinalis and *Philometra ovata* from the body cavity of 3 cyprinid fishes (*Abramis brama, Rutilus rutilus, Blicca bjoerkna*) were analysed for heavy metals using atomic absorption spectrometry. The **Pb, Cr and Cd** levels in *L. intestinalis* plero cercoids were 15X, 6X and 2.6X higher, respectively, than those in fish muscle, whereas the levels in *P. ovata* (adult females) were **106X, 43X and 119X higher**, respectively, than those in fish muscle.

[Comparison of the metal accumulation capacity between the acanthocephalan Pomphorhynchus laevis and larval nematodes of the genus Eustrongylides sp. infecting barbel (Barbus barbus)](#)
Parasit Vectors. 2013;6:21-21.

Mean (±S.D.) element concentrations (a–d) in organs of barbels and in its parasites *Pomphorhynchus laevis* and *Eustrongylides* sp.

- 

Parasites: an uneasy alliance

Cambridge Test Journal / Volume 138 / Issue 11 / September 2011, pp 1400-1405

The parasitic nematodes *Hysterothylacium* sp. type MB larvae as bioindicators of lead and cadmium: a comparative study of parasite and host tissues

H. KHALEGHZADEH-AHANGAR[a1], M. MALEK[a1] [c1] and K. McKENZIE[a2]
[a1] School of Biology, College of Science, University of Tehran, Iran
[a2] School of Biological Sciences, University of Aberdeen, Aberdeen, UK

SUMMARY
Cadmium and lead concentrations were compared in tissues of cutlassfish, *Trichiurus lepturus* L., its intestinal nematode *Hysterothylacium* sp. type MB larvae, and in water from the same location in the Sea of Oman. Metal accumulation in hosts, parasites and sea water was measured by ICP-OES. *Hysterothylacium* larvae from the intestinal lumen and visceral cavity showed much higher metal concentrations than in host tissues or sea water. Statistical analyses revealed no significant differences in metal accumulation between infected and uninfected hosts. Cadmium concentration in the host muscle was lower than in intestine, liver and gonad tissues.

The mean concentrations **of lead and cadmium in nematodes were 289·03 and 81·5 times higher than in host intestine, 188·4 and 225 times higher than in host muscle, 108·6 and 65·3 times higher than in host gonads, 70·5 and 19·5 times higher than in host liver and 3351 and 148 times higher than in sea water. The results show the value of this and possibly related nematodes as bioindicators of heavy metals** and their potential use in environmental studies.

Our own tests

- We had a patient's rope parasite evaluated for metal content by a Chicago based laboratory. The results were compared with the results of hair analysis
- Aluminum: 228 times increase
- Lead: 72 times increase
- Zinc: 62 times increase
- Magnesium 94 increase

Treatment

- Step 1: stop foods, behaviors and supplements that feed the parasite (processed foods, mushy foods, sweet and processed foods, unfermented dairy, all cooked or baked grains
- Step 2: establish toxic metal detoxification and mineral (=good metals) replacement protocol
- Step 3: Simon Yu 6 week protocol (medical anti-parasitics)
- Enema/suppository protocols: daily, 18 months, repeated courses of oral anti-helminthics (klinghardt protocols for liposomal **artemisinin cocktail, curcumin/ginger mix (CurcuSyn), mimosa pudica** and freeze dried garlic. Cleanest products:www.biopureUS.com)
- Optional (but powerful): elecromagnetic fields (sputnik, PEMF Dr.Gordon, Rife, etc.)

Environmental Health Perspectives
Vol. 46, pp. 57-62, 1982

Controlled Clinical Evaluations of Chlorine Dioxide, Chlorite and Chlorate in Man

by Judith R. Lubbers,* Sudha Chauan,* and Joseph R. Bianchine*

To assess the relative safety of chronically administered chlorine water disinfectants in man, a controlled study was undertaken. The clinical evaluation was conducted in the three phases common to investigational drug studies. Phase I, a rising does tolerance investigation, examined the acute effects of progressively increasing single doses of chlorine disinfectants to normal healthy adult male volunteers. Phase II considered the impact on normal subjects of daily ingestion of the disinfectants at a concentration of 5 mg/l. for twelve consecutive weeks. Persons with a low level of glucose-6-phosphate dehydrogenase may be expected to be especially susceptible to oxidative stress; therefore, in Phase III, chlorite at a concentration of 5 mg/l. was administered daily for twelve consecutive weeks to a small group of potentially at-risk glucose-6-phosphate dehydrogenase-deficient subjects. Physiological impact was assessed by evaluation of a battery of qualitative and quantitative tests. The three phases of this controlled double-blind clinical evaluation of chlorine dioxide and its potential metabolites in human male volunteer subjects were completed uneventfully. There were no obvious undesirable clinical sequellae noted by any of the participating subjects or by the observing medical team. In several cases, statistically significant trends in certain biochemical or physiological parameters were associated with treatment; however, none of these trends was judged to have physiological consequence. One cannot rule out the possibility that, over a longer treatment period, these trends might indeed achieve proportions of clinical importance. However, by the absence of detrimental physiological responses within the limits of the study, **the relative safety of oral ingestion of chlorine dioxide and its metabolites, chlorite and chlorate, was demonstrated.**

The Gubarev Protocols

This protocols are from Gubarev's patents and communications:
All enemas are preceded with a 2 liter cleaning water enema. Each type can be done once in 4 days.
They should be followed by another 2 liter water enema at the end of the day, or the next morning.
This is when the ropes come out. Find the type of enema that is the most effective.

1. 1 quart of whole milk with 2 table spoons of salt. Room temperature. Hold for 2 hours. A pound of mucus may come out as a result

2. 1 quart of water with 2 table spoons of baking soda. Room temperature. Hold for 2 hours.

3. Eucalyptus leaves (30 grams) boiled in water for 15 minutes, then cooled to 42C with added 30-100 drops of eucalyptus oil. Hold for 2 hours. After this juice from 5-6 lemons in 1 quart water. Hold for 2hours.

4. "Dead" water enema. Hold for 2 hours (electrolysis water. Instructions for making the instrument are given elsewhere)

5. One or two table spoons of vinegar in 1 liter of water. Hold for 2 hours. Best for fecal stones.

- As a result of these enemas the rope worms migrate to sinuses and lungs. Eucalyptus inhalations and “dead” water drops in the nose take care of that.
  These procedures are quite involved and time consuming, but chronic symptoms are relieved almost instantaneously when the large ones come out.
- Source: eucalyptus leaves: www.amazon.com, oil: “Young living oils” or “NOW”
- Alternative to “dead” water: inhalation with 5 ml ASEA (Omron hand held device)

Heavy Metals: what do we know?

- Are stored or even utilized by less evolved organisms (yeast, parasites, Borrelia, Babesia)
- Stored metals protect the parasite or microbe
- Metals in contaminated body compartments are transported by the macrophages and overload and exhaust our immunesystem
- Mercury and aluminum permanently disable functions of our immunesystem
- Metals cause autoimmunity and confusion in the host

Lessons learned from diagnosing and treating parasites for over 30 years

- Parasites thrive in metal contaminated environments
- Metal-enriched parasites cannot be eliminated by our own immunesystem
- Parasites modulate our immune system: TNF alpha goes up, so does TGF beta 1, IL-6, hsCRP. We become inflamed
- Patients with parasites develop nutritional deficiencies, convert testosterone and estrogen to cancer-causing estrones, cause any lab abnormality that we diagnose daily
- Parasite patients often behave like parasites, or are odd in thinking and feeling
- Parasite elimination requires work from both ends of the body and radical toxin elimination
- When the patient is successful and gets rid of the “thing”, the patient gets well, no matter what the original diagnosis was
- Lyme and co-infections, mold, auto-immunity, food allergies, CFIDS, MCS, colon, liver and pancreatic cancer are consequences, not cause

Biological Effects of Heavy Metal Toxicity

- **Neurotoxic: damage brain structures; lower IQ; down-regulates dopamine activity**
- **Nephrotoxic**
- **Immune dysregulation**
- **Cardiovascular**
- **Blood/Circulatory: Anemia, Raynaud's**
- **Bone & tissue deposits**
- **Dysbiosis: fungal mycotoxins**
- **Endocrine disruption: thyroid, adrenal, sex hormone**
- **Cognitive problems: ADHD, Alzheimer's**
- **Mood disorders: anxiety, depression, OCD**
- **Metabolic dysregulation: energy decline, Weight gain, type II diabetes, hypertension, elevated serum lipids, etc**

Mercury: Metal Toxicity Mechanisms

- **Denaturing enzymes**
- **Displace minerals in cells and tissues**
- **Interfere with cell membranes functions, ie transport, uptake and release**
- **Create free radicals/oxidative stress**
- **Induction of Inflammatory Cytokines**
- **Mitochondrial damage**
- **DNA damage**

Clarkson TW. The three modern faces of mercury. Environ Health Perspect 2002;110:11-23

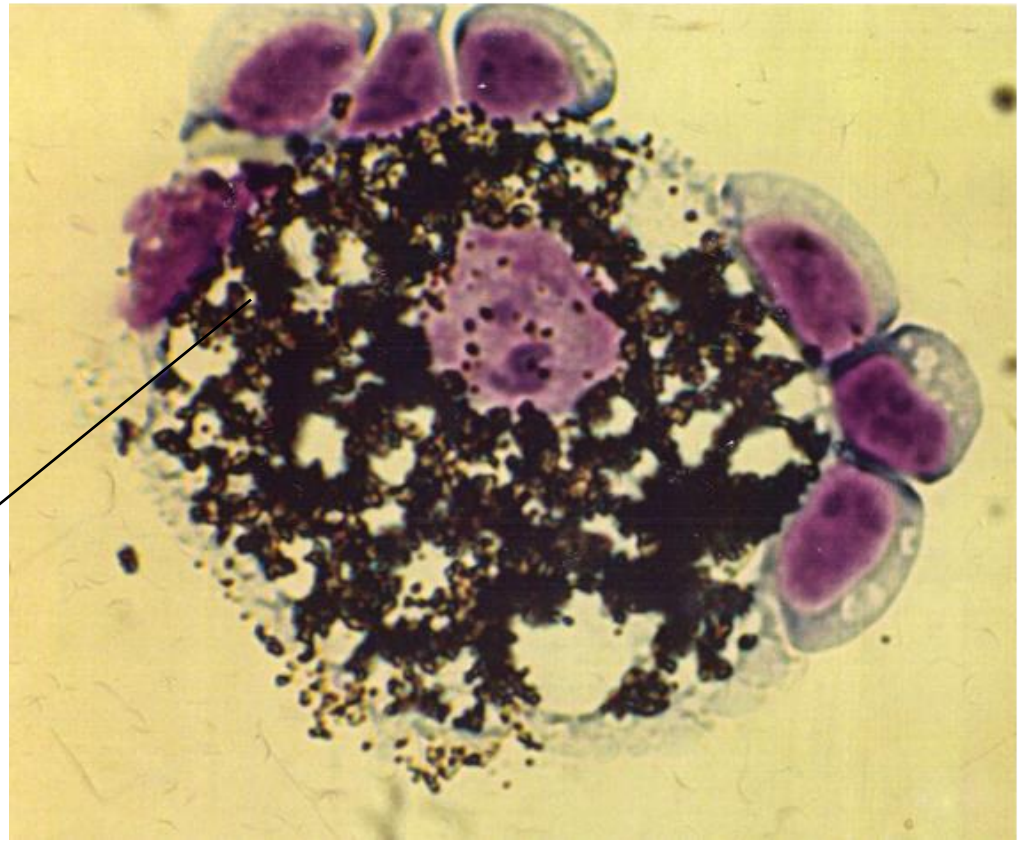
National Research Council. Toxicological Effects of Methylmercury. Washington, DC: National Academy Press 2000:31-70

Yee S, Choi BH. Oxidative Stress in Neurotoxic effects of methymercury poisoning. Neurotoxicology 1996;17:17-26

Mercury ,the Intestines and yeast

- Ingested or inhaled mercury inhibits neutrophils and their subsequent TH1 and TH2 cytokine effects which control Candida. Lowered neutrophil activity allows proliferation of candida.
- Candida organisms methylate mercury vapor from the mouth in the intestines and the mouth.
- Also candida albicans may trap mercury.
- Candida albicans and its mycotoxins are associated with chronic fatigue and autoimmune disorders.

Macrophages cannot handle metals and act as transporters of metal ions throughout the body



Titanium

Differentiate between:

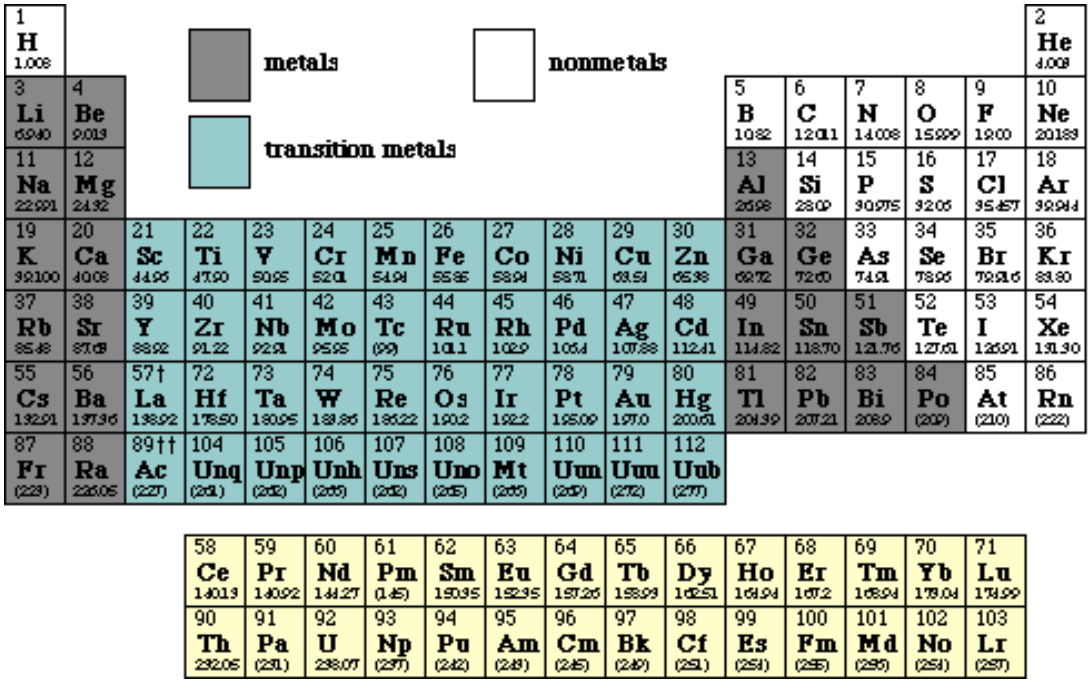
TOXIC EFFECTS

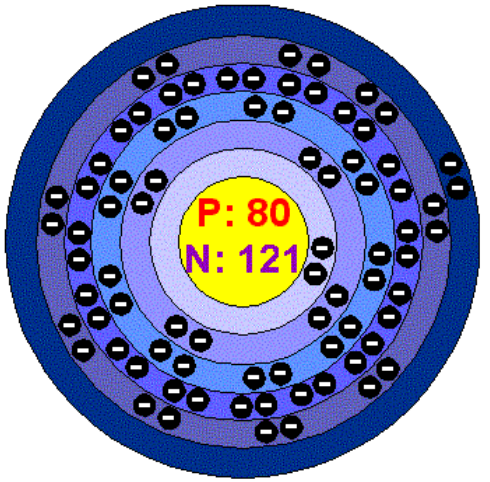
- higher doses
- single exposure
- lower specificity
- lower genetic influence

