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.eu)

Chlorella and the chelation of heavy metals

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

Chlorenergy (BioPure.eu)

- 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

Melatonin to clear the brain of mercury

C *Pharmacology & Toxicology* 2003, **93**, 290–296. *ISSN 0901-9928*

Melatonin Protects Against Mercury(II)-Induced Oxidative Tissue Damage in Rats

Göksel S ̧ener, A. Özer S ̧ehirli, Gül Ayanog˘lu-Dülger

Marmara University, School of Pharmacy, Department of Pharmacology, Istanbul, Turkey (Received February 10, 2003; Accepted September 29, 2003)

Abstract: Mercury exerts a variety of toxic effects in the body. Lipid peroxidation, DNA damage and depletion of reduced glutathione by Hg(II) suggest an oxidative stress-like mechanism for Hg(II) toxicity. Melatonin, the main secretory product of the pineal gland, was recently found to be a potent free radical scavenger and antioxidant. N-Acetylcysteine, a precursor of reduced glutathione and an antioxidant, is used in the therapy of acute heavy metal poisoning. In this study the protective effects of melatonin in comparison to that of N-acetylcysteine against Hg-induced oxidative damage in the kidney, liver, lung and brain tissues were investigated. Wistar albino rats of either sex (200–250 g) were divided into six groups, each consisting of 8 animals. Rats were intraperitoneally injected with 1) 0.9% NaCl, control (C) group; 2) a single dose of 5 mg/kg mercuric chloride (HgCl2), Hg group; 3) melatonin in a dose of 10 mg/kg, 1 hr after HgCl2 injection, Hg-melatonin group; 4) melatonin in a dose of 10 mg/kg one day before and 1 hr after HgCl2 injection, melatonin-Hg-melatonin group; 5) N-acetylcysteine in a dose of 150 mg/kg, 1 hr after HgCl2 injection, Hg-N-acetylcys- teine group, and 6) N-acetylcysteine in a dose of 150 mg/kg one day before and 1 hr after HgCl2 injection, N-acetylcys- teine-Hg-N-acetylcysteine group. Animals were killed by decapitation 24 hr after the injection of HgCl2. Tissue samples were taken for determination of malondialdehyde, an end-product of lipid peroxidation; glutathione (GSH), a key antioxi- dant, and myeloperoxidase activity, an index of neutrophil infiltration. The results revealed that HgCl2 induced oxidative tissue damage, as evidenced by increases in malondialdehyde levels. Myeloperoxidase activity was also increased, and GSH levels were decreased in the liver, kidney and the lungs. All of these effects were reversed by

Treatment for Mercury Toxicity

- Prevention of mercury toxicity: mother should have mercury-amalgam fillings removed long before getting pregnant with aggressive treatment (complexing and detox agents), using DMPS, DMSA, D-Penicillamine, curcumin, chlorella, cilantro, melatonin etc.
- During pregnancy and lactation: do not remove fillings or root canal filled teeth! Use chlorella as primary fetus-protective strategy
- Hg detox: always supplement nanonized minerals ("MicroMinerals" and electrolyte ("Matrix Electrolyte") to keep kidneys open and fill the vacant sites with minerals after toxic metals leave their binding sites
- Bedtime: lipo-melatonin (1-8 mg), chlorella (250 mg tbl, 10-25) and other binders at night (ZeoClear, MicroSilica)
- Daytime: chlorella, microsilica, cilantro, DMPS, DMSA, Homeo-K Clear (BioPure.EU)
- To lower testosterone: use both high potency homeopathic testosterone and DHEA (www.sophiaNurition.com), high dose Vit A and/or Ketoconazole (Nizoral)
- Ionic foot bath with oral cilantro

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

REVIEW

Mercury Toxicity and Systemic Elimination Agents

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

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

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

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

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

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

DENTAL AMALGAMS ARE A MAJOR SOURCE OF MERCURY TOXICITY

A single dental amalgam filling with a surface area of only 0.4 cm^{-2} is estimated to release as much as 15 μg Hg day^{-1} primarily through mechanical wear and evaporation [1, 9–11]. The average individual has eight amalgam fillings and could absorb up to 120 μg Hg day^{-1} from their amalgams. These levels are consistent with reports of 60 μg Hg day^{-1} collected in human feces [12]. By way of contrast, estimates of the daily absorption of all forms of mercury from fish and seafood is 2.3 μg and from all other foods, air and water is 0.3 μg per day [13]. Currently, Germany, Sweden and Denmark severely restrict the use of amalgams [1].

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

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

The poisoning of our brain with Aluminum

- Orally absorbed aluminum is not very toxic on its own. However, it has devastating synergistic effects with traces of mercury in the body
- There is no mercury-free person on the planet, no river or body of water without mercury, it's in the air and in the food, and often in the teeth
- We have no biological barrier to inhaled aluminum from the ongoing and evident but secretive climate modification program
- Detoxing aluminum improves brain and CNS function, eliminates inflammation in the body and leads to numerous other improvements of health parameters. The increasing aluminum burden parallels the increase in body burden of parasites and may be the cause of it

Immunome Res 2013, 9:1 http://dx.doi.org/10.4172/1745-7580.1000069

Aluminum's Role in CNS-immune System Interactions leading to Neurological Disorders

Shaw CA1,2,3*, Kette SD4, Davidson RM5 and Seneff S6
Neural Dynamics Research Group, Department of Ophthalmology and

Abstract

Multisystem interactions are well established in neurological disorders, in spite of conventional views that only the central nervous system (CNS) is impacted. We review evidence for **mutual interactions between the immune and nervous systems** and show how these seem to be **implicated in the origin and progression of nervous system disorders**. Well-established immune system triggers leading to autoimmune reactions are considered. Of these, **aluminum, a known neurotoxicant, may be of particular importance.** We have demonstrated elsewhere that aluminum has the potential to induce damage at a range of levels in the CNS leading to neuronal death, circuit malfunction and ultimately, system failure. Aluminum is widely used as an adjuvant in various vaccine formulations and has been implicated in a multisystem disorder termed "autoimmune/inflammatory syndrome induced by adjuvants" (ASIA). The implications of aluminum-induced ASIA in some disorders of the CNS are considered. We propose a unified theory capturing a progression from a local response to a systemic response initiated by

