Retroviruses, prions and the synergy of mercury, lead, aluminum and agrochemicals: new insights, new approaches and improved outcomes (for affected children, teenagers and adults)

Dietrich Klinghardt MD, PhD www.KlinghardtInstitute.com May 2019

7 simple, science-based interventions to improve the life of an autistic person:

1. Remove fluoride, aluminum, mercury and glyphosate (from all organs and tissues)
2. Reduce electromagnetic radiation exposure wherever possible
3. Treat parasites, mold, Lyme and Co, retroviruses
4. Silence infectious proteins and amyloid
5. Biomedical approach: reduce remaining brain and gut inflammation, replace nutrients
6. Brain entrainment techniques: biofeedback, exercise, colour and sound
7. Familysystems oriented psychotherapy (constellation work)

The Klinghardt APN-Method* to Help the Recovery of Autistic Persons (www.KlinghardtInstitute.com)

The Method follows 7 steps (developed in 45 years of practice under the influence of extensive lab testing, imaging and EEG studies - and ART)

1. **Open the lymphatic and venous drainage of the brain**
2. **Biophysical stress: protect from EMR and geopathic influences**
3. **Remove key toxins: glyphosate, mercury, aluminum, and lead**
4. **Remove key pathogens (parasites, mold, Lyme and co, viruses, prions)**
5. **Mitigate adverse immune reactions: gut repair, LDA/LDI, quercetin, frankincense, CBD, GliLia (luteolin and PEA), Sophia Flow cream**
6. **Replace missing nutrients (BioMedical Approach)**
7. **Heal the psychological environment: Family Constellation work**

APN: **Applied **P**sycho**N**eurobiology*

Before anything else: Improve the mechanics of brain health!

Autism most often results of both epigenetic and intra-uterine influences (toxicity, infections and mom's own anti-bodies, that cross react with CNS tissues of the fetus) and other insults in the first 2 years of life. How does the brain keep itself clean and healthy - or how does it fail?

Neurochem Res. 2015 Dec;40(12):2583-99. doi: 10.1007/s11064-015-1581-6. Epub 2015 May 7.

The Glymphatic System: A Beginner's Guide.

Jessen NA[1], Munk AS[2], Lundgaard I[2], Nedergaard M[2].

Author information

Abstract

The glymphatic system is a recently discovered macroscopic waste clearance system that utilizes a unique system of perivascular tunnels, formed by astroglial cells, to promote efficient elimination of soluble proteins and metabolites from the central nervous system. Besides waste elimination, the glymphatic system also facilitates brain-wide distribution of several compounds, including glucose, lipids, amino acids, growth factors, and neuromodulators. Intriguingly, the glymphatic system function mainly during sleep and is largely disengaged during wakefulness. The biological need for sleep across all species may therefore reflect that the brain must enter a state of activity that enables elimination of potentially neurotoxic waste products, including β-amyloid. Since the concept of the glymphatic system is relatively new, we will here review its basic structural elements, organization, regulation, and functions. We will also discuss recent studies indicating that glymphatic function is suppressed in various diseases and that failure of glymphatic function in turn might contribute to pathology in neurodegenerative disorders, traumatic brain injury and stroke.

KEYWORDS: Aging; Astrocytes; Cerebrospinal fluid secretion; Neurodegenerative diseases; Perivascular spaces; Sleep; The glymphatic system; Traumatic brain injury; Virchow–Robin spaces

The Glymphatic System Clears the Brain During the Night, but Only in Deep Delta Sleep