IMMUNOLOGIC EFFECTS

- lower doses
- chronic exposure
- higher specificity
- higher genetic influence, only certain individuals are affected

Metals cause allergy and autoimmunity



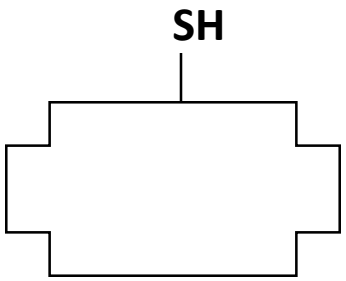


Mercury is potent allergen

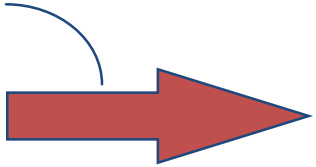
Transition metals used in dentistry bind strongly to proteins and thus are immunologically active

Metals bind to sulfur (SH) groups and change their configuration. Such cells are recognized by immune system as “foreign” and are attacked

Own cells



Hg, Ag, Au, Ni, Ti



Changed cells

S-Hg, S-Ag, S-Au, S-Ni, S-Ti

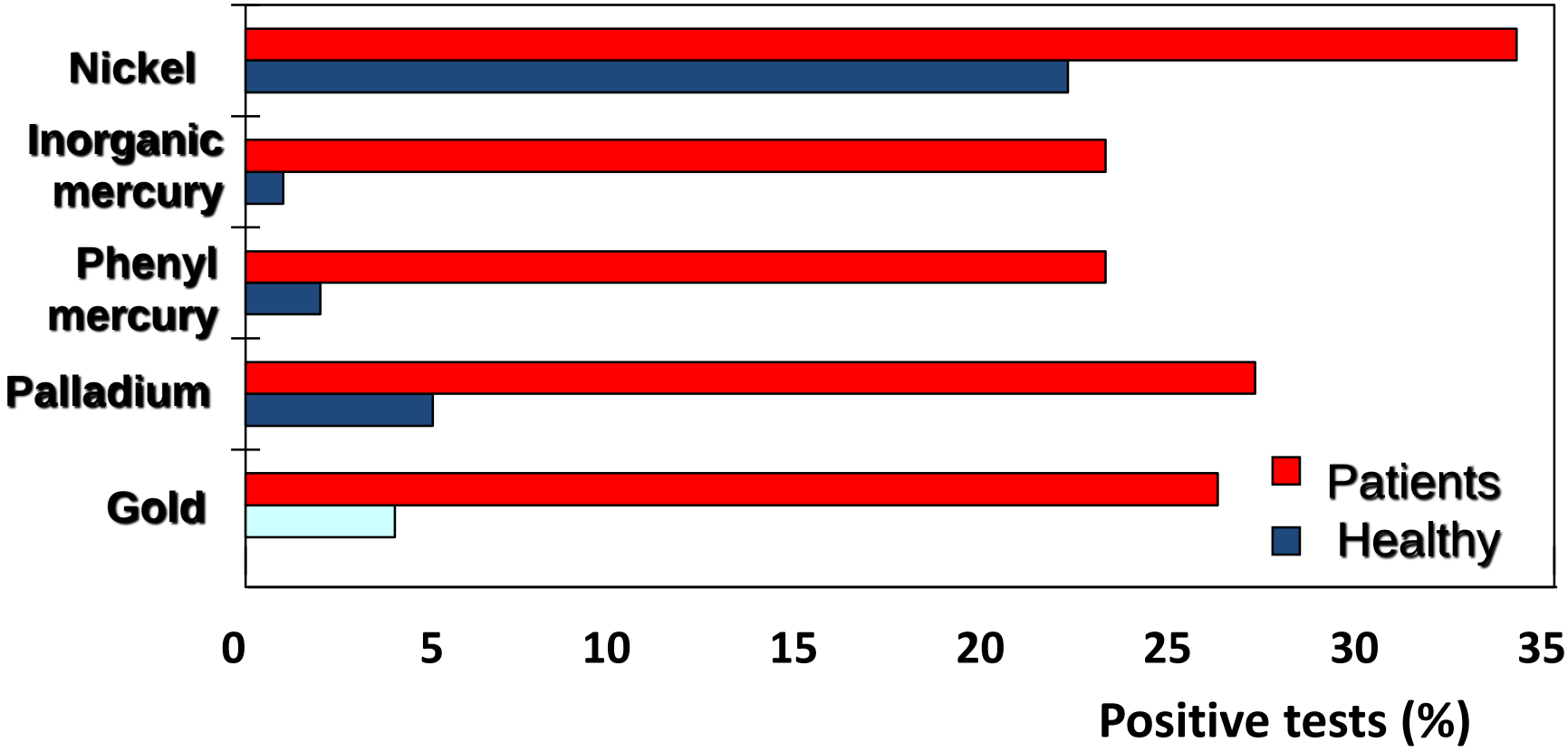
Does not stimulate the immune system

Stimulates the immune system

→ Allergy

→ Autoimmunity

Most frequent metal allergens in 3,162 patients with chronic fatigue-syndrome and in 116 healthy subjects



Metal-specific lymphocytes: biomarkers of sensitivity in man
Stejskal, V, et al. Neuroendocrinology Letters 1999; 20:289-298

Sources of Mercury in the Human

1. Vaccines

Joachim Mutter: ***Mercury and Autism: Accelerating Evidence***. Neuroendocrinol *Lett ers 2005; 26 (5): 439-446,* Institute for Environmental Medicine and Hospital Epidemiology, University Hospital Freiburg, Germany

Geier DA, Sykes LK, Geier MR: J Toxicol Environ Health B Crit Rev. 2007 Dec;10(8):575-96

A review of Thimerosal *(Merthiolate) and its ethylmercury breakdown product: specific historical considerations regarding safety and effectiveness*

Thimerosal (ethyl-mercury thiosalicilate) from vaccines, Rh-prevention (Rhogam), other medications

Autism and ASD is absent in the Amish community where children are not vaccinated. As soon as they do, they also become ill

2. **Environmental** (Environmental mercury release, special education rates and autism disorder: an ecological study of Texas. F.Palmer et al., Health and Place, Vol 12, Issue 2, June 2006, pp 203-209) "on average, for each 1000 lb of environmentally released mercury, there was….a 61% increase in the rate of autism"

1977-2002 increase in environmental Hg 3–5 fold (UNEP,2002)

790-1990 increase of environmental Hg 20 fold, in fish at least 1000 fold
(Bender 2002 Mercury Policy Project,USA)

Air: today 25 times higher Hg level then 200 years ago

3. **Mother** (2/3rds of body burden passed on to child during gestation and breastfeeding) .
70-80 % of mother's Hg burden from amalgam fillings
Stoz et al 1995: Hg in umbilical chord vein 0.2-5ng/ml
Jedrychowski et al 2005: Neurodevelopmental problems in children, when Hg in chord blood over 0.8 ng/ml

Toxin transfer during gestation and lactation period can be significantly reduced by giving the mother regular doses of the algae chlorella (BioPure/sound cracked): 12 -30 tbl. 3 times /day

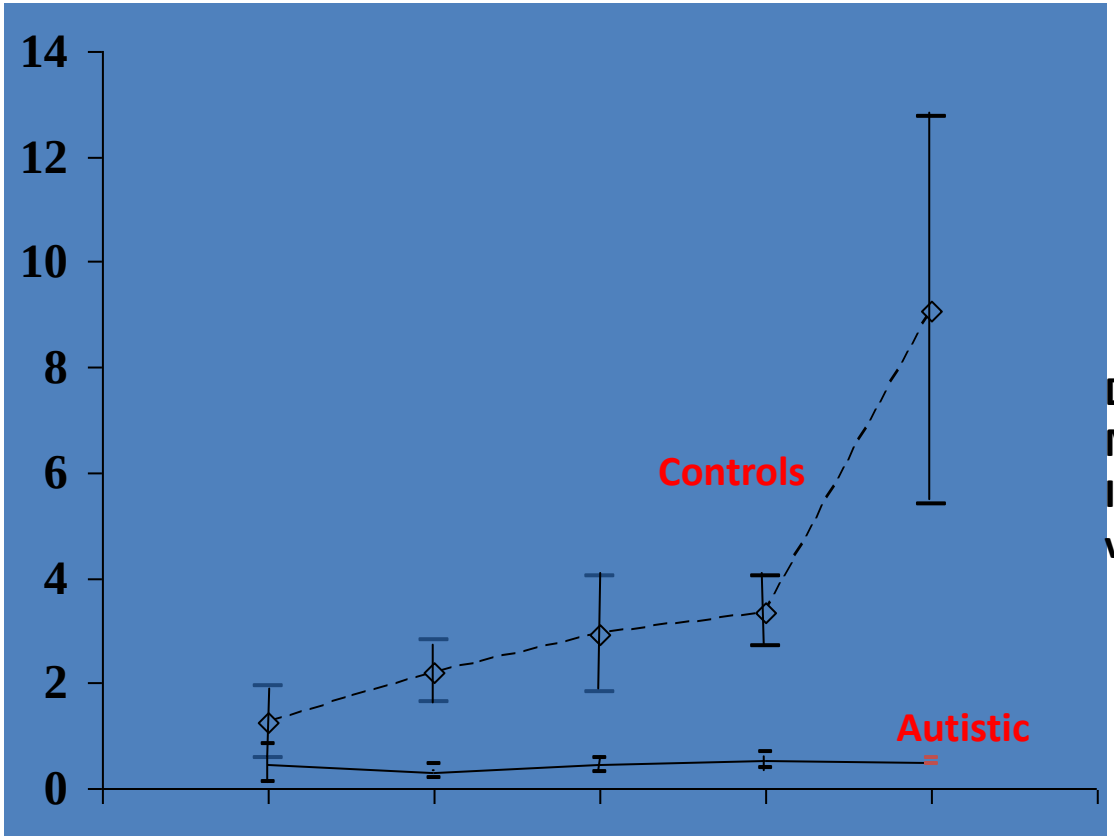
"Algenpraeparat hilfreich bei Amalgamausleitung"
D.Klinghardt Erfahrungsheilkunde 7/1999, 435-438

Maternal amalgam dental fillings as the source of mercury exposure in developing fetus and newborn. Palkovicova L, Ursinyova M, Masanova V, Yu Z, Hertz-Picciotto I. **J Expo Sci Environ Epidemiol. 2007 Sep 12.**

Dental amalgam is a mercury-based filling containing approximately 50% of metallic mercury (Hg(0)). Human placenta does not represent a real barrier to the transport of Hg(0); hence, fetal exposure occurs as a result of maternal exposure to Hg, with possible subsequent neurodevelopmental disabilities in infants. This study represents a sub-study of the international NIH-funded project "Early Childhood Development and polychlorinated biphenyls Exposure in Slovakia". The main aim of this analysis was to assess the relationship between maternal dental amalgam fillings and exposure of the developing fetus to Hg. The study subjects were mother-child pairs (N=99). Questionnaires were administered after delivery, and chemical analyses of Hg were performed in the samples of maternal and cord blood using atomic absorption spectrometry with amalgamation technique. The median values of Hg concentrations were 0.63 mug/l (range 0.14-2.9 mug/l) and 0.80 mug/l (range 0.15-2.54 mug/l) for maternal and cord blood, respectively. None of the cord blood Hg concentrations reached the level considered to be hazardous for neurodevelopmental effects in children exposed to Hg in utero (EPA reference dose for Hg of 5.8 mug/l in cord blood). A strong positive correlation between maternal and cord blood Hg levels was found (rho=0.79; P<0.001). **Levels of Hg in the cord blood were significantly associated with the number of maternal amalgam fillings (rho=0.46, P<0.001) and with the number of years since the last filling** (rho=-0.37, P<0.001); these associations remained significant after adjustment for maternal age and education. Dental amalgam fillings in girls and women of reproductive age should be used with caution, to avoid increased prenatal Hg exposure.

MERCURY BIRTH HAIR LEVELS VS. AMALGAM FILLINGS IN AUTISTIC AND CONTROL GROUPS

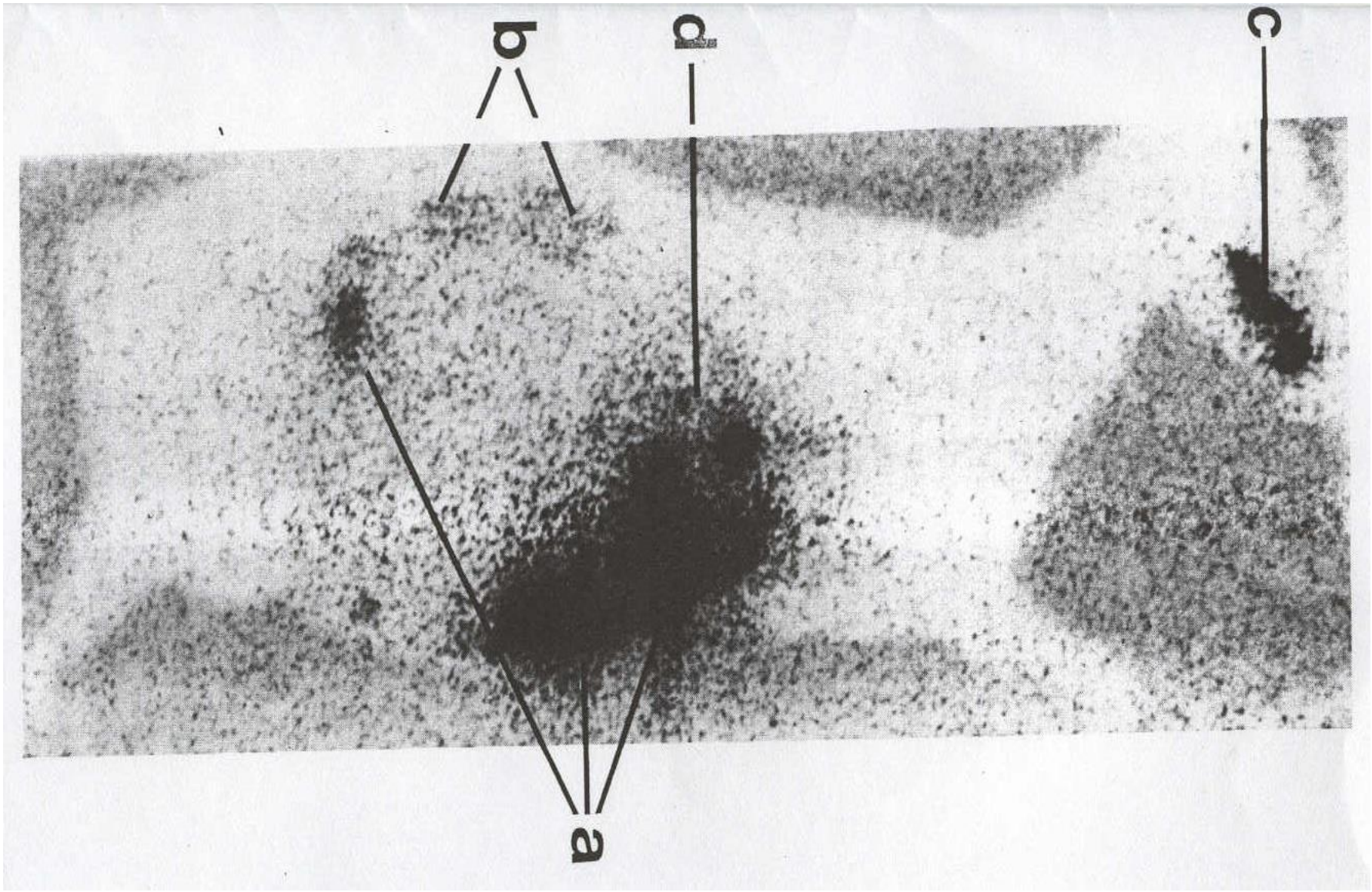
AUTISTICS SEEM LESS CAPABLE OF EXCRETING MERCURY AS INFANTS.