Entropy **2012**, *14*(11), 2227-2253; doi:10.3390/e14112227

Empirical Data Confirm Autism Symptoms Related to Aluminum and Acetaminophen Exposure

Stephanie Seneff , Robert M. Davidson and Jingjing Liu

- **Abstract**

Autism is a condition characterized by impaired cognitive and social skills, associated with compromised immune function. The incidence is alarmingly on the rise, and environmental factors are increasingly suspected to play a role. This paper investigates word frequency patterns in the U.S. CDC Vaccine Adverse Events Reporting System (VAERS) database. Our results provide strong evidence supporting **a link between autism and the aluminum in vaccines**. A literature review showing toxicity of aluminum in human physiology offers further support. Mentions of autism in VAERS increased steadily at the end of the last century, during a period when mercury was being phased out, while aluminum adjuvant burden was being increased. Using standard log-likelihood ratio techniques, we identify several signs and symptoms that are significantly more prevalent in vaccine reports after 2000, including **cellulitis, seizure, depression, fatigue, pain and death, which are also significantly associated with aluminum-containing vaccines**.

We propose that **children with the autism diagnosis are especially vulnerable to toxic metals such as aluminum and mercury due to insufficient serum sulfate and glutathione**. A strong correlation between autism and the MMR (Measles, Mumps, Rubella) vaccine is also observed, which may be partially explained via an increased sensitivity to acetaminophen administered to control fever.

Keywords: autism; vaccines; MMR; HEP-B; glutathione; sulfate; cholesterol sulfate; aluminum; mercury;acetaminophen

PK Vaccination schedule and parental exposure

Mother: Amalgams: exposure to inorganic mercury in utero and through breast feeding - potentially increase metal mobilisation by oral antibiotics

Father: Autoimmune genetic susceptibility

PK age	Vaccine	Adjuvant		Contaminants/ culture
2 months	DTP/Polio/Hib	Aluminium Hydroxide	0.5mg	VERO
	Hep B	Aluminium Hydroxide	0.5 mg	Saccharomyces cerevisiae
3 months	repeat	Aluminium Hydroxide	1.0 mg	
4 months	repeat	Aluminium Hydroxide	1.0 mg	
9 months	Men C	Aluminium Hydroxide	0.5 mg	
12 months	MMR	-		Lactose, eggs

Toxic Rain in the US (D.Lim). UK: much worse

EPA: Normal Values for Aluminum in Rain: 0 - 0.5 µg/l

Location	Sample	Aluminium	Barium
Redding, US	Rain	1010	25
California, US	Rain	2190	43
California, US	Rain	3450	
Lincolnshire, UK	Rain	70	<10
Portsmouth, UK	Rain	350	16
Florida, US	Rain	182	
Florida, US	Rain	127	
California, US	Snow	61,100	83
Brisbane, AU	Rain	1900	11
Hawaii, US	Rain	400	39

Int. J. Environ. Res. Public Health 2015, 12, 9375-9390

J. Marvin Herndon, *Received: 29 June 2015 / Accepted: 5 August 2015 / Published: 11 August 2015*

Abstract: The widespread, intentional and increasingly frequent chemical emplacement in the troposphere has gone unidentified and unremarked in the scientific literature for years. The author presents evidence that toxic coal combustion fly ash is the most likely **aerosolized particulate sprayed by tanker-jets for geoengineering, weather-modification and climate-modification purposes** and describes some of the multifold consequences on public health. Two methods are employed: (1) Comparison of 8 elements analyzed in rainwater, leached from aerosolized particulates, with corresponding elements leached into water from coal fly ash in published laboratory experiments, and (2) Comparison of 14 elements analyzed in dust collected outdoors on a high-efficiency particulate air (HEPA) filter with corresponding elements analyzed in un-leached coal fly ash material. The results show: (1) the assemblage of elements in rainwater and in the corresponding experimental leachate are essentially identical. At a 99% confidence interval, they have identical means (T-test) and identical variances (F-test); and (2) the assemblage of elements in the HEPA dust and in the corresponding average un-leached coal fly ash are likewise essentially identical.

The consequences on public health are profound, including exposure to a variety of toxic heavy metals, radioactive elements, and neurologically-implicated chemically mobile aluminum released by body moisture *in situ* after inhalation or through transdermal induction.

Diagnosis: nanonized aluminum does not show up in the hair analysis and hardly with any mobilization test. Best tests: whole blood or i.v. Desferal/EDTA/Vit C challenge. Very best and only reliable test: apheresis and toxicity testing on the eluat or energetic testing (ART)