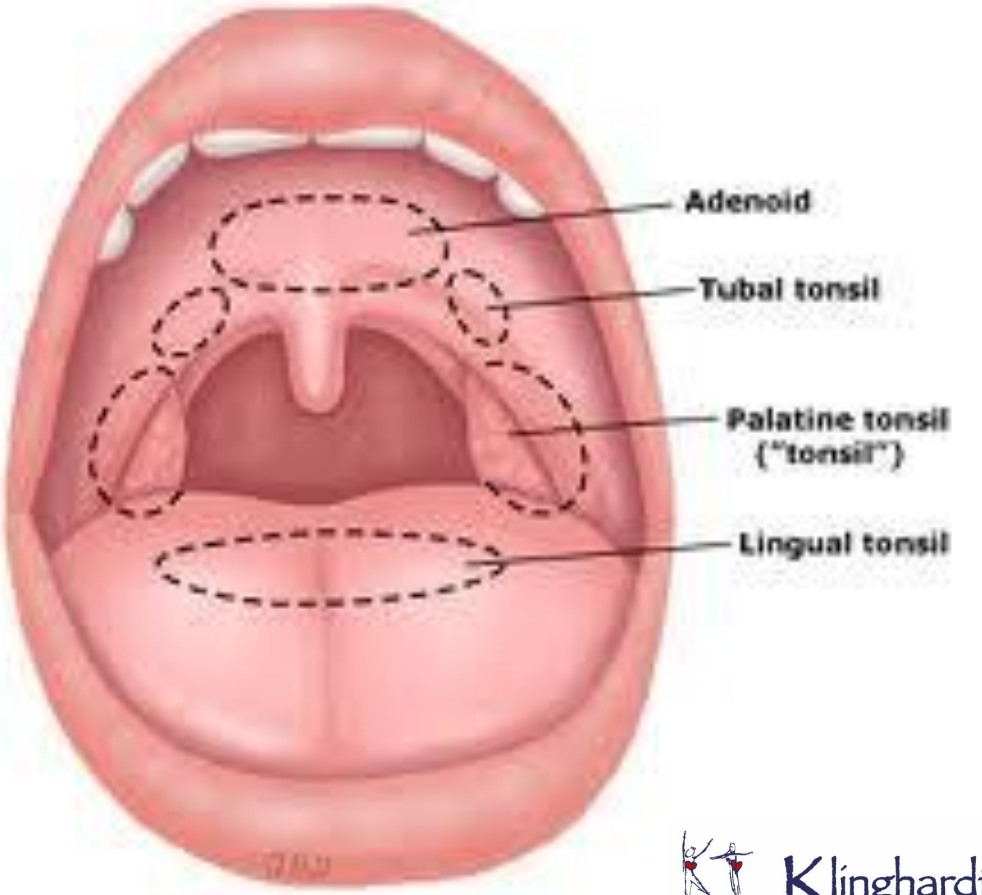
- By pumping cerebral spinal fluid through your brain's tissues, the glymphatic system flushes the waste from your brain back into your body's circulatory system. From there, the waste eventually reaches your liver, where it's ultimately eliminated.
- This system ramps up its activity *during sleep*, thereby allowing your brain to clear out toxins, including harmful proteins called amyloid-beta, the build-up 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,_(8) thereby disallowing effective waste removal during wakefulness

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

2 exit-routes for brain waste **1: Brain- and Neck Lymphatics**

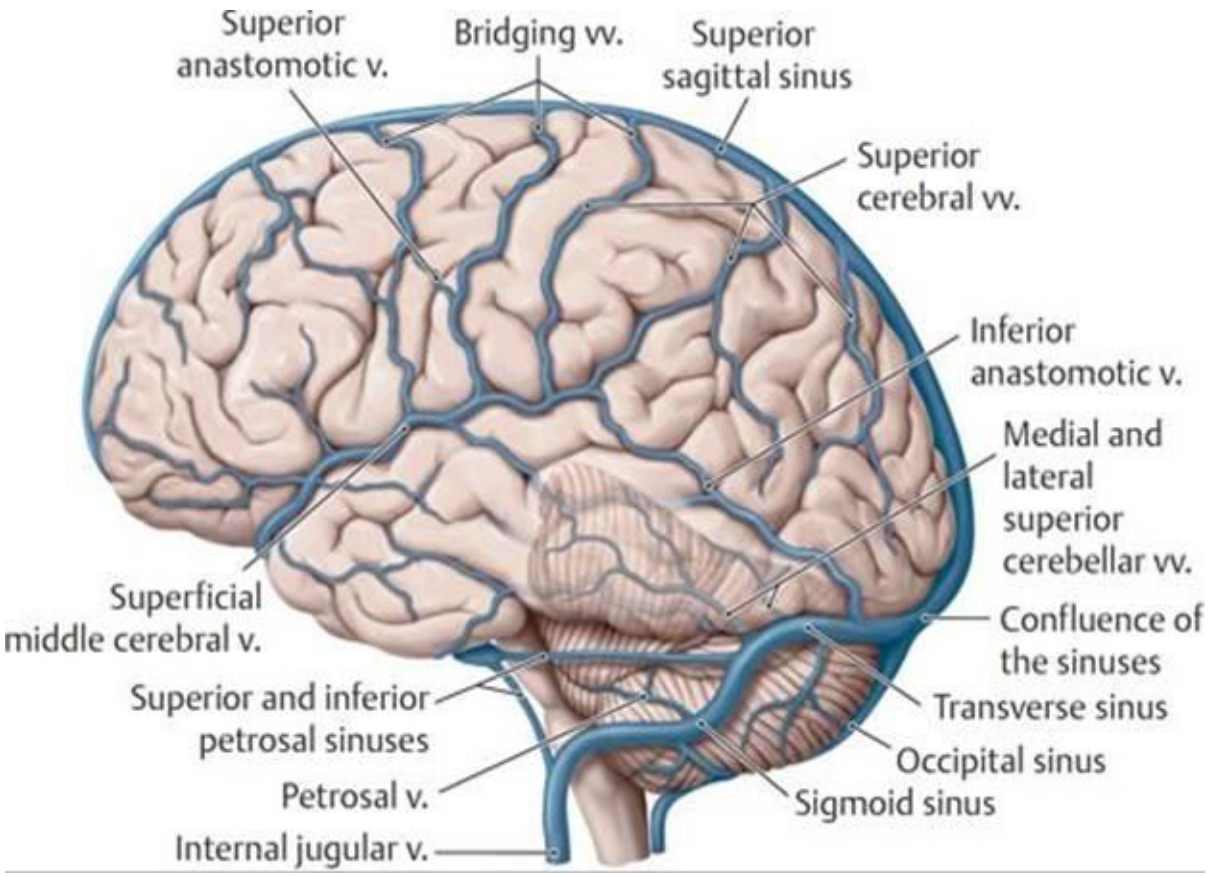
a. direct lymphatic drainage through the cribriform plate to Waldeyer's Ring (adenoids – tonsils – anterior neck lymph nodes – deep neck veins – research from Russia)
b. lymphatics alongside the veins inside the brain and along the neck veins, eventually dumping the content into the veins