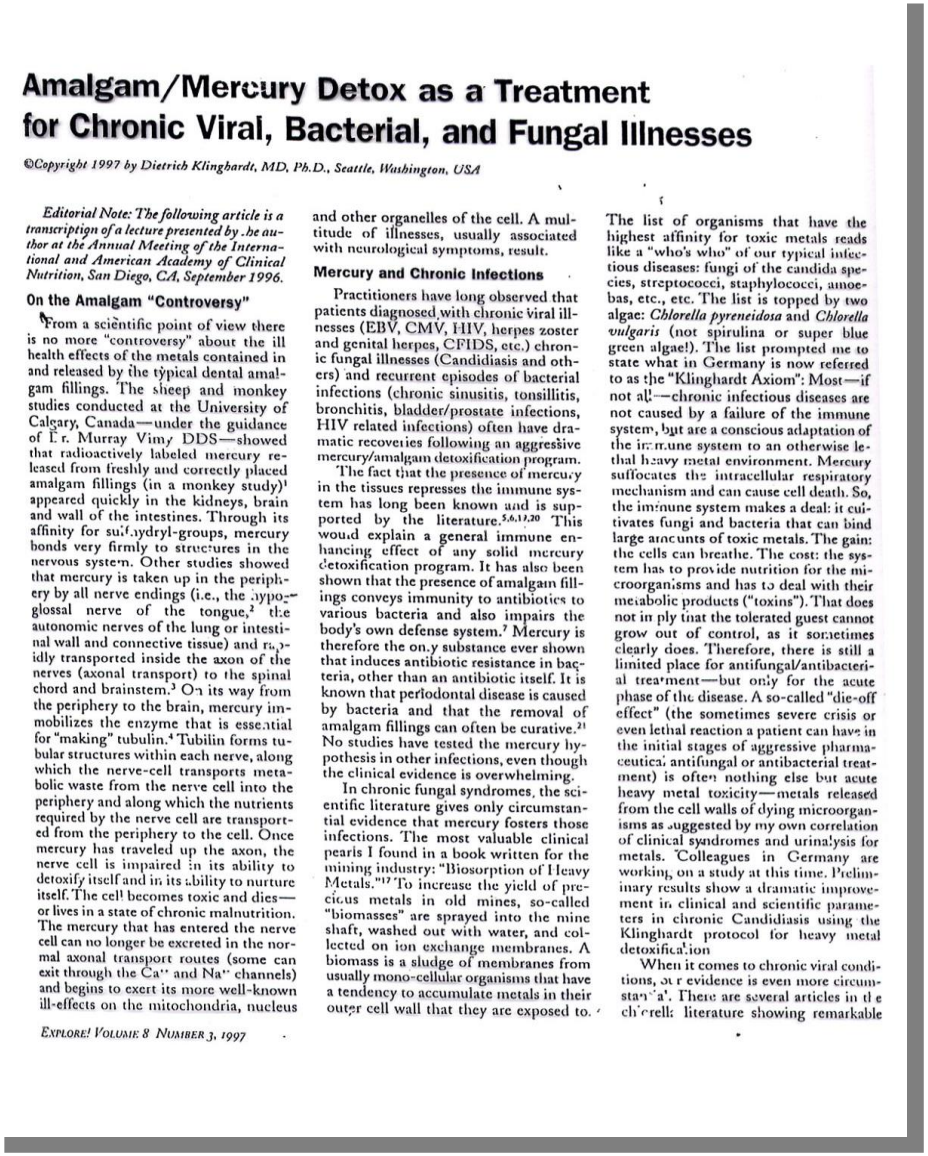
Data from A. Holmes, M. Blaxill & B. Haley, Int. J. of Toxicology v22, 2003

Number of amalgams:	0-3	4-5	6-7	8-9	≥10
Control: autistic ratio:	2.64	6.93	6.70	6.32	17.91
N:	15	22	29	30	43

Mercury compartmentalizes in a sheep after placement of several amalgam fillings (Vimy,Lorscheider et al)



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 rapidly 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] Tubilin 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 nurture 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, result.

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 represses the immune system has long been known and is supported by the literature.[5,6,17,20] This would explain a general immune enhancing effect of any solid 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.[7] 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.[21] 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."[17] To increase the yield of precious metals in old mines, so-called "biomasses" 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, amoebas, etc., etc. The list is topped by two algae: *Chlorella pyreneidosa* and *Chlorella vulgaris* (not spirulina or super blue green algae!). The list prompted me to state what in Germany is now referred 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, our evidence is even more circumstantial. There are several articles in the chlorella literature showing remarkable

- 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

Candida thrives in the Hg and lead contaminated terrain

- M.L.S.Queiroz et al, "Immunoglobulin Levels in Workers Exposed to Inorganic Mercury", Pharmacol Toxicol 74:72-75, 1994; & "Presence of Micronuclei in lymphocytes of mercury exposed workers', Immunopharmacol Immunotoxicol, 1999, 21(1):141-50; & D.C.Santos, "Immunoglubuline E in mercury exposed workers", 1997, 19(3):383-92..
- S. Yannai et al, "Transformationss of inorganic mercury by candida albicans and saccharomyces cerevisiae", Applied Envir Microbiology,1991, 7:245-247
- B.J. Shenker et al, Dept. Of Pathology,Univ. Of Penn. School of Dental Med.,"Immunotoxic effects of mercuric compounds on human lymphocytes and monocytes:Alterations in cell viability" and "Immune suppression of human T-cell activation", Immunopharmacologicol Immunotoxical, 1992, 14(3):555-77
- P.E. Bigazzi, "Autoimmunity and Heavy Metals", Lupus, 1994; 3: 449-453.
- (235) H.J.Hamre, Mercury from Dental Amalgam and Chronic Fatigue Syndrome", The CFIDS Chronicle, Fall 1994, p44-47.
- M. E. Godfrey, Candida, Dysbiosis and Amalgam. J. Adv. Med. vol 9 no 2 (1996); & Romani L, Immunity to Candida Albicans: Th1,Th2 cells and beyond. Curr Opin Microbiol 1999, 2(4):363-7

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Journal of the Neurological Sciences xx (2008) xxx–xxx

Journal of the Neurological Sciences

www.elsevier.com/locate/jns

Thimerosal exposure in infants and neurodevelopmental disorders: An assessment of computerized medical records in the Vaccine Safety Datalink

Heather A. Young[a], David A. Geier[b], Mark R. Geier[c,*]

[a] *The George Washington University School of Public Health and Health Services, Department of Epidemiology and Biostatistics, United States*
[b] *The Institute of Chronic Illnesses, Inc., Silver Spring, MD 20905, United States*
[c] *The Genetic Centers of America, United States*

Toxicological & Environmental Chemistry
Vol. 90, No. 5, September–October 2008, 997–1008

Taylor & Francis
Taylor & Francis Group

Hepatitis B triple series vaccine and developmental disability in US children aged 1–9 years

Carolyn Gallagher* and Melody Goodman

Graduate Program in Public Health, Stony Brook University Medical Center, Health Sciences Center, State University of New York at Stony Brook, Stony Brook, New York, USA

(Final version received 14 November 2007)

Avoid unnecessary vaccines

Neurotoxic Effects of Postnatal thimerosal are mouse strain dependent

Mol Psychiatry 2004 Sep.;9(9): 833-45

Horning M, Chian D, Lipkin WI., Jerome L. and Dawn - 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 recent 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

GeoEngineering in Action

SOPHIAHEALTH INSTITUTE

The synergistic toxicity of aluminum adjuvants and mercury has made the vaccines more toxic then ever. But here are other hidden sources

- Is aluminum the “new” fluoride, mercury, candida and lyme combined?
- **The “ropeworm” is loaded with aluminum, barium and increased amounts of lead, nanonized plastic and different species of mercury**
- Most patients get well, wenn Pb, Hg, Al - and the “ropeworm” are removed from the system
- Aluminium: a natural adjuvant in Leishmania transmission via sand flies? Maingon R, Khela A, Sampson C, Ward R, Walker K & Exley C (2008). Transactions of the Royal Society of Tropical Medicine and Hygiene 102, 1140-1142.

- # HPV Vaccine and Aluminum

Cautions for Amorphous Aluminum Hydroxyphosphate Sulfate Injured Patients

Do not use any therapies that increase oxygenation, due to the amorphous aluminum hydroxyphosphate sulfate in the **Gardasil vaccine.** The aluminum must be chelated out, with tissue or fecal pre and post treatment testing for aluminum.
The aluminum shifts phosphate excretion from the kidneys to intestinal excretion.
The aluminum creates intestinal blockages, abdominal pain and bloating, lethargy, anorexia, nutritional hypophosphatemia, and creates disturbed carbohydrate metabolism with hypoglycemia, hyperglycemia, and hyperinsulinemia. The lethargy occurs from phosphorus depletion, depleting the red cells of ATP (adenosine triphophate).
Aluminum salts cause acid irritation from hydrolysis, and can cause acroanesthesia in fingers and toes.
Ingredients which amalgamate with aluminum and are problematic in vaccines:

- Polysorbate 80 (a cell toxin, allergen, peroxidzer, & hemolysis promoter)
- Viral proteins (when combined with Polysorbate 80 can create thrombocytopenia)
- L-Histadine (converts to Histamine, and can accelerate allergic reactions & when combined with Polysorbate 80 can cause fatal blood clots)
- Amorphous aluminum hydroxyphosphate sulfate (see above)

The aluminum and polysorbate 80 must be removed with Chlorella and cilantro initially, and once the patient is able to defecate regularly, intravenous infusions of tapioca based sodium ascorbate, alpha lipoic acid, and glutathione administered over a 3-4 hour period should be run twice weekly until patient becomes asymptomatic. Mineral, amino acid, and vitamin supplementation should be formulated from patient lab results, and given to support recovery.
The patient should plan on 1-2 year recovery time, with ample bed rest and very controlled short periods of exercise. It is vitally important that the patient not be exposed to any new sources of aluminum, by eating ALL organically grown foods, avoiding metropolitan air sources, and pesticide exposure in public buildings and airplanes.
-

Aluminum in vaccines has never been shown to be safe

- **Trends in Immunology,** Volume 31, Issue 3, 103-109, 11 February 2010

10.1016/j.it.2009.12.009

-
- **Authors**
- **Christopher Exley , Peter Siesjö, Håkan Eriksson**
-

- The Birchall Centre, Lennard-Jones Laboratories, Keele University, Staffordshire, ST5 5BG, UK Department of Clinical Sciences, Glioma Immunotherapy Group, The Rausing Laboratory, Division of Neurosurgery, BMC D14, Lund University, SE-221 84 Lund, Sweden Department of Biomedical Laboratory Science, Health and Society, Malmö University, SE-20506 Malmö, Sweden

- **Summary**

- Aluminium adjuvants potentiate the immune response, thereby ensuring the potency and efficacy of typically sparingly available antigen. Their concomitant critical importance in mass vaccination programmes may have prompted recent intense interest in understanding how they work and their safety. Progress in these areas is stymied, however, by **a lack of accessible knowledge pertaining to the bioinorganic chemistry of aluminium adjuvants**, and, consequently, the inappropriate application and interpretation of experimental models of their mode of action. The objective herein is, therefore, to identify the many ways that aluminium chemistry contributes to the wide and versatile armoury of its adjuvants, such that future research might be guided towards a fuller understanding of their role in human vaccinations.

Can J Neurol Sci. 1989 Nov;16(4 Suppl):490-7.

New evidence for an active role of aluminum in Alzheimer's disease.

McLachlan DR, Lukiw WJ, Kruck TP.
Department of Physiology, University of Toronto, Ontario, Canada.

Abstract

Application of molecular biological techniques and sensitive elemental analysis have produced new evidence implicating aluminum as an important factor **in down regulation of neuronal protein metabolism**. Aluminum in Alzheimer's disease May act by electrostatically crosslinking proteins, particularly the methionine containing histone H1(0), and DNA. The Consequence of such crosslinking is reduced transcription of at least one neuron specific gene, the low molecular weight component of neurofilaments. In the superior temporal gyrus in Alzheimer's disease, down regulation of this gene occurs in approximately 86% of surviving neurons and, therefore, aluminum must be considered as having an active role in the pathogenesis.Epidemiological studies are reviewed that independently support the hypothesis that **environmental aluminum is a significant risk factor**. Preliminary evidence also suggests that a disorder in phosphorylation may be an important initiating factor.