Treatment: Aluminum detox

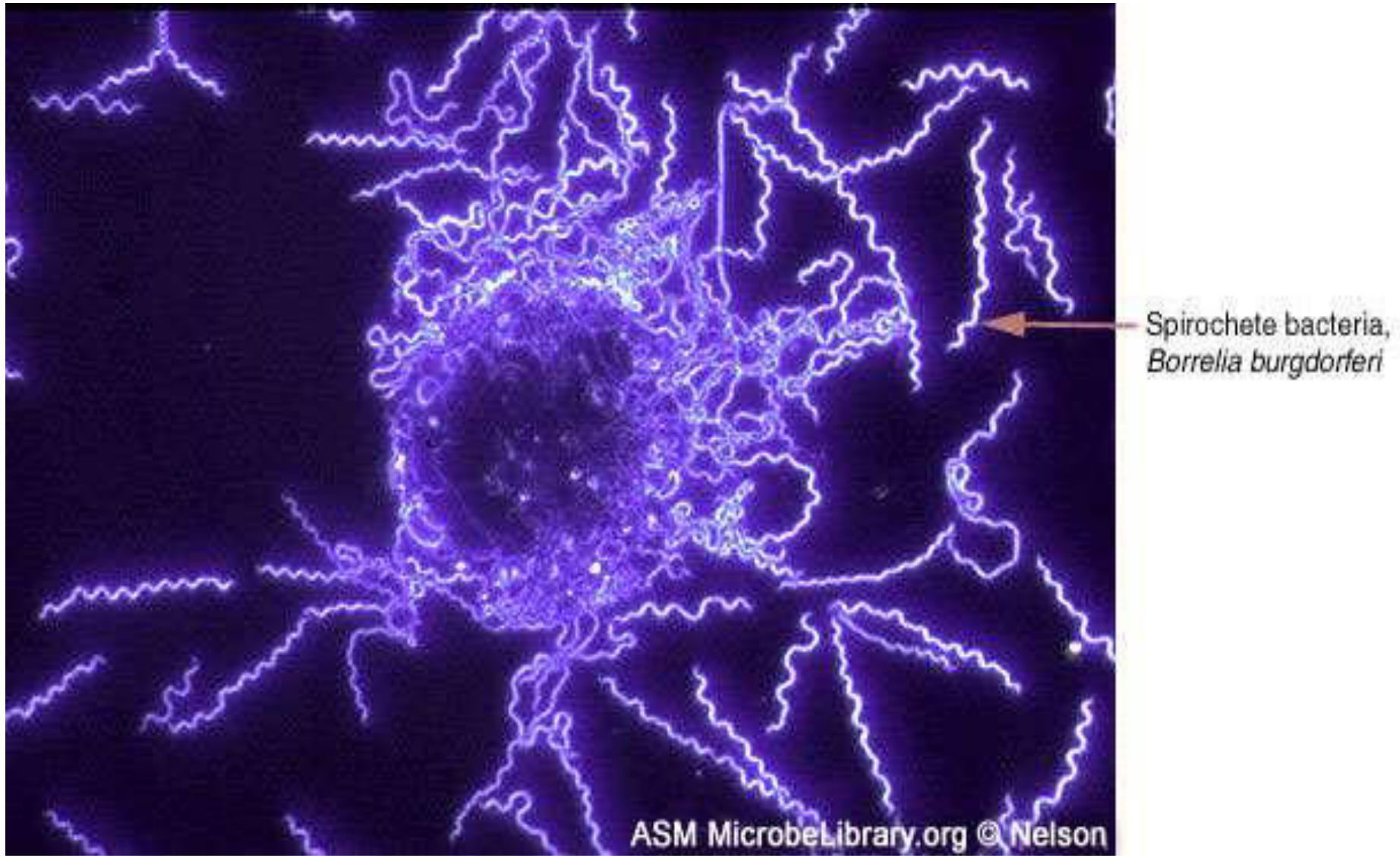
1. Dr.Omura's **Cilantro:** take 1 sublingual tablet per day followed by hand acupressure for uptake

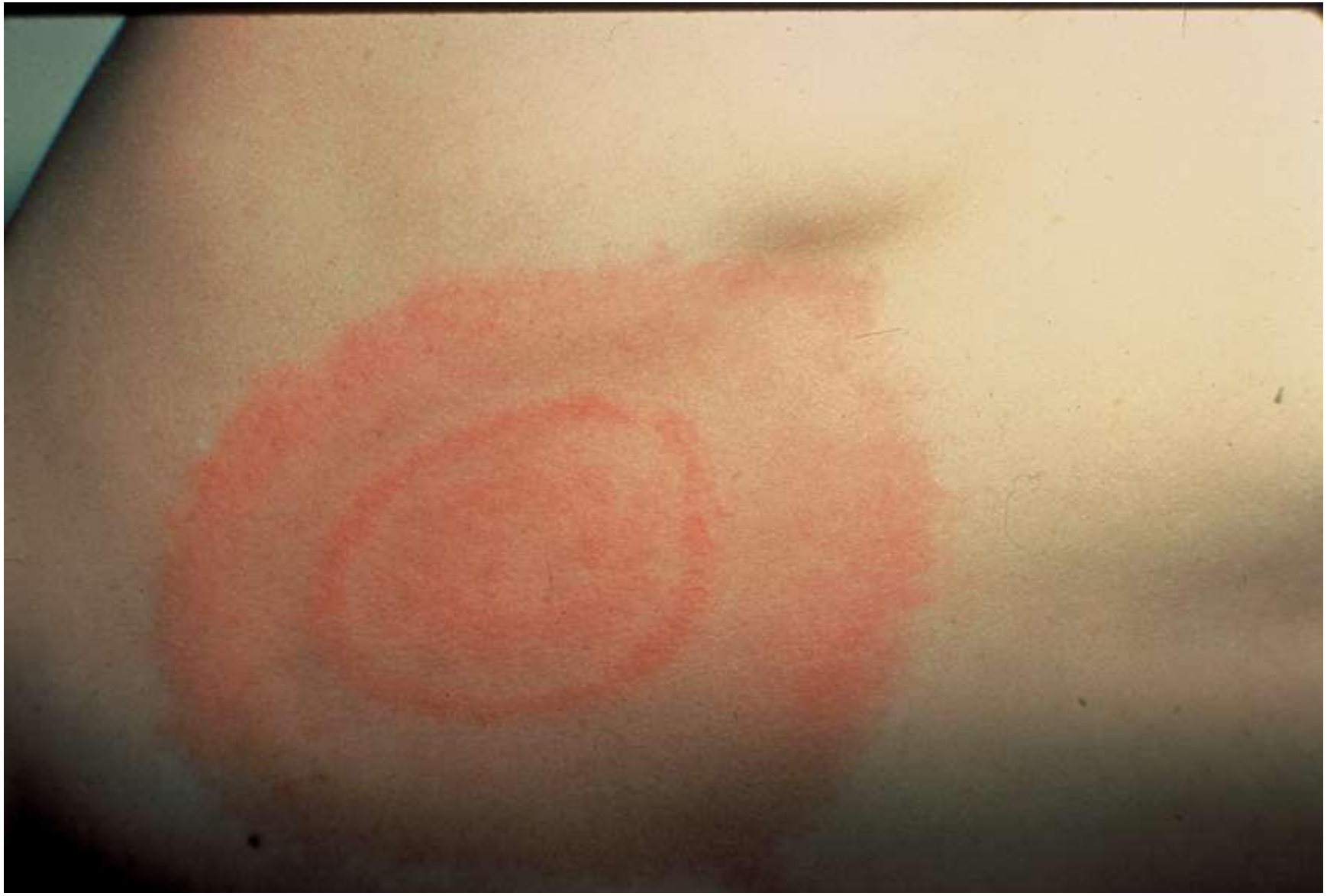
   Omura's Study: in 39 days body burden reduced by 50% (AL und Pb)
   Omura, Y. et al: Acupunct.Electrother.Res.1995; 20(3-4): 195-229