- Waldeyer's ring is an annular ring of lymphatic tissue formed by the:
  - Pharyngeal tonsil ("Adenoids")
  - Two tubal tonsils-posterior to Eustachian tu
  - Two palatine tonsils ("Tonsils")
  - Lingual tonsil-base of tongue
  - Laryngeal tonsil-near the vocal cords in the larynx
- Protection against pathogens and toxins
- Lymphatic detox/excretion organ
- Greatest immune modulator

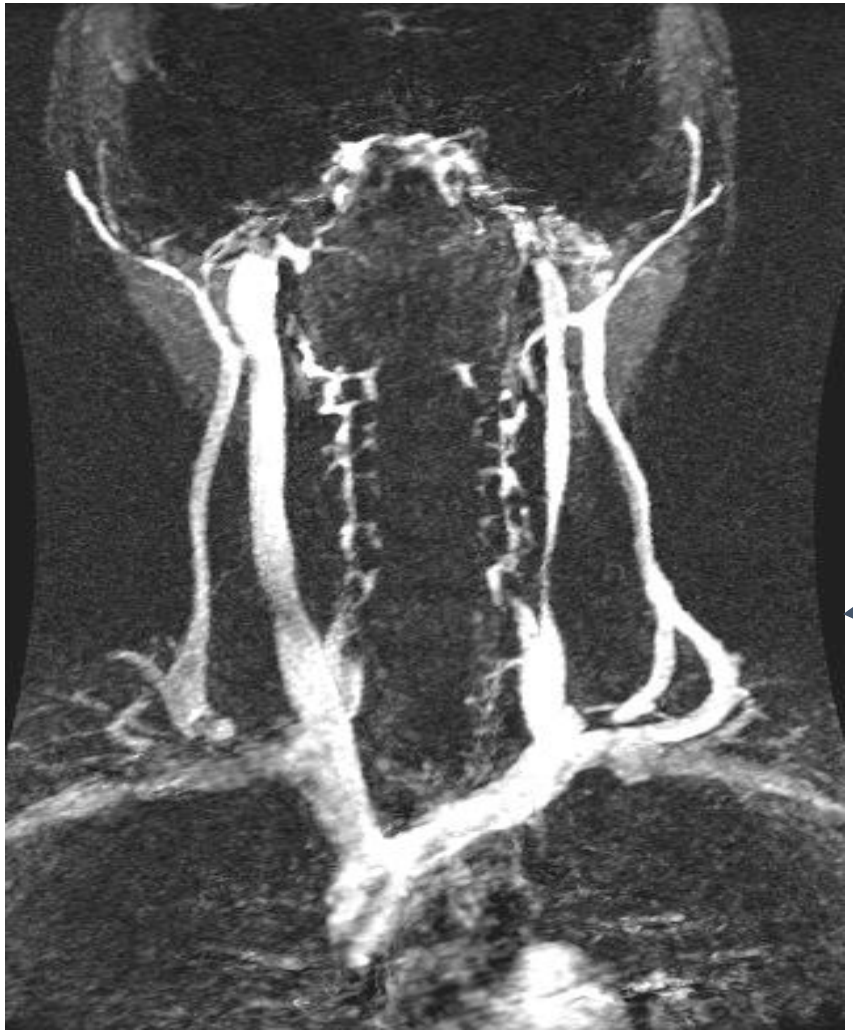


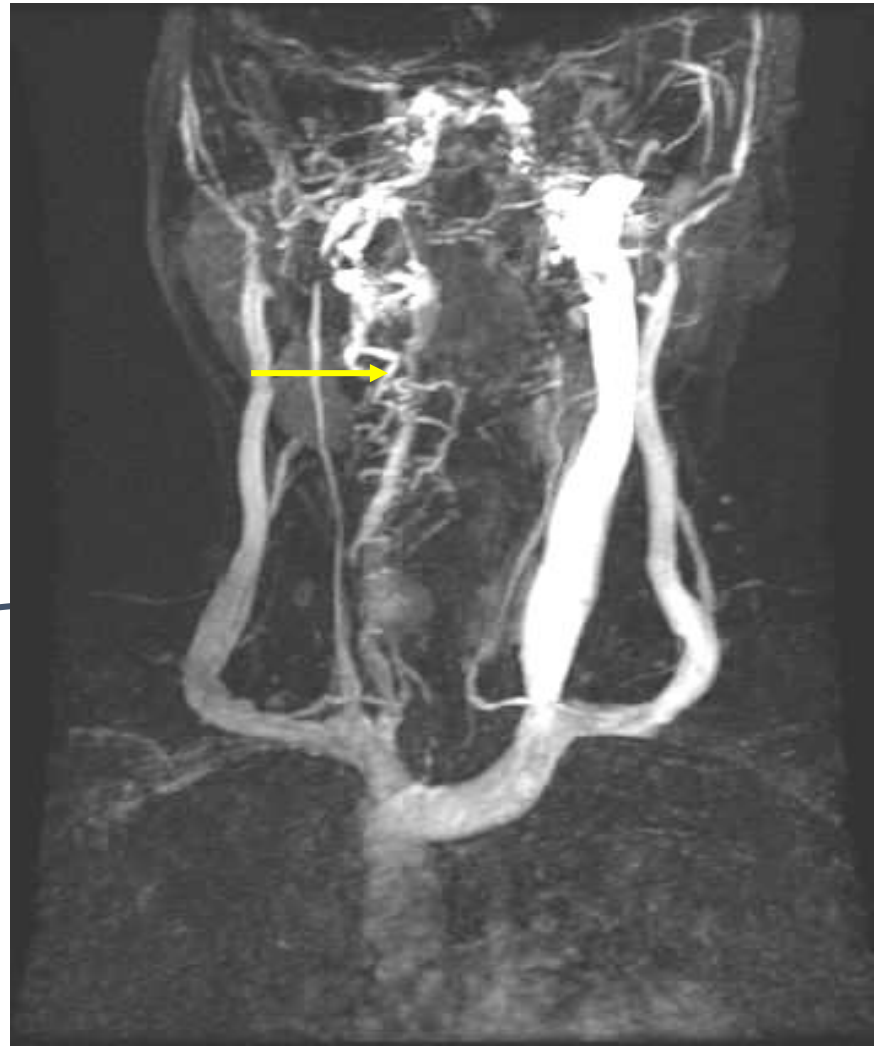
2. Neck Veins: Venous Drainage of the Brain

- The blood exits the brain via the internal and external jugular veins
- The glymphatic system piggybacks on the blood vessels in your brain- if the venous drainage is compromised, it impairs lymph drainage of the brain
- In autistic kids and adults with neurological illness the endothelium of these veins is covered with a lawn of pathogens (Rickettsia, mold, viruses, Bartonella, etc.)
- The immune reactions triggered by the pathogens causes inflammation and narrowing of the veins – and both decreased brain blood flow and lymph drainage (chronic cerebro-spinal venous insufficiency or CCSVI)



Left: Stenosis at the stump of the LIJV with collateral input from the vertebral system (32 year old autistic man)
Right: String like jugular in the RIJV (19 year old autistic man)





Antonucci, N., S. Pacini, and M. Ruggiero. "Clinical Experience of Integrative Autism treatment with a Novel type of Immunotherapy." *Madridge J Vaccines* 3.1 (2019): 71-76.