PMID: 2680008 [PubMed - indexed for MEDLINE]

Journal of Inorganic Biochemistry, Volume 99, Issue 9, September 2005, Pages 1895–1898

Nanomolar aluminum induces pro-inflammatory and pro-apoptotic gene expression in human brain cells in primary culture

Walter J. Lukiw[a,*], Maire E. Percy[b], Theo P. Kruck[b]

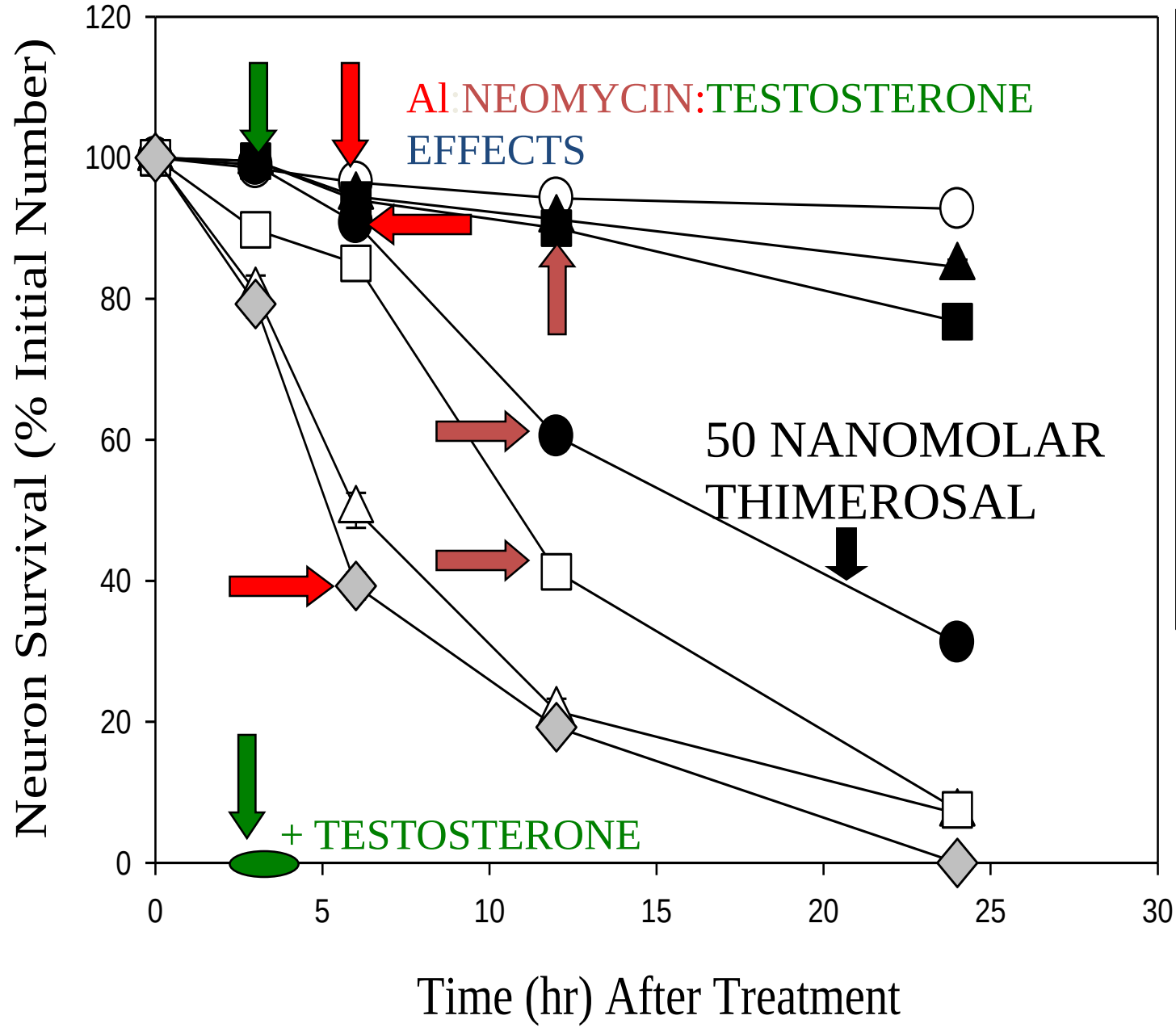
Lancet, Volume 337, Issue 8753, 1 June 1991, Pages 1304–1308
Originally published as Volume 1, Issue 8753,

Intramuscular desferrioxamine in patients with Alzheimer's disease

T.Kruck, PhD, et al

Aluminium, tau and Alzheimer’s disease. Exley C (2007) Journal of Alzheimer’s Disease 12, 313-315.

SYNERGISTIC TOXICITIES of Hg and Al



DR. MARK LOVELL

COLLABORATOR

Aluminum in vaccines is not safe

Immunologic Research

July 2013, Volume 56, Issue 2-3, pp 299-303

Adverse events following immunization with vaccines containing adjuvants

S. Cerpa-Cruz, P. Paredes-Casillas, E. Landeros Navarro, A. G. Bernard-Medina, G. Martínez-Bonilla, S. Gutiérrez-Ureña

Abstract

- A traditional infectious disease vaccine is a preparation of live attenuated, inactivated or killed pathogen that stimulates immunity. Vaccine immunologic adjuvants are compounds incorporated into vaccines to enhance immunogenicity. Adjuvants have recently been implicated in the new syndrome named ASIA autoimmune/inflammatory syndrome induced by adjuvants. The objective describes the frequencies of post-vaccination clinical syndrome induced by adjuvants. We performed a cross-sectional study; adverse event following immunization was defined as any untoward medical occurrence that follows immunization 54 days prior to the event. Data on vaccinations and other risk factors were obtained from daily epidemiologic surveillance. Descriptive statistics were done using means and standard deviation, and odds ratio adjusted for potential confounding variables was calculated with SPSS 17 software. Forty-three out of 120 patients with moderate or severe manifestations following immunization were hospitalized from 2008 to 2011. All patients fulfilled at least 2 major and 1 minor criteria suggested by Shoenfeld and Agmon–Levin for ASIA diagnosis. The most frequent clinical findings were pyrexia 68 %, arthralgias 47 %, cutaneous disorders 33 %, muscle weakness 16 % and myalgias 14 %. Three patients had diagnosis of Guillain–Barre syndrome, one patient had Adult-Still's disease 3 days after vaccination. A total of 76 % of the events occurred in the first 3 days post-vaccination. Two patients with previous autoimmune disease showed severe adverse reactions with the reactivation of their illness. Minor local reactions were present in 49 % of patients. **Vaccines containing adjuvants may be associated with an increased risk of autoimmune/inflammatory adverse events following immunization.**

- Aluminium: A potential pro-oxidant in sunscreens/sunblocks? Nicholson S & Exley C (2007) Free Radical Biology and Medicine 43, 1216-1217.

Let them be dumb and tired!

J Inorg Biochem. 2009 Nov;103(11):1571-8. doi: 10.1016/j.jinorgbio.2009.08.005.
Epub 2009 Aug 20.

Long-term persistence of vaccine-derived aluminum hydroxide is associated with chronic cognitive dysfunction. Couette M, Boisse MF, Maison P, Brugieres P, Cesaro P, Chevalier X, Gherardi RK, Bachoud-Levi AC, Authier FJ.
INSERM, Unite U955, Team 1, Creteil F-94010, France.

Abstract
Macrophagic myofasciitis (MMF) is an emerging condition, characterized by specific muscle lesions assessing long-term persistence of aluminum hydroxide within macrophages at the site of previous immunization. Affected patients mainly complain of arthromyalgias, chronic fatigue, and cognitive difficulties. We designed a comprehensive battery of neuropsychological tests to prospectively delineate MMF-associated cognitive dysfunction (MACD). Compared to control patients with arthritis and chronic pain, MMF patients had pronounced and specific cognitive impairment. MACD mainly affected (i) both visual and verbal memory; (ii) executive functions, including attention, working memory, and planning; and (iii) left ear extinction at dichotic listening test. Cognitive deficits did not correlate with pain, fatigue, depression, or disease duration. Pathophysiological mechanisms underlying MACD remain to be determined. In conclusion, long-term persistence of vaccine-derived aluminum hydroxide within the body assessed by MMF is associated with cognitive dysfunction, not solely due to chronic pain, fatigue and depression.

Exley C, Swarbrick L, Gheradi R & Authier J-F (2009) A role for the body burden of aluminium in vaccine-associated macrophagic myofasciitis and chronic fatigue syndrome. Medical Hypotheses 72, 135-139.

Journal of Neuroimmune Pharmacology
December 2008, Volume 3, Issue 4, pp 286-295

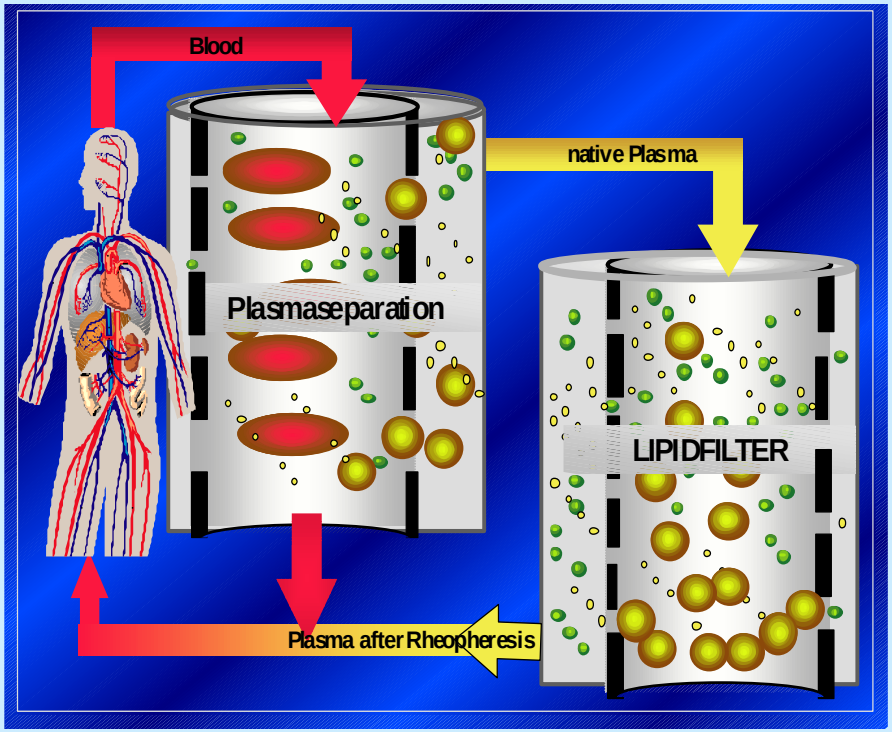
Manufactured Aluminum Oxide Nanoparticles Decrease Expression of Tight Junction Proteins in Brain Vasculature

Lei Chen, Robert A. Yokel, Bernhard Hennig, Michal Toborek

Abstract

- Manufactured nanoparticles of aluminum oxide (nano-alumina) have been widely used in the environment; however, their potential toxicity provides a growing concern for human health. The present study focuses on the hypothesis that **nano-alumina can affect the blood-brain barrier and induce endothelial toxicity.** In the first series of experiments, human brain microvascular endothelial cells (HBMEC) were exposed to alumina and control nanoparticles in dose- and time-responsive manners. Treatment with nano-alumina markedly reduced HBMEC viability, altered mitochondrial potential, increased cellular oxidation, and decreased tight junction protein expression as compared to control nanoparticles. Alterations of tight junction protein levels were prevented by cellular enrichment with glutathione. In the second series of experiments, rats were infused with nano-alumina at the dose of 29 mg/kg and the brains were stained for expression of tight junction proteins. Treatment with nano-alumina resulted in a marked fragmentation and disruption of integrity of claudin-5 and occludin. These results indicate that cerebral vasculature can be affected by nano-alumina. In addition, our data indicate that **alterations of mitochondrial functions** may be the underlying mechanism of **nano-alumina toxicity**.

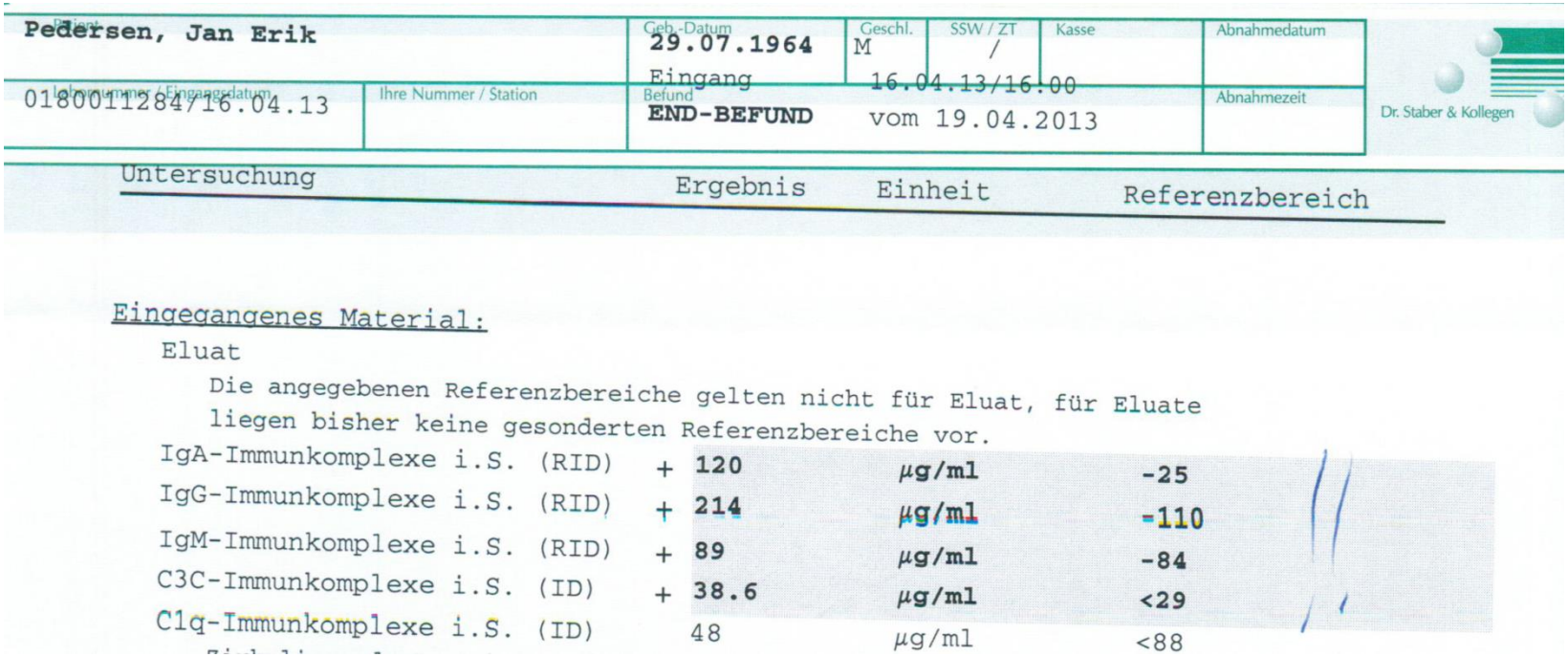
Principle of Membrane Differential Filtration (MDF)



Modern technology to perform MDF

Fig. 1

What does Apheresis remove?



Patient: **Pedersen, Jan Erik**

Geb.-Datum: **29.07.1964** | Geschl.: M | SSW / ZT: / | Kasse | Abnahmedatum

Labornummer / Eingangsdatum: 0180011284/16.04.13 | Ihre Nummer / Station | Befund: **END-BEFUND** | Eingang: 16.04.13/16:00 vom 19.04.2013 | Abnahmezeit

Dr. Staber & Kollegen

Untersuchung		Ergebnis	Einheit	Referenzbereich
Eingegangenes Material:				
Eluat				
Die angegebenen Referenzbereiche gelten nicht für Eluat, für Eluate liegen bisher keine gesonderten Referenzbereiche vor.				
IgA-Immunkomplexe i.S. (RID)	+	**120**	**µg/ml**	**-25**
IgG-Immunkomplexe i.S. (RID)	+	**214**	**µg/ml**	**-110**
IgM-Immunkomplexe i.S. (RID)	+	**89**	**µg/ml**	**-84**
C3C-Immunkomplexe i.S. (ID)	+	**38.6**	**µg/ml**	**<29**
C1q-Immunkomplexe i.S. (ID)		48	µg/ml	<88

Where is the source of this aluminum?