2. **Silica**: fill a glassbottle with 60% **horsetail tea** and 40% **stingy nettle** leaf tea made with RO-water, add the juice of a freshly squeezed lemon. Add 6 pipettes of Cilantro tincture. Drink throughout the day. Or "BioSil" drops
3. **Liposomal Malate** (6 year old: 1 tsp/day) or Magnesium Malate
4. **Bioavailable zinc and manganese with food** ("Core" from BioPure.eu)
5. Binders: **Enterosgel**, **chlorella (BioPure.eu)**
6. **Laser detox** with different aluminum salts
7. Homeopathic **Alumina met.30 C** and alum. sulf 30 C
8. Short term: Desferal s.c injection once weekly or oral capsules (Novartis)

Borrelia

- One of 8 genera of spirochetes
- 100ds of species in these 8 genera
- “Borrelia” is the genus, “Burgdorferi” the species
- Other famous spirochetes: treponema pallidum (syphilis), leptospira (leptopspirosos from animal feces contaminated drinking water, common in Maui, New Mexico, etc)
- Bb sensu lato includes B. Afzelii, B. garinii, B.lonstari, B.andersonii and many others
- Bb sensu stricto refers only to Bb, but includes many species that cause identical symptoms
- In Europe, 5 strains of Bb sensu lato, in Japan 61 strains
- Also be aware that microbes constantly exchange via plamids DNA with each other and we found Bb microbes with properties usually only found in Babesia or mycoplasma, etc. There are no fixed boundaries between many of these microbes.


Spirochete bacteria,
Borrelia burgdorferi
ASM MicrobeLibrary.org © Nelson



LYME BORRELIOSIS: GREAT IMITATOR

Lyme is a spirochetal illness resembling syphilis.

**Can mimic MS, myelopathy,
polyneuropathy,
brain tumor,
encephalopathy.**
(Neurosurgery.1992May;30(5):769-73)

**Can cause meningitis,
encephalitis,
neuritis,mania,
depression, OCD,
schizophrenia, anorexia, dementia.**
(Am J Psychiatry. 1994 Nov;151(11):1571-83)

LYME BORRELIOSIS: GREAT IMITATOR

90% of chronic fatigue patients are Lyme positive.
(Informal study by American Lyme Disease Alliance at www.lymealliance.org)

Most fibromyalgia patients are Lyme positive.
(Rheum Dis Clin North Am. 1998 May;24 (2):323-51 & report of Lida Mattman,M.D.)

Borrelia can cause Parkinsonism
(Arch.of Path.& Lab.Med.127(9):1204-6)

LYME BORRELIOSIS: GREAT IMITATOR

Borrelia is found in the CSF of most MS & ALS patients
(Communications from Jo Anne Whitaker,M.D. and Lida Mattman,M.D.)

Many patients with arthritis have Lyme but only 24% of Lyme patients have arthritis
(Z Rheumatol.2003 Oct;62(5):450-8)

Borrelia may cause sarcoidosis
(Chin Med J.1992 Jul;105(7):560-3)

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.

2014 Oct 14.

Maternal immune activation and abnormal brain development across CNS disorders.

Knuese I, Chicha L, Britschgi M, Schobel SA, Bodmer M, Hellings JA, Toovey S, Prinssen EP

Abstract

Epidemiological studies have shown **a clear association between maternal infection and schizophrenia or autism in the progeny.** Animal models have revealed maternal immune activation (mIA) to be a profound risk factor for neurochemical and behavioural abnormalities in the offspring. Microglial priming has been proposed as a major consequence of mIA, and represents a critical link in a causal chain that leads to the wide spectrum of neuronal dysfunctions and behavioural phenotypes observed in the juvenile, adult or aged offspring. Such diversity of phenotypic outcomes in the mIA model are mirrored by recent clinical evidence suggesting that infectious exposure during pregnancy is also associated with epilepsy and, to a lesser extent, cerebral palsy in children. Preclinical research also suggests that mIA might precipitate the development of Alzheimer and Parkinson diseases. Here, we summarize and critically review the emerging evidence that mIA is a shared environmental risk factor across CNS disorders that varies as a function of interactions between genetic and additional environmental factors. We also review ongoing clinical trials targeting immune pathways affected by mIA that may play a part in disease manifestation. In addition, future directions and outstanding questions are

The association between tick-borne infections, Lyme borreliosis and autism spectrum disorders

Medical Hypotheses, Volume 70, Issue 5, 2008, Pages 967–974

Robert C. Bransfield , Jeffrey S. Wulfman, William T. Harvey, Anju I. Usman[d]

Summary

Chronic infectious diseases, including tick-borne infections such as *Borrelia burgdorferi* may have direct effects, promote other infections and create a weakened, sensitized and immunologically vulnerable state during fetal development and infancy leading to increased vulnerability for developing autism spectrum disorders. A dysfunctional synergism with other predisposing and contributing factors may contribute to autism spectrum disorders by provoking innate and adaptive immune reactions to cause and perpetuate effects in susceptible individuals that result in inflammation, molecular mimicry, kynurenine pathway changes, increased quinolinic acid and decreased serotonin, oxidative stress, mitochondrial dysfunction and excitotoxicity that impair the development of the amygdala and other neural structures and neural networks resulting in a partial Klüver–Bucy Syndrome and other deficits **resulting in autism spectrum disorders** and/or exacerbating autism spectrum disorders from other causes throughout life.

Support for this hypothesis includes multiple cases of mothers with Lyme disease and children with autism spectrum disorders; fetal neurological abnormalities associated with tick-borne diseases; similarities between tick-borne diseases and autism spectrum disorder regarding symptoms, pathophysiology, immune reactivity, temporal lobe pathology, and brain imaging data; positive reactivity in several studies with autistic spectrum disorder patients for *Borrelia burgdorferi* (22%, 26% and 20–30%) and 58% for mycoplasma; similar geographic distribution and improvement in autistic symptoms from antibiotic treatment. It is imperative to research these and all possible causes of autism spectrum disorders in order to prevent every preventable case and treat every treatable case until this disease has been eliminated from humanity.