Abstract: In this study, we report the results associated with a novel type of immunotherapy based on **a supplement containing low-molecular-weight chondroitin sulfate, phosphatidylcholine, and vitamin D3**. In October 2018, the Biomedical Centre for Autism Research and Treatment of Bari, Italy, started using this approach on autistic subjects. By January 2019, several scores of patients have been treated; here, we describe the cases of three autistic patients for whom the treatment was remarkably effective. In order to obtain the most objective and complete evaluation of results, we used the Autism Treatment Evaluation Scale (ATEC), a diagnostic assessment tool that has been demonstrated effective in evaluating interventional effects in autism. Our observations indicate that some of the most representative symptoms of autism were completely normalized after eight weeks of treatment and other showed a trend toward normalization. No adverse effects were reported. Based on the cases described in this report and on our ongoing research, we are convinced that this type of supplement-based immunotherapy can be beneficial to autistic patients and represents a new and promising treatment. We expect that the described approach will play a central role in future treatments for autism, both alone and in combination with other therapies such as behavioral therapies, nutritional interventions or **manual lymphatic massage**.

Keywords: Autism; GcMAF; Immune system;

Neuroinflammation; Immunotherapy.

Citation: Marco Ruggiero., et al. "Clinical Experience of Integrative Autism Treatment with Manual Lymphatic Drainage". EC Neurology 11.1 (2019): 21-28.