Multielementanalyse (TOX)

Untersuchungsmaterial: **Eluat**

Nur orientierend sind nachfolgend die Referenzwerte für Serum in µg/l angegeben:
Arsen < 2,1; Blei <0.8; Cadmium < 0,4; Cobalt < 0,4; Gold < 0.2; Indium < 0.2;
Molybdän 0,3-1,2; Nickel < 3; Palladium < 0,2; Platin < 0.2; Quecksilber < 2; Silber
< 0,3; Wismut < 0,2; Thallium < 0,3; Zinn < 2; Zink 700-1500.

Aluminium	120	µg/l
Arsen		

Nach neueren Erkenntnissen ist eine Bestimmung des Arsens im eingesandten Röhrchen aufgrund von Kontaminationen aus dem Glas nicht sinnvoll.

Blei	0.9	µg/l
Cadmium	0.2	µg/l

Days after Toxic Fallout from GeoEngineering

Patient	Geburtsdatum	Tagesnummer	IMD Berlin-Potsdam MVZ GbR Nicolaistraße 22, 12247 Berlin (Steglitz) Telefon: +49 30 77001-220, Fax: +49 30 77001-236
Klinghardt Dr., Dietrich	**14.10.1950**	**0326262368**	

Eingang	Ausgang	Versicherung	Kennz. OI/II/III
11.04.2013	**18.04.2013**	**IGeL**	

Untersuchung / Material : **Lymphozytentransformationstest** (Heparinblut)

	SI
Aluminium	16,9

Hinweise zur Untersuchungsmethode:

Die angegebenen Werte neben den Balken sind die Stimulationsindizes (SI) für das jeweilige Allergen (Mittelwert). Dieser ergibt sich aus dem Mittelwert von 3 isoliert untersuchten Stimulationsansätzen. Dieser Wert ist zusätzlich als Balken dargestellt. Der Stimulationsindex ist der Quotient aus der allergeninduzierten- und der unstimulierten Thymidineinbaurate (Leerwert in cpm).
Ein SI > 3 bedeutet eine mehr als dreifache Aktivierung im Vergleich zum Leerwert und beweist die Existenz von zirkulierenden allergenspezifischen T-Zellen im Patientenblut (positives Ergebnis, zelluläre Sensibilisierung).
Ein SI < 2 gilt als sicher negativ. Ergebisse zwischen 2 und 3 sind als grenzwertig anzusehen (schwache bzw. fragliche Sensibilisierung), die ggf. kontrolliert werden sollten.

The enemy in us

Bestimmung	Resultat		Referenzbereich
Multielementanalyse (TOX)			
Untersuchungsmaterial:	**Eluat**		
Nur orientierend sind nachfolgend die Referenzwerte für Serum in µg/l angegeben: Arsen < 2,1; Blei <0.8; Cadmium < 0,4; Cobalt < 0,4; Gold < 0.2; Indium < 0.2; Molybdän 0,3-1,2; Nickel < 3; Palladium < 0,2; Platin < 0.2; Quecksilber < 2; Silber < 0,3; Wismut < 0,2; Thallium < 0,3; Zinn < 2; Zink 700-1500.			
Aluminium	33.4	µg/l	
Arsen	1.3	µg/l	
Blei	< 0.2	µg/l	
Cadmium	< 0.2	µg/l	
Cobalt	4.0	µg/l	
Gold	< 0.2	µg/l	
Indium	< 0.2	µg/l	

SOPHIAHEALTH INSTITUTE

Bestimmung	Resultat		Referenzbereich
Untersuchungsmaterial:	Eluat		
Aluminium	90	µg/l	
Barium	14.0	µg/l	
Strontium	90.8	µg/l	

Grass Analysis after Chemtrail-Fallout

Ch emi s c h -Te c h n i s c h e s La b o r a t o r i um Lu e r s KG
E-Mail labor@luers.de www.luers.de
Amtsgericht Bremen HRA 21432 HB Datum 25.10.2012

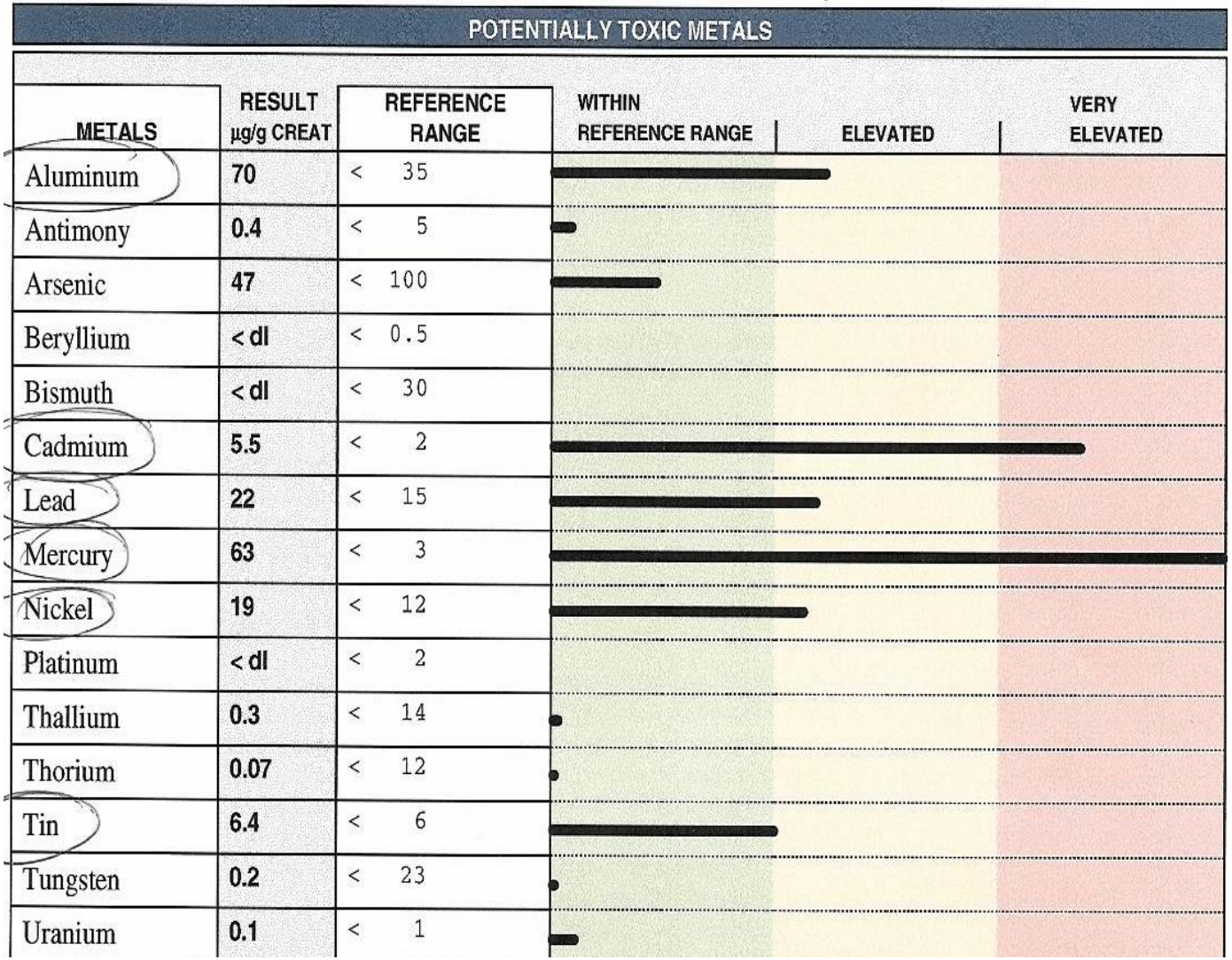
Probenart : **Feststoff/ Boden** **Pflanzen/Boden**

•	Aluminium mg/kg TS	soil:	25.150	grass: 22.900
•	Barium mg/kg TS		140	155
•	Blei (Lead) mg/kg TS		29	16
•	Arsen mg/kg TS		4,8	2,8
•	Cadmium mg/kg TS		0,28	0,18
•	Nickel mg/kg TS		21	20
•	Palladium mg/kg TS		< 5	< 5

DIAGNOSIS of metal toxicity

- History and symptoms
- Neurology (hyperreflexia, clonus, Babinski, burning, numbness, etc.)
- Whole blood
- Red cells
- White cells
- Hair
- Stool
- Serum
- Urine
- Urine porphyrins
- ART
- EDS
- Apheresis

Challenged Urine Panel



POTENTIALLY TOXIC METALS

METALS	RESULT µg/g CREAT	REFERENCE RANGE
Aluminum	70	< 35
Antimony	0.4	< 5
Arsenic	47	< 100
Beryllium	< dl	< 0.5
Bismuth	< dl	< 30
Cadmium	5.5	< 2
Lead	22	< 15
Mercury	63	< 3
Nickel	19	< 12
Platinum	< dl	< 2
Thallium	0.3	< 14
Thorium	0.07	< 12
Tin	6.4	< 6
Tungsten	0.2	< 23
Uranium	0.1	< 1

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Email : contact@labbio.net

07/11/2007

Urinary porphyrins

HPLC-UV+Fluorescence

	nmol/l	nmol/gCr	%	reference nmol/gCr	Interpretation
Uroporphyrins I &III (UP)	37	33	7,9%	8-20	Increased rate
Heptacarboxy porphyrin (7cxP)	4,2	3,8	0,9%	2,5-4,5	Average Rate
Hexacarboxy porphyrin (6cxP)	1,3	1,1	0,3%	0,5-1,5	Average Rate
Pentacarboxy porphyrin (5cxP)	5,8	5,2	1,2%	2-4	Slighltly increased rate
Precoproporphyrin (PrCP)	18,0	16,0	3,8%	5-9	Increased rate
Coproporphyrins I & III (CP)	405	362	89,3%	100-200	Increased rate

PrCP/UP	PrecoP/Uro ratio	0,48		0,2-0,5
(5cxP+PrCP)/(UP+7cxP)	ratio	0,6		0,3-0,6
PrCP/5cxP		3,1		1,5-3
PrCP/CP	PrecoP/COP ratio	4,4	%	2-6
CP / UP	copro/uro ratio	10,90		5-9

Interpretation

Urinary Porphyrin Profile suggestive a moderate mercury toxic effect on bodily physiology high in coproporphyrin

Urinary porphyrin profile ia a powerful biochemical tool in diagnosis of intoxication associating sensitivity, specificity and quantificity

* sensitivity- because heme biosynthesis is highly sensitive to inhibition by many inorganic toxicants such as Mercury, Lead, Arsenic, Aluminium as well as organic agents: chlorinated benzene, biphenyls (PCB), dioxins (TCDD) and also alcohol.

* Specificity-because nearly each toxics generates a specific urinary porphyrine excretion pattern for example: Biphenyls, Dioxins, Aluminium inhibit an early enzyme on porphyrin biosynthesis pathway Uro-Decarboxylase, Mercury inhibits Copro-oxydase and L

*Quantificity or quantitative relationship between increase of specific porphyrins species and toxic or heavy metal body burden with a high degree of correlation designating it as a reliable biomarker for chelation therapy

urinary creatinine 1120 mg/l

$Ca\text{-}Na_2\text{-}EDTA$ (caveat: this is not sodium EDTA!!!)
Ca-EDTA slow push/fast drip
50 mg/kg, not to exceed 3 gm
$T^{1/2}$ about 30-45 minutes
6 hr. urine collection

DMPS challenge
IV: 3-5 mg/kg (250 mg max), **slow** push (5-10 min.)
Oral: 10 mg /kg BW (5 mg/kg children), empty stomach(empty bladder).Withhold food about 2 hrs.Encourage ~ 0.5L fluid over next few hrs. Collect all urine for 6 hrs.

DMSA challenge (oral):20-30 mg DMSA/kg BW as oral bolus on empty stomach (≤ 2 gms).Withhold food about 2 hrs. Encourage ~ 0.5L fluid over next few hrs. Collect all urine for 6 hrs.
J Nutr Envir Med (1998) 8:219-231

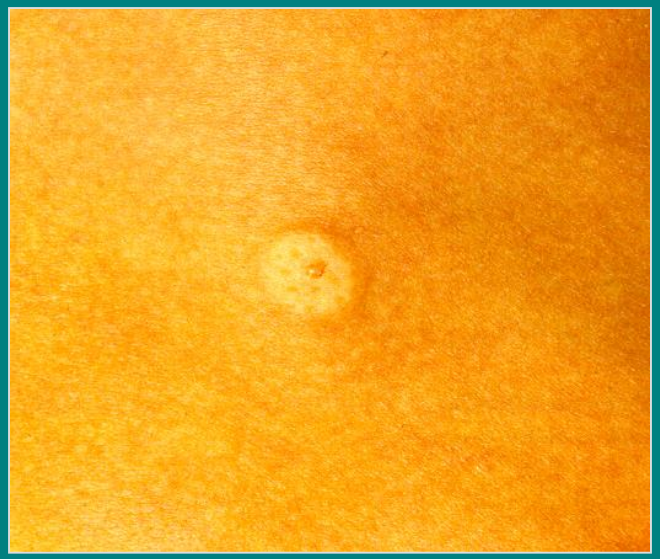
D-Pencillamine protocol- 500mg three times per day 2 days per week (R.Jaffe PhD)

Desferal: reconstitute vial with 10 ml distilled water. Inject half segmentally subcutaneously around the abdomen. The other half 2-3 days later. Keep refridgerated

Other intravenous options

- IV Vitamin C: 37-50 grams in 500 ml distilled water with 10 ml Ca gluconate
- Glutathione: 600-4000 mg 1-3x weekly, IV push (always include i.m or i.v Magnesium once-twice weekly)
- Alpha-lipoic acid: 600 mg in normal saline (250 cc) over 1 hr
- Phospholipids (Lipostabil): 2 ampoules diluted with client's blood (50:50) given slow IV over 3 minutes
- Konventionelles NaEDTA Protokoll (ACAM)
- Zinc DTPA: 1 Ampulle 1 mal/Woche i.v.