The Psychoimmunology of Lyme/Tick-Borne Diseases and its Association with Neuropsychiatric Symptoms

The Open Neurology Journal, 2012, 6, (Suppl 1-M3) 88-93
1874-205X/12 2012 Robert C. Bransfield

Abstract: Disease progression of neuropsychiatric symptoms in Lyme/tick-borne diseases can be better understood by greater attention to psychoimmunology. Although there are multiple contributors that provoke and weaken the immune system, infections and persistent infections are significant causes of pathological immune reactions. Immune mediated ef-fects are a significant contributor to the pathophysiological processes and disease progression. These immune effects in-clude persistent inflammation with cytokine effects and molecular mimicry and both of these mechanisms may be present at the same time in persistent infections. Sickness syndrome associated with interferon treatment and autoimmune limbic encephalopathies are models to understand inflammatory and molecular mimicry effects upon neuropsychiatric symp-toms. Progressive inflammatory reactions have been proposed as a model to explain disease progression in depression, psychosis, dementia, epilepsy, autism and other mental illnesses and pathophysiological changes have been associated with oxidative stress, excitotoxicity, changes in homocysteine metabolism and altered tryptophan catabolism. Lyme dis-ease has been associated with the proinflammatory cytokines IL-6, IL-8, IL-12, IL-18 and interferon-gamma, the chemokines CXCL12 and CXCL13 and increased levels proinflammatory lipoproteins. *Borrelia burgdorferi* surface glycolipids and flagella antibodies appear to elicit anti-neuronal antibodies and anti-neuronal antibodies and *Borrelia burgdorferi* lipoproteins can disseminate from the periphery to inflame the brain.

Autism spectrum disorders associated with Lyme/tick-borne diseases may be mediated by a combination of inflammatory and molecular mimicry mechanisms. Greater interaction is needed between infectious disease specialists, immunologists and psychiatrists to benefit from this awareness and to further understand these

Typical lab indicators for Lyme &Co

- Low wbc (DD: viral infection, mycotoxins)
- Elevated MCV resistant to MeB12 and Folinic Acid. Needs high dose L5-MTHF to correct (MTHFR homo-or heterozygous SNP)
- Elevated fasting blood sugar 90 -110 (mild insulin resistance)
- Low alkaline phosphatase (microbially induced HPU = low zinc, magnesium, manganese, biotin, taurin, etc.)
- Moderately elevated LDL (= neuroprotective)
- Low urine specific gravity (microbially induced pituitary dysfunction = low ADH, low hGH, other low pituitary hormones)
- The sickest patients have very low triglycerides, low cholesterol (= precise, immediate and gentle action needed)
- Elevated C3a and C4a and TGF beta 1

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PALO ALTO, CA 94303
(800)832-3200

PATIENT: KELLY, MARIA
DOB: 04/2[redacted]

SAMPLE ID: 103704

DIETRICH KLINGHARDT, MD

1200 112TH AV NE STE A100
BELLVUE, WA
98004

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 NL 3/17/3

Continued on next page

Dia[illegible] Western - Blot
int[illegible] ratory tests alone. Results should be
cal symptoms and patient history.

IMMUNOSCIENCES LAB., INC.
8693 Wilshire Blvd., Beverley Hills, CA 90211
Tel: 310-657-1077 Fax: 310-657-1053

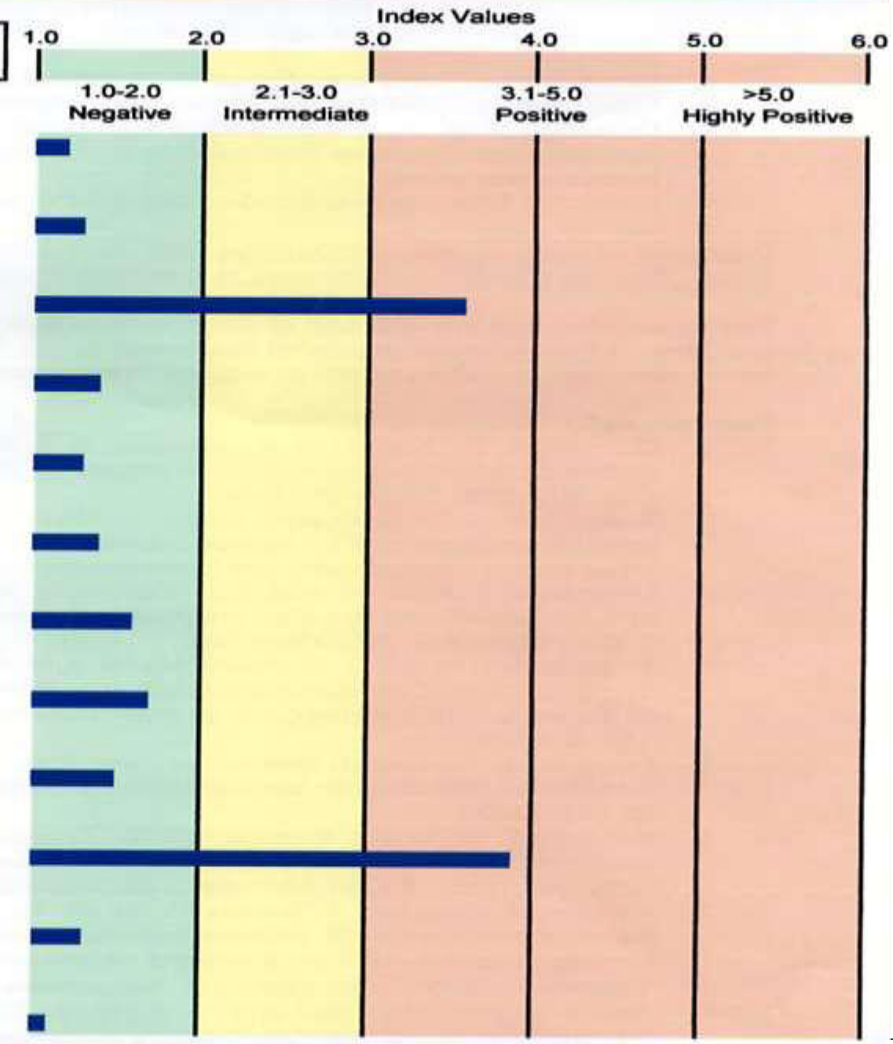
Patient Name: **Klinghardt, Dietrich**
Report Number: **175056**
Blood Drawn: **12/21/2004**
Date Reported: **1/14/2005**

Clinic:

Klinghardt, Dietrich
1200-112th Avenue, NE, Suite a-100
Bellevue WA 98104 USA

IgM Antibodies to Borrelia burgdorferi and Cross Reactive Antigens:

Antigen	Index Value Normal	Abnormal
Unrelated Spirochete IgM	1.2	
Borrelia Lysate IgM	1.3	
OSPA IgM		3.6
OSPE 1+2 IgM	1.4	
LFA IgM	1.3	
C2 + C6 IgM	1.4	
VR1+VR2 IgM	1.6	
B.B. Sensu Stricto IgM	1.7	
B. Garinii IgM	1.5	
B. Afzeli IgM		3.9
Babesia IgM	1.3	
Ehrlichia IgM	1.1	



Co-Infections include:

Babesia (protozoa)

FL 1953 (Stephen Fry: protozoa, protomyxzoa rheumatica)

Ehrlichia (intracellular bacteria)

Coxiella (rickettsia)

Bartonella (cat scratch disease: bacteria)

Mycoplasma (L-form)

Opportunistic infections/infestations, e.g.:

Parasites (lungworm V.Klapowi, helminths, protozoa)

Mould (aspergillus sp., penicillum sp., cladysporium, etc.)

Viruses (HSV 1 &2, CMV,EBV, HHV-6, XMRV, Coxsackie, HSV, HHV-6, etc.)

Medical Hypotheses; May 2012, Vol 78, Issue 5, 606-615

"Long term antibiotic therapy may be an effective treatment for children co-morbid with Lyme disease and Autism Spectrum Disorder" Kuhn, M, S.Grave, R.Bransfield, S.Harris

Patients diagnosed with Lyme disease share many of the same physical manifestations as those diagnosed with an Autism Spectrum Disorder (ASD). In this study four male children (ages 26–55 months) who have an ASD diagnosis and one male child (age 18 months) who displayed behaviors consistent with an ASD, were assessed using the SCERTS Assessment Process Observation (SAP-O) form. The SAP-O meets state and federal requirements for providing a comprehensive, ongoing assessment of a child with an ASD [33] . The SAP-O form measures children's abilities using observational, authentic assessment procedures in the domains of joint attention, symbol use, mutual regulation, and self regulation via observations of specific behaviors in familiar settings [33] . The five children tested positive for Lyme disease and their SAP-O score was evaluated before and after 6 months of antibiotic therapy. Each child was prescribed 200 mg of amoxicillin three times per day and three of the five children were prescribed an additional 50 mg of Azithromycin once per day.

All of the children's scores on the SAP-O assessment improved after 6 months of antibiotic therapy. The assessors also reported anecdotal data of improved speech, eye contact, sleep behaviors, and a reduction of repetitive behaviors.

Herbal Drugs: Ethnomedicine to Modern Medicine
2009, 173-194, DOI: 10.1007/978-3-540-79116-4_11

Artemisinin: A Versatile Weapon from Traditional Chinese Medicine

Thomas Efferth

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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

Artemisia Extracts: Artesunate, Artemisinin

Sensitivity of human herpesvirus 6 and other human herpesviruses to the broad-spectrum anti-infective drug artesunate.

J Clin Virol. 2009 Jun 3

Milbradt J, Auerochs S, Korn K, Marschall M.
Institute for Clinical and Molecular Virology, Medical Center Erlangen, University of Erlangen-Nuremberg, Germany.

BACKGROUND: Antiviral therapy for HHV-6 infection with conventional anti-herpesviral drugs is problematic so novel drugs are required. Artesunate is a well-tolerated drug approved for malaria therapy which possesses antiviral activity. OBJECTIVE: The artesunate sensitivity of HHV-6 was analyzed and compared to that of several other human herpesviruses. STUDY DESIGN: Cultured human cells were productively infected with strains of HHV-6 or other human herpesviruses to measure artesunate inhibition of viral protein synthesis (Western blot analysis) or viral genome replication (qPCR), and to determine IC(50) values by immunofluorescence or plaque reduction assays. RESULTS: **Sensitivity of HHV-6** to artesunate was demonstrated with an IC(50) of 3.80+/-1.06muM. This is in a range similar to IC(50) values for H**CMV** and **EBV**. Artesunate treatment of HHV-6-infected cells significantly reduced viral early and late protein synthesis that occurred in the absence of drug-induced apoptosis or necrotic cytotoxicity. HHV-6A genome replication was markedly reduced by artesunate.

CONCLUSION: Artesunate possesses anti-HHV-6 activity in vitro and may be useful for the treatment of HHV-6 infections.

Effect of artemisinin/artesunate as inhibitors of hepatitis B virus production in an in vitro replicative system

Antiviral Research 2005, vol. 68, nº2, pp. 75-83

ROMERO Marta R. (1) ; EFFERTH Thomas (2) ; SERRANO Maria A. (1) ; CASTANO Beatriz (3) ; MACIAS Rocio I. R. (3) ; BRIZ Oscar (3) ; MARIN Jose J. G. (3)

Résumé / Abstract

The antiviral effect against hepatitis B virus (HBV) of artemisinin, its derivative artesunate and other compounds highly purified
from traditional Chinese medicine remedies, were investigated. HBV production by permanently transfected HepG2 2.2.15 cells
was determined by measuring the release of surface protein (HBsAg) and HBV-DNA after drug exposure (0.01-100 μM) for 21
days. The forms of HBV DNA released were investigated by Southern blotting. Neutral Red retention test was used to evaluate
drug-induced toxicity on host cells. The compounds were classified according to their potential interest as follows: (i) none: they
had no effect on viral production (daidzein, daidzin, isonardosinon, nardofuran, nardosinon, tetrahydronardosinon and
quercetin); (ii) low: they were able to markedly reduce viral **production**, but also induced toxicity on host cells (berberine and
tannic acid) or they had no toxic effect on host cells but only had a moderate ability to reduce viral production (curcumin,
baicalein, baicalin, bufalin, diallyl disulphide, glycyrrhizic acid and puerarin); (iii) high: **they induced strong inhibition of viral
production at concentrations at which host cell viability was not affected (artemisinin and artesunate).** Moreover, artesunate

J Liposome Res. 2010 Dec 16

Conventional and long-circulating liposomes of artemisinin: preparation, characterization, and pharmacokinetic profile in mice.

Isacchi B, Arrigucci S, Marca GL, Bergonzi MC, Vannucchi MG, Novelli A, Bilia AR.

Department of Pharmaceutical Sciences, University of Florence, Florence, Italy.