Corresponding Author: Marco Ruggiero, Silver Spring Sagl, Arzo-Mendrisio, Switzerland

Abstract: In this study we report the results of a protocol for improving brain lymphatic flow in autism through lymphatic drainage massage, a technique successfully used in a variety of conditions where **intracranial lymphatic circulation is hampered by obstacles at the level of deep cervical nodes.** At the end of May 2018, the Biomedical Centre for Autism Research and Treatment started implementing a protocol of manual lymphatic drainage of the deep cervical nodes on autistic subjects. By October 2018, several scores of patients had been treated with this protocol. In this report, we describe the cases of three autistic patients for whom manual lymphatic massage was remarkably effective. To our knowledge, this is the first report of lymphatic drainage massage at the level of the deep cervical nodes in autism. **Symptomatic improvement was robust and we attribute these results to the effects of the massage on the intracranial lymph or sometimes referred to as the glymphatic circulation with improvement of brain lymphatic drainage believed leading to a decrease of neuroinflammation.** In addition to stimulating lymphatic drainage, we postulate that the protocol may serve also as vagus nerve stimulation. The protocol also targets the larynx in a manner similar as described for laryngeal manual therapy for the treatment of dysphonia, and this factor may be contributing to the overall improvement of symptoms, with particular reference to speech. Based on the cases described in this report and on our ongoing research, we are convinced that this type of inexpensive, harmless and easy-to-implement approach of manual lymphatic drainage can be beneficial to autistic patients and represents a new and promising treatment. We expect that the described protocol will play a central role in future treatments for autism, both alone and in combination with other therapies such as behavioral therapies or nutritional interventions.

Keywords: Autism; Brain Lymphatic System; Massage; Vagus Nerve; Larynx

In this clinical study we could show, that when ART finds dysfunction in an organ or tissue, ultrasound can confirm the dysfunction in all cases. ART is a reliable tool to detect tissue dysfunction, establish an accurate diagnosis and effective treatment protocols

The Ruggiero-Klinghardt (RK) Protocol for the Diagnosis and Treatment of Chronic Conditions with Particular Focus on Lyme Disease

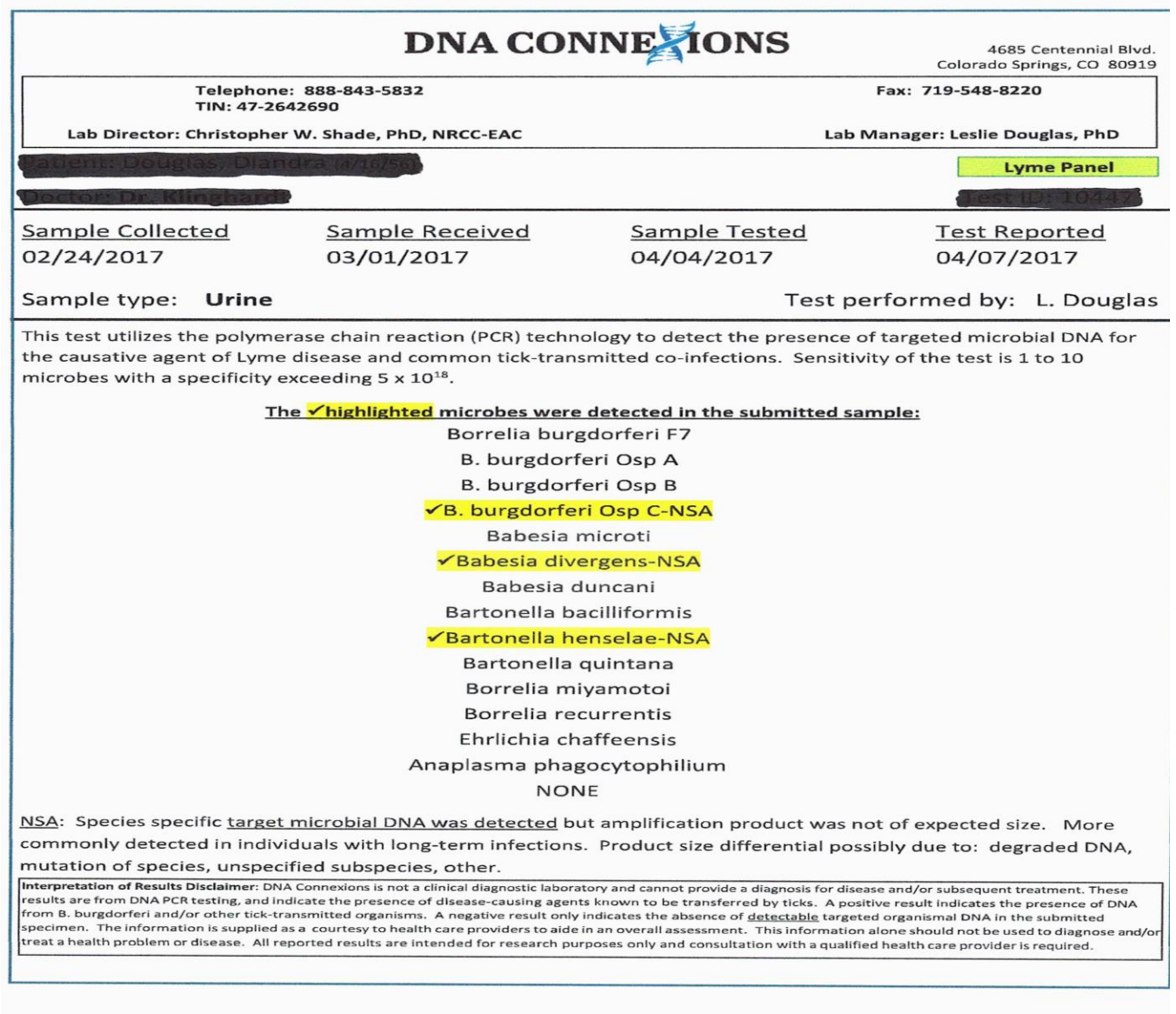
American Journal of Immunology, March 2017