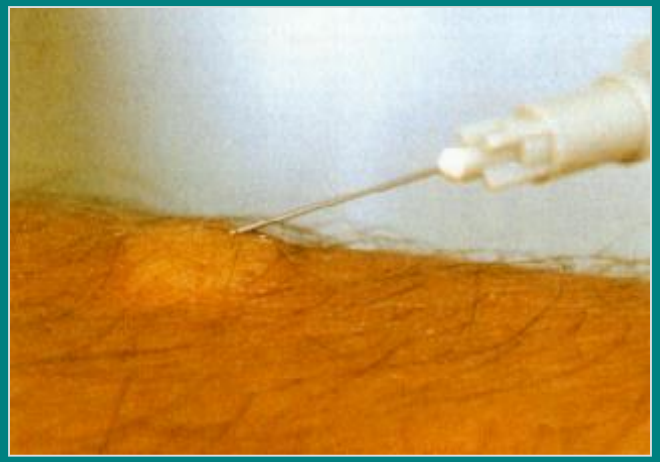
Die Quaddel (*intracutan !*)

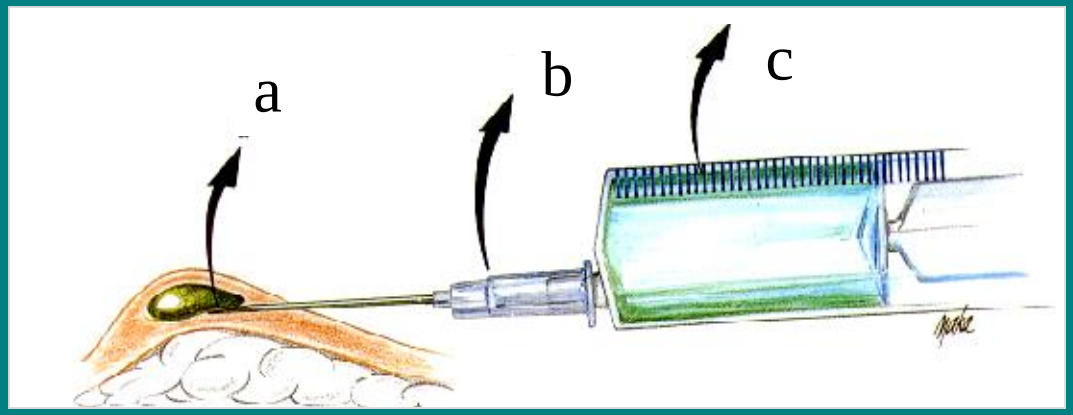


a Kanülenschliff nach oben

b Spritzenkonus nach unten

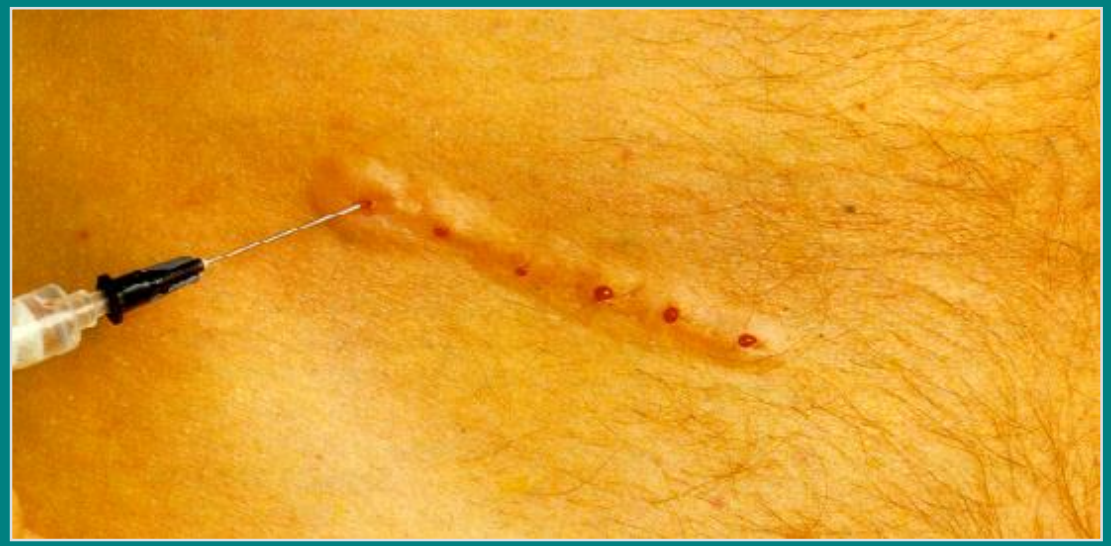
c Spritzenskala nach oben



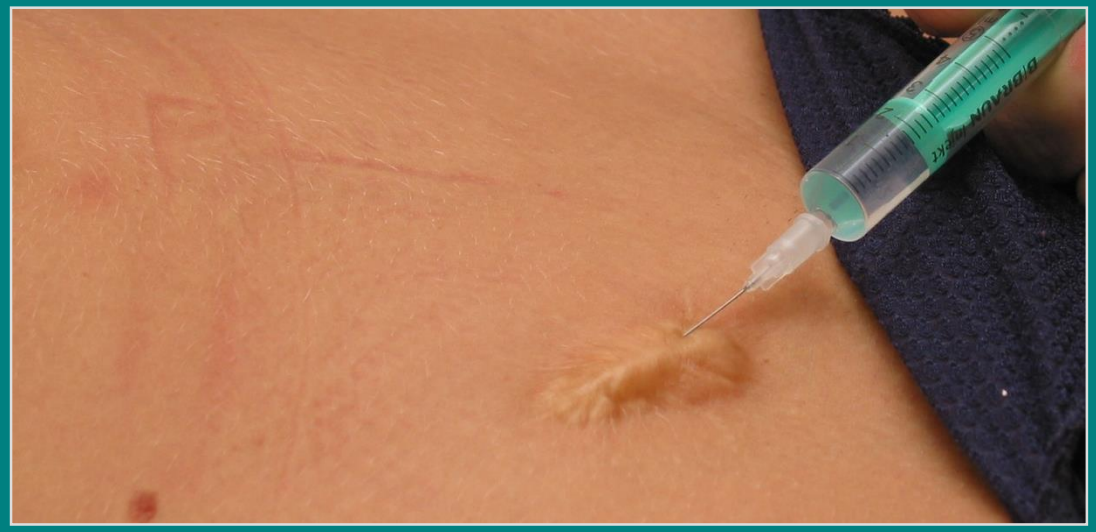


Segment-Therapie

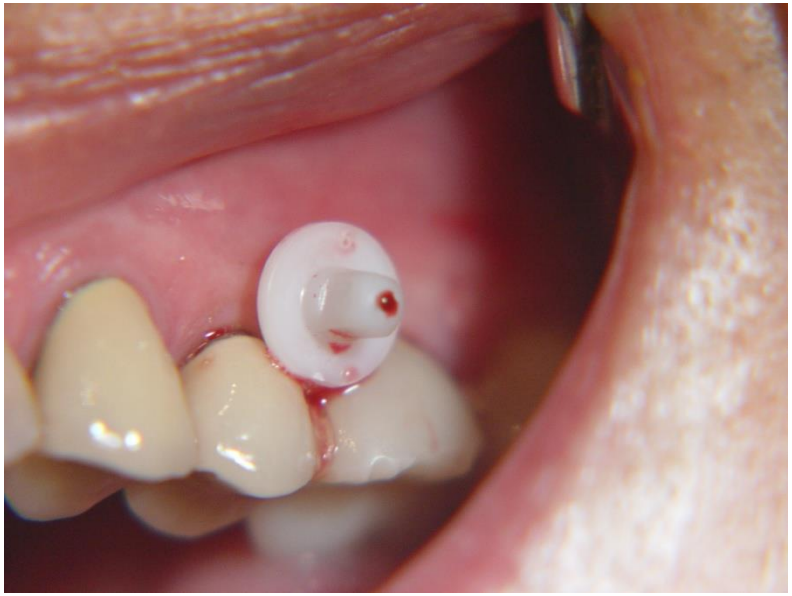
Injektion in Narben

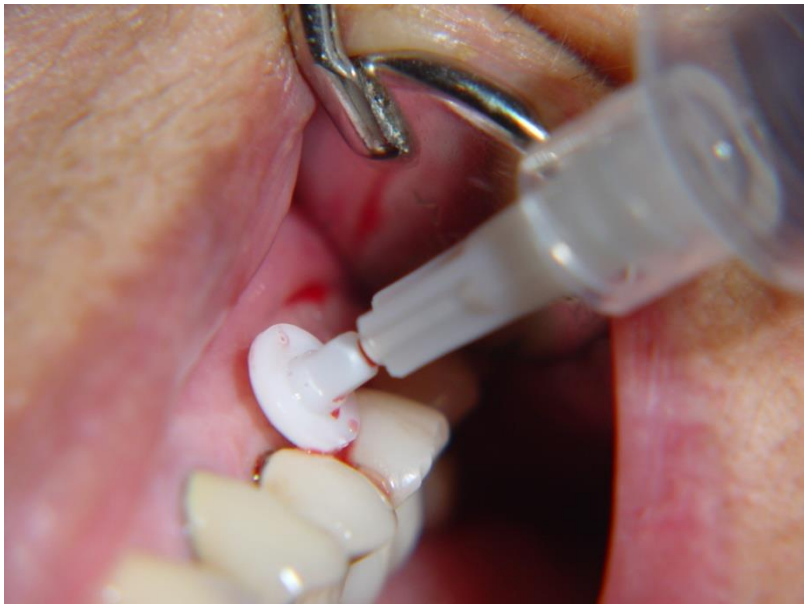


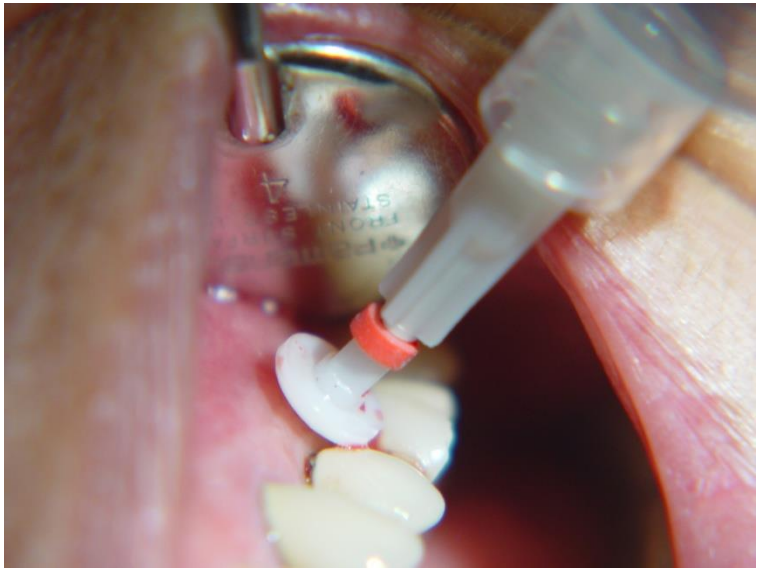
Quaddelung



Infiltration







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 woman 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 **psychological intervention** (APN/MFT)	
1/24/1998	2100	2700
2/3/1998		**2900**
4/3/1998	1500	930
4/18/1998		370

J Appl Toxicol. 2010 Mar 12

Detoxification and antioxidant effects of curcumin in rats experimentally exposed to mercury. Agarwal R, Goel SK, Behari JR.

Curcumin, a safe nutritional component and a highly promising natural antioxidant with a wide spectrum of biological functions, has been examined in several metal toxicity studies, but its role in protection against mercury toxicity has not been investigated. Therefore, the detoxification and antioxidant effects of curcumin were examined to determine its prophylactic/therapeutic role in rats experimentally exposed to mercury (in the from of mercuric chloride-HgCl(2), 12 micromol kg(-1) b.w. single intraperitoneal injection). Curcumin treatment (80 mg kg(-1) b.w. daily for 3 days, orally was found to have a protective effect on mercury-induced oxidative stress parameters, namely, lipid peroxidation and glutathione levels and superoxide dismutase, glutathione peroxidase and catalase activities in the liver, kidney and brain. Curcumin treatment was also effective for reversing mercury induced serum biochemical changes, which are the markers of liver and kidney injury. Mercury concentration in the tissues was also decreased by the pre/post-treatment with curcumin. However, histopathological alterations in the liver and kidney were not reversed by curcumin treatment. Mercury exposure resulted in the induction of metallothionein (MT) mRNA expressions in the liver and kidney. Metallothionein mRNA expression levels were found to decrease after the pre-treatment with curcumin, whereas posttreatment with curcumin further increased MT mRNA expression levels. Our findings suggest that curcumin pretreatment has a protective effect and that curcumin can be used as a therapeutic agent in mercury intoxication.

The study indicates that curcumin, an effective antioxidant, may have a **protective effect** through its routine dietary intake **against mercury** exposure (we use specially formulated CurcuSyn from Biopure)

Chlorella and Metal Binding

Cadmium

Hagino et al.: Effect of chlorella on fecal and urinary cadmium excretion in Itai-itai. *Jap. J. Hyg.* 30: 77, 4/1975

Nagano, T./Suketa, Y., et al.: Absorption and excretion of chlorella ellipsoidea cadmium-binding protein and inorganic cadmium in rats. *Jpn. J. Hyg.*, 38: 741-747, 1983

Carr, H.P., Carino, F.A., et al.: Characterization of the cadmium-binding capacity of chlorella vulgaris. *Bull. Environ. Contam. Toxicol.*, 60: 433-440, 1998

Uranium

Horikoshi, T./ Nakajima, A., et al.: Uptake of uranium by various cell fractions of chlorella vulgaris. *Radioisotopes* 28: 485-488, 1979

Nakajima, A; Horikoshi, T; Sakagushi, T.: Recovery of uranium by immobilised micro-organisms. *Evr. J. Appl. Microbiol. Biotech,* 16: 88-91, 1982.

Lead

Protective effects of chlorella vulgaris in lead exposed mice infected with Listeria monocytogenes M.Queiroz et al International Immunopharmacology 3 (2003) 889-900

Mercury

Shieh, Y.J.; Barger, J: Uptake of mercury by chlorella and its effect on potassium regulation. *Planta,* 109: 49-60, 1973

Klinghardt,D. :Algenpraeparat hilfreich bei der Amalgamausleitung
Erfahrungsheilkunde Band 48, Heft 7, Juli 1999

D.Klinghardt and J. Mercola: Mercury toxicity and systemic elimination agents D.Klinghardt and J. Mercola, J of Nutritional and environmental Medicine (2001) 11, 53-62

Chlorella Vulgaris

- The Influence of Parachlorella beyerinckii CK-5 (BioPureChlorenergy) on the absorption and excretion of methylmercury (MeHg) in mice. T.Uchikawa, A.Yasutake et al. *J. of Toxicological Sciences*, Vol35,No1.101-105.2010
- Preventive effects of Chlorella (BioPureChlorenergy) on cognitive decline in age-dependent dementia model of mice

  Y.Nakashima, I.Ohsawa et al. *Neuroscience Letters* 464 (2009)193-198
- Chlorella vulgaris culture supernatant (CVE) reduces psychological stress-induced apoptosis in thymocytes in mice
- T.Hasegawa, K.Noda et al. *International Journal of Immunopharmacology* 22(2000) 877-887

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. 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.