Abstract

Artemisinin (qinghaosu), a unique endoperoxide sesquiterpene lactone isolated from Artemisiaannua L., is a very active antimalarial drug, including severe and cerebral malaria. However, its therapeutical efficacy is limited due to its scarce bioavailability. In this article, artemisinin-loaded conventional and polyethylene glycol (PEGylated) liposomes were proposed as carriers to increase biopharmaceutical properties of the drug. Encapsulation efficacy was determined by high-performance liquid chromatography/diode array detection/electrospray ionization-mass spectrometry, dimensional analysis was performed by dynamic light scattering, and morphology was performed by trasmission electron microscopy. After dialysis, both liposomal formulations showed an encapsulation efficacy of more than 70%; mean diameter of all the artemisinin-loaded vesicles was approximately 130-140 nm. The polydispersity index of the formulations ranged from 0.2 to 0.3 and resulted as suitable for intraperitoneal (i.p.) administration. Pharmacokinetic profile and the main pharmacokinetic parameters of the carriers were evaluated in healthy mice i.p. Free artemisinin was rapidly cleared from plasma and hardly detected 1 hour after administration. Conversely, both liposomal formulations showed much longer blood-circulation time than free artemisinin; artemisinin was **still detectable after** 3 and **24 hours** of administration, respectively, for conventional and PEGylated liposomes. AUC(0-24h) values were increased by approximately 6 times in both of the liposomal formulations, in comparison with free artemisinin. A strong effect of formulation on **the half-life of artemisinin was enhanced by more than 5-fold** by the incorporation of PEG into liposomes. Liposomes loaded with artemisinin, especially the long-circulating vesicles, could really represent a new strategy for developing **smart, well-tolerated, and efficacious** therapeutic nanocarriers **to treat tumors**, but could also be very useful to treat **parasitic disease**

Klinghardt Lyme Cocktail 2014

(The amounts suggested are for a 160 lbs person)

Equipment: 1. Blender (i.e.from Sears) 2. "Ultrasonic cleaner " (http://www.harborfreight.com/ultrasonic-cleaner-3305.html)

Foundation: tease or bait microbes out of biofilm with 4 dropperful of Hyaluronic acid sublingually twice daily (taken away from food)

The Base: Put ½ water and 1/2 olive oil into blender to barely cover the blade. Add 200-800 mg ***artemisinin***, 2-4 pipettes ***Cistus tincture***, 1 tsp of ***Lipohealth*** and 1-2 tsp of coconutoil or ghee. Blend for 10 min, then place the glass container (from blender) into water-filled ultrasound unit. Vibrate 15 minutes. Cool down in fridge until gel-like. Can be prepared for 1 week (= suggested above dose times 7). Take entire daily amount as single dose. *After 20 minutes: give binders: Clorella 8-20 tabl, ZeoClear 1-2 scoops, MicroSilica 1-2 scoops (or MetalSweep)*

Supportive Cocktail: is taken in 3 divided doses over the day. Blend items with water or juice for 10 minutes and keep cool : ***Quintessence*** (Lyme, Ehrlichia, Bartonella) 6-8 pipettes, ***Mimosa Pudica*** (worms, neuro regenerative)1/2-1 tsp., ***Czaga***: ½-1 tsp.. (anti-microbial), Oliveoil: 1 tbsp. (mycoplasma), 3-4 tbsp. colloidal silver; ***Cistus tincture*** 2-4 piptettes (biofilm), ***Cryptolepsis***: 3 pipettes(Babesia), ***LipoHealth***: 2 scoops***, D-galactose*** : 1 tsp or 5 grams (ATP), 2 pipettes Brazilian ***Green Propolis*** tincture (anti-viral, MSH, melatonin), 1 apple, pear or orange for taste and fiber. Ok to add protein powder, but not high anti-oxidant foods

In the evening give anti-oxidant mix: 1 tsp rosehip powder (ascorbates and plant adaptogens), 1 tsp. Deep Purple (anti-oxidants and vascular protective agents)

Mycoplasma responds to high olive oil diet (S.Buhner)

Olive-Oil-Derived Oleocanthal Enhances β-Amyloid Clearance as a Potential Neuroprotective Mechanism against Alzheimer's Disease: In Vitro and in Vivo Studies.

Abuznait AH, Qosa H, Busnena BA, El Sayed KA, Kaddoumi A.

ACS Chem Neurosci. 2013 Feb 25. [Epub ahead of print]

Department of Basic Pharmaceutical Science, College of Pharmacy, University of Louisiana at Monroe , 1800 Bienville Drive, Monroe, Louisiana 71201, United States.

Abstract

Oleocanthal, a phenolic component of extra-virgin olive oil, has been recently linked to reduced risk of Alzheimer's disease (AD), a neurodegenerative disease that is characterized by accumulation of β-amyloid (Aβ) and tau proteins in the brain. However, the mechanism by which oleocanthal exerts its neuroprotective effect is still incompletely understood. Here, we provide in vitro and in vivo evidence for the potential of oleocanthal to enhance Aβ clearance from the brain via up-regulation of P-glycoprotein (P-gp) and LDL lipoprotein receptor related protein-1 (LRP1), major Aβ transport proteins, at the blood-brain barrier (BBB). Results from in vitro and in vivo studies demonstrated similar and consistent pattern of oleocanthal in controlling Aβ levels. In cultured mice brain endothelial cells, oleocanthal treatment increased P-gp and LRP1 expression and activity. Brain efflux index (BEI%) studies of (125)I-Aβ(40) showed that administration of oleocanthal extracted from extra-virgin olive oil to C57BL/6 wild-type mice enhanced (125)I-Aβ(40) clearance from the brain and increased the BEI% from 62.0 ± 3.0% for control mice to 79.9 ± 1.6% for oleocanthal treated mice. Increased P-gp and LRP1 expression in the brain microvessels and inhibition studies confirmed the role of up-regulation of these proteins in enhancing (125)I-Aβ(40) clearance after oleocanthal treatment. Furthermore, our results demonstrated significant increase in (125)I-Aβ(40) degradation as a result of the up-regulation of Aβ degrading enzymes following oleocanthal treatment.

The Bee Venom peptides

(5) Lubke, L.L., and Garon, C.F.:

The Antimicrobial Agent Melittin Exhibits Powerful In Vitro Inhibitory Effects

on the Lyme Disease Spirochete.