Dietrich Klinghardt and Marco Ruggiero

Abstract: Here we describe the Ruggiero-Klinghardt (RK) Protocol that is based on **integration of Autonomic Response Testing (ART) with diagnostic ultrasonography** and on application of therapeutic ultrasounds; the latter are used as a provocation tool and as an instrument to optimize drug uptake and utilization in specific areas of the body. This protocol consists of a precise sequence of diagnostic and therapeutic procedures with the ultimate goal of improving sensitivity and specificity of diagnosis at the same time evaluating and optimizing efficacy of treatments in chronic conditions including, but not limited to, persistent Lyme disease. The RK Protocol represents **a paradigm shift in diagnostics and therapeutics: Thus, compartmentalized microbes, transformed cells, toxins and metabolites could be detected using a safe and non-invasive method**. In addition, the RK Protocol allows optimization of efficacy of drugs and other therapeutic interventions. Although the RK Protocol was initially developed for persistent Lyme disease, it shows significant potential in conditions ranging from cancer to neurodegenerative diseases and autism. In oncology, the RK Protocol may serve to facilitate early diagnosis and to increase sensitivity of cancer cells to the killing effects of a variety of remedies ranging from conventional radio- and chemotherapy to more recent forms of immunotherapy. Thus, the 1st goal of the RK Protocol is diagnostic: That is, to make pathogens, toxins, transformed cells and cells infected by viruses that are inaccessible to conventional diagnostic and therapeutic tools, “visible” to the therapist who can detect them with laboratory methods and deal with them with appropriate interventions; and also to make them “visible” to the immune system that can fight them in a physiological manner. The 2nd goal is to optimize drug uptake and utili: tissues studied and targeted with these procedures.

Keywords: Lyme, Ultrasound, Autonomic Response Testing, Immune System, Imaging, Brain

25 year old autistic woman with POTS and severe fatigue and brain fog. She tested negative with the Western Blot and appropriate IgG/IgM tests, Immunfluorescence - but positive for all 3 pathogens with ART



DNA CONNEXIONS

4685 Centennial Blvd.
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Telephone: 888-843-5832
TIN: 47-2642690
Fax: 719-548-8220

Lab Director: Christopher W. Shade, PhD, NRCC-EAC
Lab Manager: Leslie Douglas, PhD

[redacted]
Lyme Panel
[redacted]

Sample Collected	Sample Received	Sample Tested	Test Reported
02/24/2017	03/01/2017	04/04/2017	04/07/2017

Sample type: **Urine**
Test performed by: L. Douglas

This test utilizes the polymerase chain reaction (PCR) technology to detect the presence of targeted microbial DNA for the causative agent of Lyme disease and common tick-transmitted co-infections. Sensitivity of the test is 1 to 10 microbes with a specificity exceeding 5×10^{18}.

The ✓highlighted microbes were detected in the submitted sample:

Borrelia burgdorferi F7
B. burgdorferi Osp A
B. burgdorferi Osp B
✓B. burgdorferi Osp C-NSA
Babesia microti
✓Babesia divergens-NSA
Babesia duncani
Bartonella bacilliformis
✓Bartonella henselae-NSA
Bartonella quintana
Borrelia miyamotoi
Borrelia recurrentis
Ehrlichia chaffeensis
Anaplasma phagocytophilium
NONE

NSA: Species specific target microbial DNA was detected but amplification product was not of expected size. More commonly detected in individuals with long-term infections. Product size differential possibly due to: degraded DNA, mutation of species, unspecified subspecies, other.

Interpretation of Results Disclaimer: DNA Connexions is not a clinical diagnostic laboratory and cannot provide a diagnosis for disease and/or subsequent treatment. These results are from DNA PCR testing, and indicate the presence of disease-causing agents known to be transferred by ticks. A positive result indicates the presence of DNA from B. burgdorferi and/or other tick-transmitted organisms. A negative result only indicates the absence of detectable targeted organismal DNA in the submitted specimen. The information is supplied as a courtesy to health care providers to aide in an overall assessment. This information alone should not be used to diagnose and/or treat a health problem or disease. All reported results are intended for research purposes only and consultation with a qualified health care provider is required.