PMID: 12781705 [PubMed - in process]

Chlorella in pregnant and breastfeeding mothers

- Effect of chlorella pyreneidosa on fecal excretion and liver accumulastion of polychlorinated dibenzo-p-dioxin in mice Chemosphere 2005;59 297-304

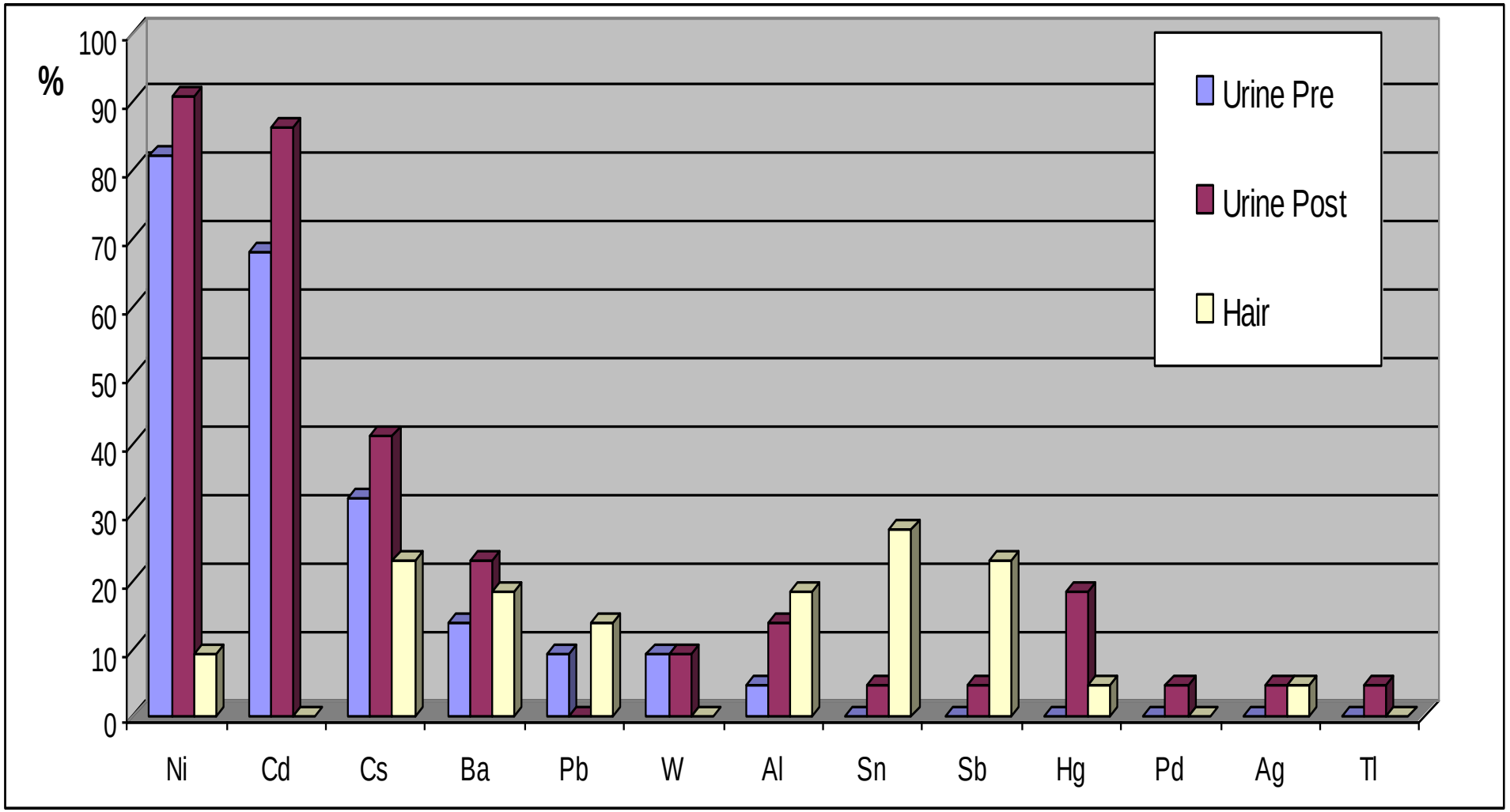
- Maternal-fetal distribution and transfer of dioxins in pregnant women in Japan, and attempts to reduce maternal transfer with Chlorella (Chlorella pyrenoidosa) supplements

  S.Nakano et al Chemosphere, April 2005

- Chlorella Pyreneidosa supplementation decreases Dioxin and increases Immunoglobulin A concentrations in breast milk

  Shiro Nakano et al J Med Food 10 (1) 2007, 134-142).

Toxic metal ions in urine and hair after provocation with a 10 drops BioPure ***energized cilantro*** tincture tid for 6 weeks



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]

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[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

ISSN 1359-0847 print/ISSN 1364-6907 online/01/010053-10 © Taylor & Francis Ltd
DOI: 10.1080/13590840020030267

Diagnosis: Aluminum shows up best in whole blood metal tests (has to be added on!). Very best test: aluminum in apheresis eluat

Aluminum Detox:

Biological: Omura's Cilantro Soup

Boil water. Put handful of finely chopped cilantro into it and let steep for 10 minutes to eliminate parasites (parsley often carries lungworm infected tiny snails). Then add 1 tbsp organic fermented soy miso. Drink this every night – for life. Omura's animal study: in 39 days ½ body burden of lead and aluminum gone, even though the test animals were further poisoned.

Omura, Y. et al: Acupunct.Electrother.Res.1995; 20(3-4): 195-229

Medical: 1 vial reconstituted Desferal used i.m or sub-cutaneously in Neural therapy fashion once to twice per week

Horsetail tea (Silica), ZeoCLear and MicroSilica as binders

EDTA may have a moderate effect, DMSA and DMPS have little. Keeping intracellular glutathione high has been shown to protect

Aluminum detox

Foglio E, Buffoli B, Exley C, Rezzani R and Rodella LF (2012) Regular consumption of a **silicic acid-rich** water prevents aluminium-induced alterations of nitrergic neurons in mouse brain: histochemical and immunohistochemical studies. Histology and Histopathology 27, 1055-1066.

Exley C (2012) Reflections upon and recent insight into the mechanism of formation of hydroxy**aluminosilicates** and the therapeutic potential of silicic acid. Coordination Chemistry Reviews 256, 82-88.

Exley C (2008) Comment on "Avoidance of aluminium toxicity in freshwater snails involves intracellular **silicon-aluminium biointeraction**". Environmental Science & Technology 42, 5374.

Exley C (2007) Organosilicon therapy in Alzheimer's disease? Journal of Alzheimer's Disease 11, 301-302.

Effective detox strategies to reduce parasite burden (pry metals out of parasite and yeast cell wall)

- Binders: chlorella, charcoal, chitosan, zeolite (use ZeoCLear from BioPureUS.com), clay, MicroSilica. Take lots between meals

- Optimize phase 1, 2 and 3 liver detox and genetic glitches (MTHFR, SOD)

- Mobilizers: homeopathic SH agents: Homeo K Merc, liposomal glutathione, cilantro, curcumin, matrix metals spray

- Traditional detox agents: oral DMPS or DMSA

ElectroStress leads to Devolution: supports lower life and increases the growth of parasites, mold, Lyme, etc

Most man-made electromagnetic fields hurt life and evolution of higher species

MicroWave from cell phone broadcasting

- causes inflammation,
- Disables detoxification (blocks genetic expression of most detox genes).
- Causes oxidative stress.
- Disables immune system

Microwave- Electromagnetic Radiation




Unexposed **Exposed**

Same type of watercress grown grown inside a classroom with the same sun exposure – only the right one placed near the wireless router

High Frequency 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 processes.

Brain proteome response following whole body exposure of mice to mobile phone or wireless DECT base radiation

Electromagnetic Biology and Medicine; Posted online on January 20, 2012.
(doi:10.3109/15368378.2011.631068 (1–25) Adamantia F. Fragopoulou, Athina Samara, Marianna H. Antonelou, Anta Xanthopoulou, Aggeliki Papadopoulou, Konstantinos Vougas, Eugenia Koutsogiannopoulou, Ema Anastasiadou, Dimitrios J. Stravopodis, George Th. Tsangaris, Lukas H. Margaritis
Department of Cell Biology and Biophysics, Athens University,

Abstract:
The objective of this study was to investigate the effects of two sources of electromagnetic fields (EMFs) on the proteome of cerebellum, hippocampus, and frontal lobe in Balb/c mice following long-term whole body irradiation. Three equally divided groups of animals (6 animals/group) were used; the **first** group was exposed to a **typical mobile phone**, at a SAR level range of 0.17–0.37 W/kg for 3 h daily for 8 months, the **second** group was exposed to a **wireless DECT base** (Digital Enhanced Cordless Telecommunications/Telephone) at a SAR level range of 0.012–0.028 W/kg for 8 h/day also for 8 months and the t**hird** group comprised the **sham**-exposed animals. Comparative proteomics analysis revealed that long-term irradiation from **both EMF sources** altered significantly ($p < 0.05$) the **expression of 143 proteins** in total (as low as 0.003 fold downregulation **up to 114 fold overexpression**). Several neural function related proteins (i.e., Glial Fibrillary Acidic Protein (GFAP), Alpha synuclein, Glia Maturation Factor beta (GMF), and apolipoprotein E (apoE)), heat shock proteins, and cytoskeletal proteins (i.e., Neurofilaments and tropomodulin) are included in this list as well as proteins of the brain metabolism (i.e., Aspartate aminotransferase, Glutamate dehydrogenase) to nearly all brain regions studied. Western blot analysis on selected proteins confirmed the proteomics data. The observed **protein expression changes may be related** to **brain plasticity** alterations, indicative of **oxidative stress in the nervous system** or involved in **apoptosis** and might potentially explain human health hazards reported so far, such as **headaches, sleep disturbance, fatigue, memory deficits, and brain tumor long-term induction** under similar exposure conditions.

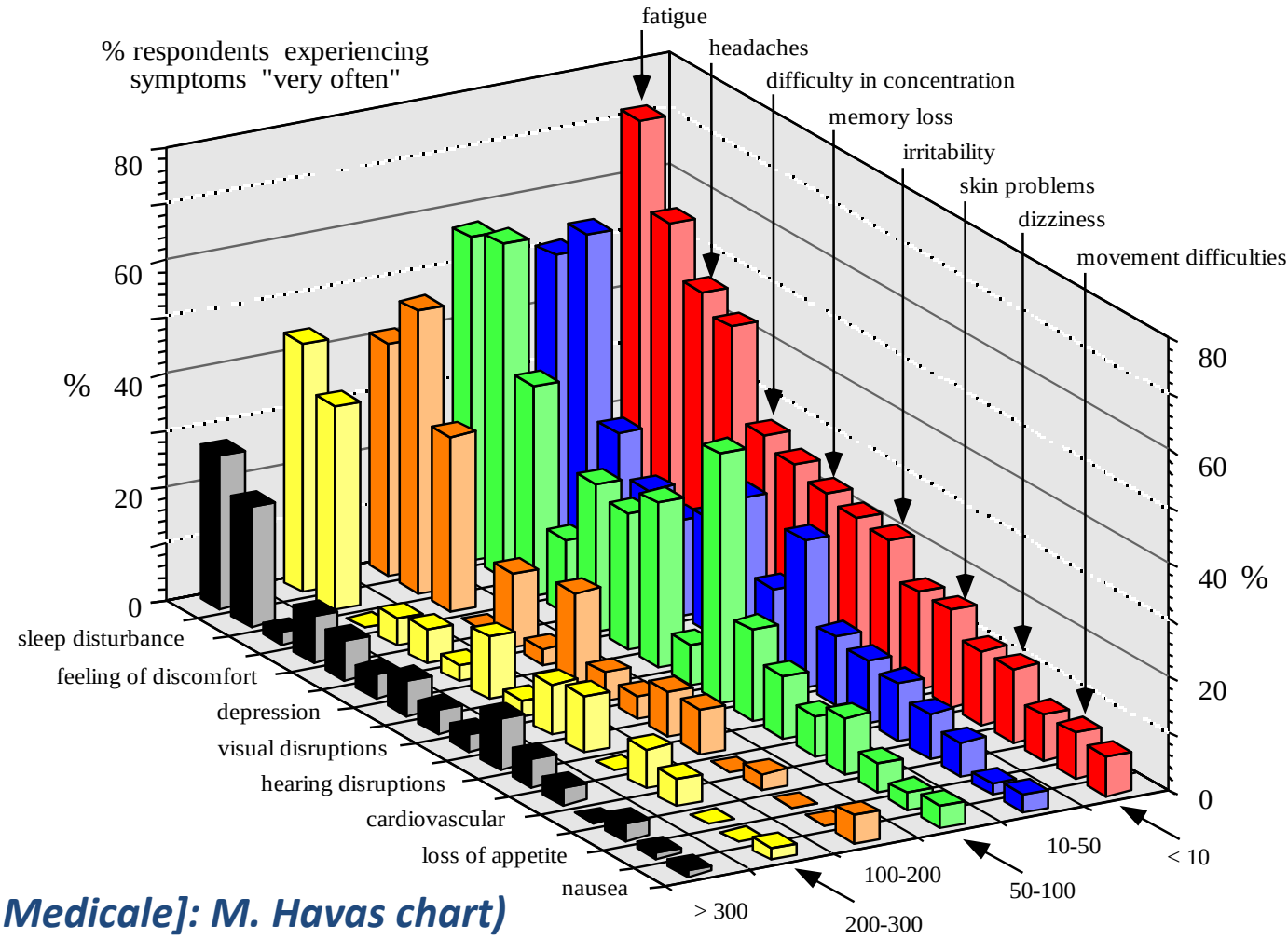
2001 Spain

Frequency of Electromagnetic Hypersensitivity Symptoms Based on Distance to Cell Phone Base Station

Electro-Hyper-Sensitivity (EHS)

1. Fatigue *
2. Sleep disturbance *
3. Headaches
4. Feeling of discomfort
5. Difficulty concentrating *
6. Depression *
7. Memory loss *
8. Visual disruptions *
9. Irritability *
10. Hearing disruptions *
11. Skin problems *
12. Cardiovascular *
13. Dizziness *
14. Loss of appetite *
15. Movement difficulties *
16. Nausea