Clinical Infectious Diseases, 1997;25 (Suppl 1):S48-51

From the Bacterial Pathogenesis Section, Rocky Mountain Laboratories Microscopy Branch, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Hamilton, Montana, USA

Abstract

Borrelia burgdorferi has demonstrated a capacity to resist the in vitro effects of powerful eukaryotic and prokaryotic metabolic inhibitors. However, treatment of laboratory cultures on Barbour-Stoenner-Kelly medium with melittin, a 26-amino acid peptide contained in honeybee venom, showed **immediate and profound inhibitory effects** when they were monitored by dark-field microscopy, field emission scanning electron microscopy, and optical density measurements.

Furthermore, at melittin concentrations as low as 100 microg/mL, virtually all spirochete motility ceased within seconds of inhibitor addition. Ultrastructural examination of these spirochetes by scanning electron microscopy revealed obvious alterations in the surface envelope of the spirochetes.

Mould exposure often creates long lasting upregulation of the immune system, neurotoxicity and chronic illness

Diagnosis:

- TGF beta 1 elevated
- High home mold ERMI score from Mycometrics.com
- Urine excretion of mycotoxins (expensive test)
- Mold antibody testing
- Mold allergy testing in the skin (+ watching for reactions –AAEM protocols)
- C4a high, C3a normal = mold
- C4a high, C3a high = Lyme
- ART testing
- www.SurvivingMold.com (R.Shoemaker MD)

The Mycotoxin Patulin Alters the Barrier Function of the Intestinal Epithelium: Mechanism of Action of the Toxin and Protective Effects of Glutathione R. Manfoud et al.; Toxicology and Applied Pharmacology, 181:209-218 (2002)

Abstract:

The active site of PTP contains a cysteine residue (Cys215) that is essential for phosphatase activity. Sulfhydryl-reacting compounds such as acetaldehyde decrease TER through covalent modification of Cys215 of PTP. We propose that the toxicity of patulin for intestinal cells involves, among other potential mechanisms, an inactivation of the active site of PTP.

Lipoic acid as a potential first agent for protection from mycotoxins and treatment of mycotoxicosis

S. A. Rogers; Arch Environ Health, 58(8):528-532, August 2003

Abstract: Mycotoxins - toxic substances produced by fungi or molds - are ubiquitous in the environment and are capable of damaging multiple biochemical mechanisms, resulting in a variety of human symptoms referred to collectively as “mycotoxicosis.” In fact, mycotoxins mimic multiple xenobiotics, not only with respect to their ultimate damage, but also in their routes of detoxification. This suggests potential therapeutic options for the challenging treatment of mycotoxicosis. In this brief review, the author examines the use of lipoic acid as an example of an inexpensive and available nutrient that has been shown to protect against, or reverse, the adverse effects of mycotoxins.

- **Our findings**: Chlorella, MicroSilica, OSR, S-Acetyl-Glutathione, DMPS, DMSA, DL-Methionine, LipoHealth and Phospholipid Exchange and other agents used for metal detox also are potent mycotoxin elimination agents
- **Dr. Ritchie Shoemaker** recommends the use of Cholestyramine 4 grams 4 times per day (or Welchol) before, during and after mould exposure. He prefers to premedicate the client with Actos to prevent the feared cytokine storm caused by mobilizing mycotoxins. When people do not tolerate Actos he recommends 240 mg EPA/180 mg

How does the brain keep itself clean?

- **Mechanics**: The pumping action of the brains lymphatic system (glymphatic system) motored by both the cranial rhythm (rhythmic fluid production and drainage) and by chewing (rhythmic stretching of the brain's membrnes
- **Biochemistry**: melatonin is the most important housekeeping molecule, anti oxidant and detox agent for mercury lead and other molecules. Orally taken melatonin does not enter the brain unless it is prepared liposomally. Olive oil clears amyloid
- **Immunological**: macrophages also clear tissues of metals and other toxins
- *Sener, G.et al: **"Melatonin protects against mercury induced oxidative tissue damage**". Basic and Clinical Pharmacology&Toxicology Vol 93, Dec 2003, pp 290-296*
- L. Xie, H. Kang, Q. Xu, M. J. Chen, Y. Liao, M. Thiyagarajan, J. O'Donnell, D. J. Christensen, C. Nicholson, J. J. Iliff, T. Takano, R. Deane, M. Nedergaard. ***"Sleep Drives Metabolite Clearance from the Adult Brain"***. *Science*, 2013; 342 (6156): 373 DOI:10.1126/science.1241224
- Glutathione may only be second best. To increase reduced glutathione in the brain NAC works, i.v injected glutathione only if it is offered liposomally

The Glymphatic System clears the brain during the night

- With the pumping action and rhythm of the CSF in the brain, the glymphatic system flushes the waste from your brain back into your body's circulatory system. From there, the waste eventually reaches your liver, where it's ultimately eliminated.
- This system ramps up its activity *during sleep*, thereby allowing your brain to clear out toxins, including harmful proteins called amyloid-beta, the buildup of which has been linked to Alzheimer's.
- During sleep, the glymphatic system becomes 10 times more active than during wakefulness. Simultaneously, your brain cells shrink by about 60 percent, allowing for greater efficiency of waste removal.
- During the day, the constant brain activity causes your brain cells to swell in size until they take up just over 85 percent of your brain's volume, thereby disallowing effective waste removal during wakefulness

NIH/National Institute of Neurological Disorders and Stroke. "Brain may flush out toxins during sleep; Sleep clears brain of molecules associated with neurodegeneration: Study." ScienceDaily. ScienceDaily, 17 October 2013. <www.sciencedaily.com/releases/2013/10/131017144636.htm>.

Melatonin clears the brain at night of toxins
It is the most potent brain anti-oxidant and detox agent

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

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

- *Sener, G.et al: **"Melatonin protects against mercury induced oxidative tissue damage"**. Basic and Clinical Pharmacology&Toxicology Vol 93, Dec 2003, pp 290-296*
- L. Xie, H. Kang, Q. Xu, M. J. Chen, Y. Liao, M. Thiyagarajan, J. O'Donnell, D. J. Christensen, C. Nicholson, J. J. Iliff, T. Takano, R. Deane, M. Nedergaard. ***"Sleep Drives Metabolite Clearance from the Adult Brain"***. *Science*, 2013; 342 (6156): 373 DOI:10.1126/science.1241224