Klinghardt Institute
The Heart Of Healing
www.KlinghardtInstitute.com

American Journal of Immunology

Original Research Paper

Tailoring the Ruggiero-Klinghardt Protocol to Immunotherapy of Autism

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Article history Received: 02-03-2019 Revised: 17-04-2019 Accepted: 24-04-2019

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Abstract: Here, we describe the adaptation to the field of autism of an original procedure denominated the "Ruggiero-Klinghardt Protocol" (RK Protocol), a procedure that represents a paradigm change with significant implications for chronic conditions where immunotherapy may prove effective; that is from silent infections to neurodegenerative diseases, autism and cancer. In the context of autism, the modified RK Protocol that we propose here serves the purpose of discovering hidden infections that may be associated with autism and contribute to its symptoms. This notion is consistent with the observation that immune modulating molecules are effective in autism treatment. In the RK Protocol modified for autism, we introduce the Autism Treatment Evaluation Checklist (ATEC), a more objective and sophisticated method of evaluation compared with the Clinical Global Impression of Improvement scale that was previously used, independently of the RK Protocol, to evaluate the effectiveness of immunotherapy of autism. The modifications that we present in this study take advantage of the experience that has accumulated in the two years after the publication of the original RK Protocol. Similarly to the original RK Protocol, this new version offers the advantage of being safe and relatively inexpensive since it does not require sophisticated instruments; because of this, it can be implemented in different parts of the world. We envisage that implementation of the RK Protocol modified for autism may contribute to decrease the burden of the disease as it enables prevention, early diagnosis and treatment on a large scale.

Keywords: Ultrasound, Autonomic Response Testing, Immune System, Imaging,

Neuroinflammation, Autism

The reported low levels of certain micronutrients (glutathione, sulfate, methyl folate, etc.) are secondary to the incorporation of homotoxins into our genome, proteome and metabolome. The reported disturbances in the immune system and presence of pathogens are secondary to microwave stress and the presence of toxins in our system, not primary. Toxins and pathogens depend on each other and are synergistic. There are only 4 main culprits.

Toxin #1: **Fluoride** (or: why is the US the sickest country amongst Western nations?)

Strunecka, Anna, Russell L. Blaylock, and Otakar Strunecky. "Fluoride, aluminum, and aluminofluoride complexes in pathogenesis of the autism spectrum disorders: A possible role of immunoexcitotoxicity." *Journal of Applied Biomedicine* 14.3 (2016): 171-176.Abstract

Autism spectrum disorders (ASD) are characterized by impairments in social interaction and communication along with stereotyped patterns of behaviors. Over the past several decades, the prevalence of ASD has increased dramatically. ASD are highly multifactorial, with many risk factors acting together. Our review suggests that most risk factors are connected to immunoexcitotoxicity. Fluoride exposure is common as a result of the artificial fluoridation of drinking water and a dramatic increase in the volume of man-made industrial fluoride compounds released into the environment. Human exposure to environmental aluminum is extensive and appears to be growing. The long-term fluoride and aluminum burden have several health effects with a striking resemblance to the ASD. Moreover, both fluoride and aluminum interfere with a number of glycolytic enzymes, resulting in a significant suppression of cellular energy production. **The synergistic interactions of fluoride and aluminum increase the potential neurotoxic effect particularly in children**. Aluminofluoride complexes have effects on cell signaling, neurodevelopment, and neuronal function.

We suggest that the burden with these new ecotoxicological factors could contribute to an alarming increase in the prevalence of ASD.

Czajka, Michael. "Systemic effects of fluoridation." *Journal of Orthomolecular Medicine* 27.3 (2012): 123.

Abstract This review article is written from a food chemistry perspective. It focuses on the systemic effects of fluoride (rather than the effects of fluoride on the teeth) since fluoride research concentrates largely on the teeth to the virtual exclusion of systemic effects. This is surprising given that fluoride is a known systemic toxin. About 400 million people (~6% of the world's population) drink fluoridated water. **The effect of fluoride on the teeth is topical (directly on the teeth) and not systemic, so drinking fluoridated water has no benefit**. Fluoride is a **lipid soluble neurotoxin and enzyme poison**. Fluoride accumulates in the pineal gland (9,000 ppm on average) and bone. Dental fluorosis is a marker for skeletal fluorosis. At 1 ppm 32 % of US children have dental fluorosis. At 1 ppm some sections of the population (e.g. infants) will ingest too much fluoride. Unfluoridated and fluoridated countries have similar rates of tooth decay. Given that fluoridation of water supplies is not necessary to maintain a reduction in tooth decay and that the side effects of ingestion are undesirable, the practice is likely to come under increasing scrutiny. More studies on the systemic effects of fluoride are urgently required.

… mostly affected by fluoride include the brain(12,13) thyroid, parathyroid and adrenal glands (14)
and … and discourage swallowing; otherwise, their children might ingest too much for their body
weight.39 … **Fluoride accumulates in the pineal gland**

(9,000 ppm on average) and bone …

The Early Studies

Luke, J. A. (1997). *The effect of fluoride on the physiology of the pineal gland* (Doctoral dissertation, University of Surrey).