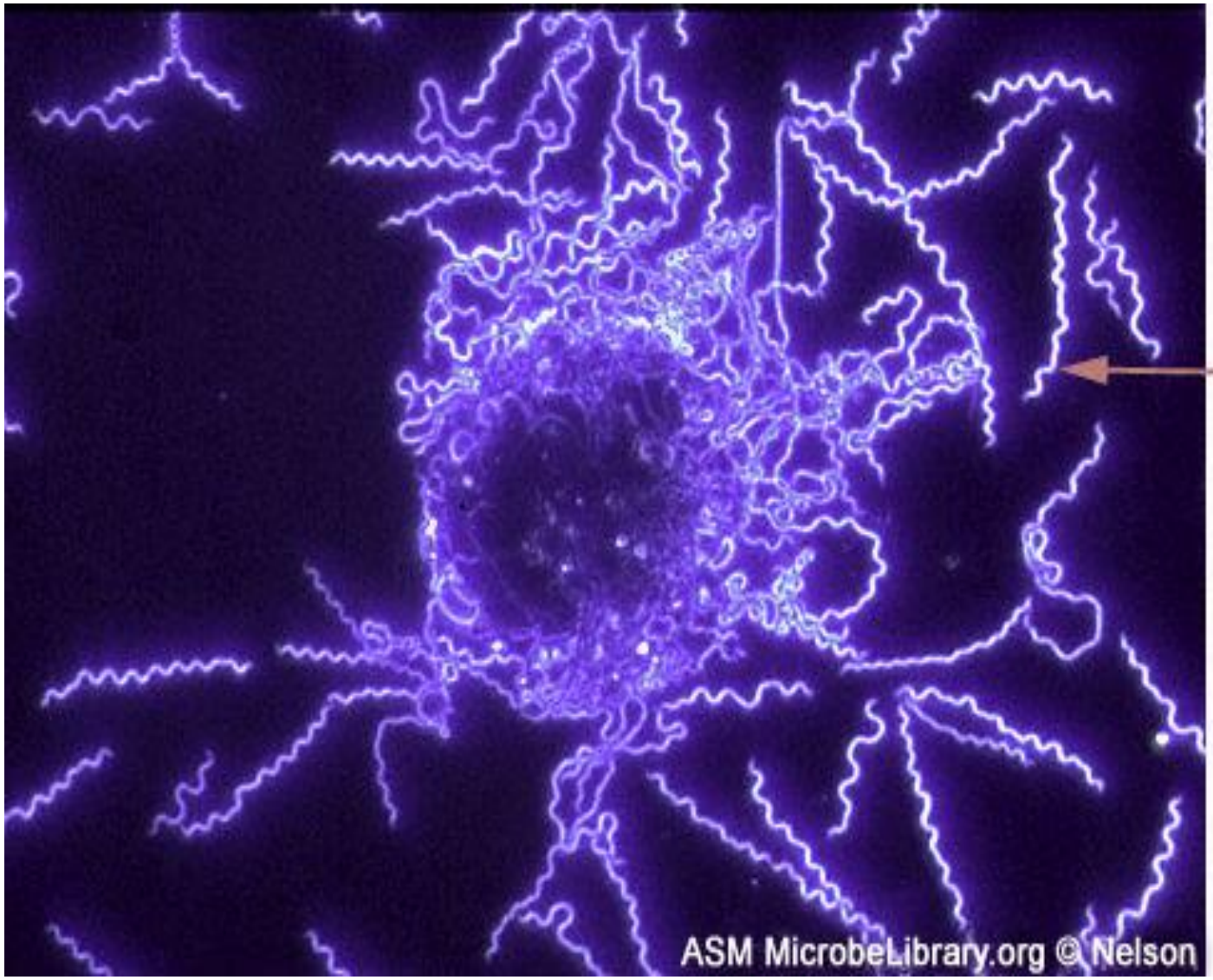
* Associated with Aging: "Rapid Aging Syndrome"

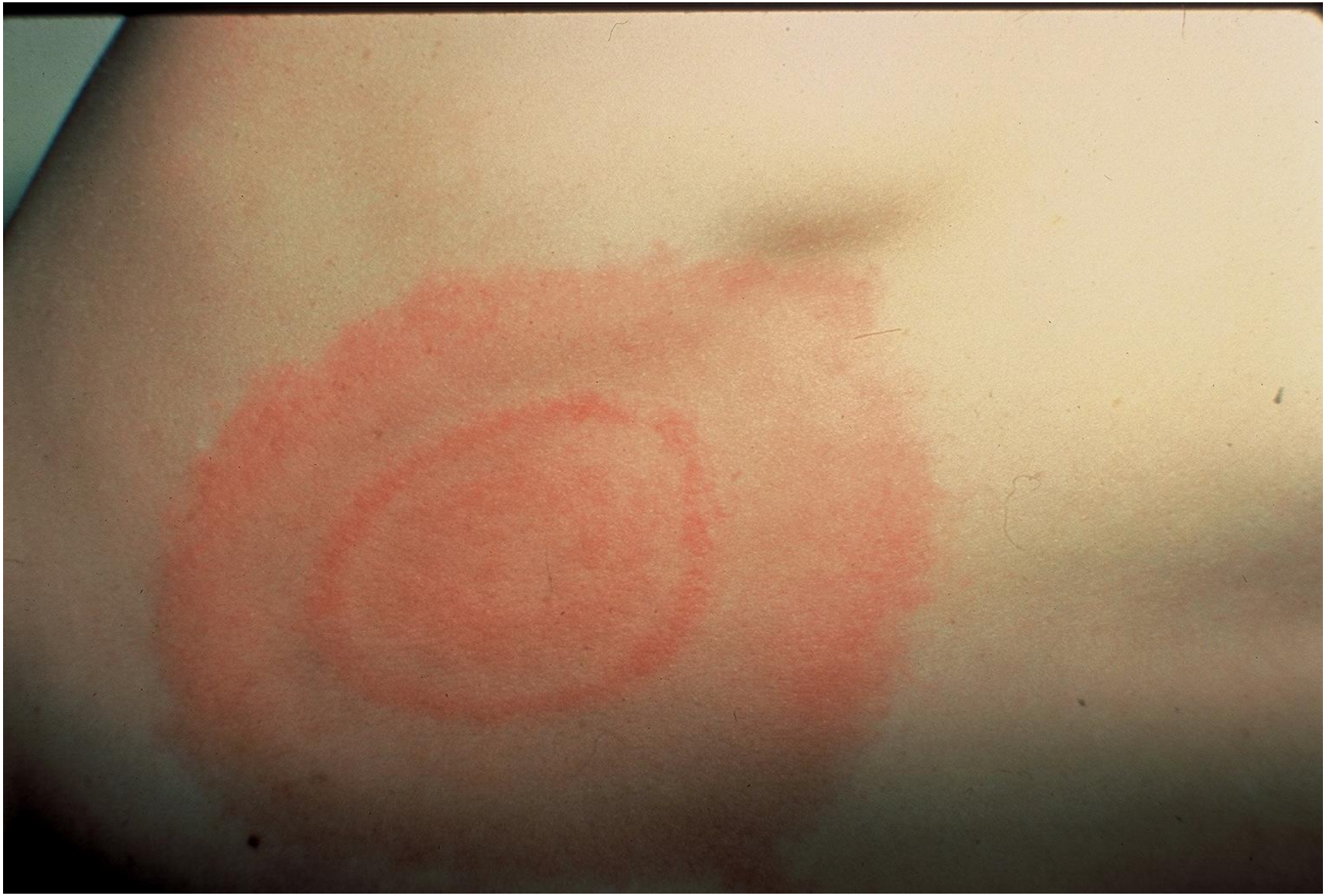


[Santini 2001, La Presse Medicale]: M. Havas chart)

Treatment of electrosmog in a "sick" sleeping location: the Faraday canopy

Lyme and Co


Spirochete bacteria,
Borrelia burgdorferi
ASM MicrobeLibrary.org © Nelson



Parasitology and Human Hormones

Testosterone – the double edged sword

Parasitology (2001), 123 : pp 365-371

DOI: 10.1017/S0031182001008599

Published online: 26 November 2001

Testosterone increases the transmission potential of tick-borne parasites

V. L. HUGHES and S. E. RANDOLPH

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Abstract

- Using laboratory-bred natural rodent hosts that had been castrated and then implanted with either testosterone or inert oil, we have shown that **testosterone causes prolonged and more intense infections of a tick-borne piroplasm, *Babesia microti***. This will result in more ticks becoming infected while feeding. Sexually active male rodents with high testosterone levels are also known to show increased locomotory activity and reduced innate and acquired resistance to tick feeding, so that more ticks are likely to be picked up and then fed successfully by these hosts. As a result, **the transmission potential of *B. microti* is significantly increased via hosts with high** rather than low **testosterone levels.**

  It is argued that **testosterone** helps to generate the observed aggregated distributions of parasites amongst their hosts, which also **enhances parasite persistence**

Question by Dr.Klinghardt: are **low hormone levels** in patients an **adaptive** intelligent response by the organism or are they indicating a failure of the system??

Lyme and Co-Infections

- Lyme is iron and magnesium dependent. Spirochetes can exchange iron for manganese if not enough iron is available. Use Mag only i.v. or i.m. We use 3 ml Mag Sulf. with 7 ml 1% preservative free procaine. This will out-feed the host, not the parasite!
- Babesia is iron and magnesium dependent. Artemisinin is a natural iron chelator and intracellular pro-oxidant. It is readily taken up by many species of microbes – and damages them! We give iron as bait in the evening and the Lyme/co-infection cocktail in the morning

J Neuroinflammation. 2011 Aug 4;8(1):90

Alzheimer's disease - a neurospirochetosis.

Analysis of the evidence following Koch's and Hill's criteria.

Miklossy J.

Abstract: It is established that chronic spirochetal infection can cause slowly progressive dementia, brain atrophy and amyloid deposition in late neurosyphilis. Recently it has been suggested that various types of spirochetes, in an analogous way to Treponema pallidum, could cause dementia and may be involved in the pathogenesis of Alzheimer's disease (AD). Here, we review all data available in the literature on the detection of spirochetes in AD and critically analyze the association and causal relationship between spirochetes and AD following established criteria of Koch and Hill. The results show a statistically significant association between spirochetes and AD (P = 1.5 x 10-17, OR = 20, 95% CI = 8-60, N = 247). When neutral techniques recognizing all types of spirochetes were used, or the highly prevalent periodontal pathogen Treponemas were analyzed, spirochetes were observed in the brain in more than 90% of AD cases. Borrelia burgdorferi was detected in the brain in 25.3% of AD cases analyzed and was 13 times more frequent in AD compared to controls. Periodontal pathogen Treponemas (T. pectinovorum, T. amylovorum, T. lecithinolyticum, T. maltophilum, T. medium, T. socranskii) and Borrelia burgdorferi were detected using species specific PCR and antibodies. Importantly, co infection with several spirochetes occurs in AD. The pathological and biological hallmarks of AD were reproduced in vitro. The analysis of reviewed data following Koch's and Hill's postulates shows a probable causal relationship between **neurospirochetosis** and AD. Persisting inflammation and amyloid deposition initiated and sustained by chronic spirochetal infection form together with the various hypotheses suggested to play a role in the pathogenesis of AD a comprehensive entity. As suggested by Hill, once the probability of a causal relationship is established prompt action is needed. Support and attention should be given to this field of AD research. Spirochetal infection occurs years or decades before the manifestation of dementia**.**

As adequate antibiotic and anti-inflammatory therapies are available, as in syphilis, one might prevent and eradicate dementia.

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SAMPLE ID: 103704

DIETRICH KLINGHARDT, MD

1200 112TH AV NE STE A100
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DRWN: 00/00/00
RCVD: 01/31/03
PRNT: 02/18/03

DIRECTOR: BOYD G. STEPHENS, M.

TEST NAME	RESULT	UNITS
LYME IgM WESTERN BLOT	.....	

The IgM WB is considered positive for the presence of AB to B. burgdorferi if two of the starred bands are present: 23-25, 31, 34, 39, 41 kDa.

The IgM WB is considered equivocal if one of the starred bands is present.

ASTPHLD/CDC Recommendations: An IgM WB is positive if two of these bands are present: 23-25, 39, 41 kDa. New York State Department of Health considers Western Blots positive that conform to the ASTPHLD/CDC criteria.

BAND INTENSITY: Low +, Medium ++, High +++, Equiv +/-

LYME IgM WESTERN BLOT	POSITIVE
18 kDa.	+/-
22 kDa.	-
**23-25 kDa.	+/-
28 kDa.	-
30 kDa.	+/-
**31 kDa.	+/-
**34 kDa.	+/-
37 kDa.	-
**39 kDa.	+
**41 kDa.	++
45 kDa.	+
58 kDa.	++
66 kDa.	+
73 kDa.	-
83 kDa.	-
93 kDa.	+

RVD MC 3/17/3

Continued on next page

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Artemisinin: A Versatile Weapon from Traditional Chinese Medicine

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Abstract

Traditional Chinese medicine (TCM) commands a unique position among all traditional medicines because of its 5000 years of tradition. Our own interest in natural products from TCM was triggered in the 1990s by sesquiterpene lactones of the artemisinin type from *Artemisia annua* L. The first description of the Chinese herb *Artemisia annua* L. (*qinghao*, Sweet wormwood) dates back to 168 B.C.E. Artemisinin (*qinghaosu*) was identified in 1972 as the active antimalarial constituent of *Artemisia annua* L. Artemisinin and its derivatives are used for the treatment of malaria. As shown in recent years, this class of compounds also shows activity against cancer cells, **schistosomiasis, and certain viruses, i.e., human cytomegalovirus, hepatitis B and C virus, and bovine viral diarrhea virus**. Interestingly, the bioactivity of artemisinin seems to be even broader and also includes the inhibition of other protozaons such as ***Leishmania, Trypanosoma*, and *Toxoplasma gondii*,** as well as some **trematodes, fungi, yeast, and bacteria.** The analysis of its complete profile of pharmacological activities, as well as the elucidation of molecular modes of action and the performance of clinical trials, will further elucidate the full potential of this versatile weapon from nature against diseases.

Treatment: Biological Antimicrobial Cocktail (BAC)

This remedy is based on 160 lbs body weight . The dosage has to be adjusted according to the weight of the child/person. The KLC has helped many Lymies and autistic children to improve significantly. Make the drink, keep in fridge , take 1/3rd am, 1/3rd at noon and 1/3rd in early evening

- 200-600 mg Artemisinin powder, 15 -20 ml Phopsholipid Exchange or 2 scoops LipoHealth, Vit C powder 4000 mg, Quintessence (Lyme, Ehrlichia, Bartonella) 6-8 dropperfull, Mimosa Pudica (worms, neuro regenerative): 2 Grams, Chaga: ½ tsp (Lyme), cordiceps: 1 tsp (mycoplasma)
  - Step 1: with 30 ml water in blender at high speed.
  - Step 2: to make liposomal compound, pour into "Ultrasonic cleaner " and vibrate for 12 minutes
  - Step 3: add back into blender, then add
- D-galactose : 5 grams (increases ATP dramatically)
- Glycine powder (Now): 10 grams/day
- 30 drops 25 % Propolis Tincture (anti-viral, restores MSH)
- ½ glass grapefruit juice (ensures continued artemisinin absorbtion)
- ½-1 glass Cistus tea as additional biofilm breaker(BioPure.eu)
- 1 apple/1 orange for fiber, nutrients and taste

- *Binders: MicroSilica 1scoop (100 mg) or BioPure Chlorella (15-60 tbl) is taken 1 hour after each cocktail drink to scoop up the fallout. 4 cups of horsetail teac/day (aluminum)*
- *Metal detox/anti-inflammatory: CurcuSyn: 2-3 caps 3 times per day with fatty meal or cocktail*

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