The purpose was to discover whether fluoride (F) accumulates in the pineal gland and thereby affects pineal physiology during early development. The [F] of 11 aged human pineals and corresponding muscle were determined using the F-electrode following HN4DS/acid diffusion. The rnýan [F] of pineal was significantly higher ($p < 0.001$) than muscle: 296 ± 257 vs. 0.5 ± 0.4 mg/kg respectively. Secondly, a controlled longituUinal experimental study was carried out to discover whether F affects the biosynthesis of melatonin, (MT), during pubertal development using the excretion rate of urinary 6-sulphatoxymelatonin, (aMT6s), as the index of pineal MT synthesis. Urine was collected at 3- hourly intervals over 48 hours from two groups of gerbils, (Meriones unguiculatus), low-F (LF) and high-F (IHF) (12 f, 12 m/group): under LD: 12 12, from prepubescence to reproductive maturity (at 9-12 weeks) to adulthood, i. e., at 7,9,11 V2and 16 weeks. The I-IF pups received 2.3 gg F/g BW/day from birth until 24 days whereafter BF and LF groups received food containing 37 and 7 mg F/kg respectively and distilled water. Urinary aMT6s levels were measured by radioimmunoassay. The BF group excreted significantly less aMT6s than the LF group until the age of sexual maturation. At 11 V2weeks, the circadian profile of aMT6s by the BF males was significantly diminished but, by 16 weeks, was equivalent to the LF males. In conclusion, F inhibits pineal MT synthesis in gerbils up until the time of sexual maturation. Finally, F was associated with a significant acceleration of pubertal development in female gerbils using body weights, age of vaginal opening and accelerated development of the ventral gland. At 16 weeks, the mean testes weight of BF males was significantly less ($p < 0.002$) than that of the LF males.

The results suggest that **Fluoride is associated with low circulating levels of Melatonin** and this leads to accelerated sexual maturation in female gerbils. The results strengthen the hypothesis that the pineal has a role in

pubertal development.

Fluoride "may" result in autism and lowers the IQ
(i.e: it affects and damages the entire brain)

Nakamoto, Tetsuo, and H. Ralph Rawls. "Fluoride exposure in early life as the possible root cause of disease in later life." *Journal of Clinical Pediatric Dentistry* 42, no. 5 (2018): 325-330. Fluoride, one of the most celebrated ingredients for the prevention of dental caries in the 20th century, has also been controversial for its use in dentifrices and other applications. In the current review, we have concentrated primarily on early-life exposure to fluoride and how it may affect the various organs. The most recent controversial aspects of fluoride are related to **toxicity of the developing brain and how it may possibly result in the decrease of intelligence quotient (IQ), autism, and calcification of the pineal gland.** In addition, it has been reported to have possible effects on bone and thyroid glands. If nutritional stress is applied during a critical period of growth and development, the organ(s) and/or body will never recover once they pass through the critical period. For example, if animals are force-fed during experiments, they will simply get fat but never reach the normal size. Although early-life fluoride exposure causing fluorosis is well reported in the literature, the dental profession considers it primarily as an esthetic rather than a serious systemic problem. In the current review, we wanted to raise the possibility of **future disease as a result of early-life exposure to fluoride**. It is not currently known how fluoride will become a cause of future disease. Studies of other nutritional factors have shown that the effects of early nutritional stress are a cause of disease in later life.

Ramesh, M., Aruna, R. M., Malathi, N., & Krishnan, R. (2014). A Review of fluoride and its diverse effects. *SRM Journal of Research in Dental Sciences*, *5*(1), 42. Increased intake of fluoride in water and diet results in dental and skeletal fluorosis. Many states in India are affected by fluorosis. The optimum level of fluoride in drinking water for anti-cariogenic effect was thought to be 1 ppm. Various effects of fluoride on plants, animals and humans are discussed here. Currently, it is identified that **fluoride has significant role in gene polymorphisms and lowered intelligent quotient.**

Fluoride and Aluminium also have a Synergism in Crippling our Children

Strunecka, Anna, et al. "Fluoride interactions: from molecules to disease." *Current Signal Transduction Therapy* 2.3 (2007): 190-213.

Abstract

Fluoride has long been known to influence the activity of various enzymes in vitro. Later it has been demonstrated that many effects primarily attributed to fluoride are caused by synergistic action of fluoride plus aluminum. Aluminofluoride complexes have been widely used as analogues of phosphate groups to study phosphoryl transfer reactions and heterotrimeric G proteins involvement. A number of reports on their use have appeared, with far-reaching consequences for our understanding of fundamental biological processes. **Fluoride plus aluminum send false messages**, which are amplified by processes of signal transduction. Many investigations of the longterm administration of fluoride to laboratory animals have demonstrated that fluoride and **aluminofluoride** complexes can **elicit impairment of homeostasis, growth, development, cognition, and behavior.** Ameliorative effects of calcium, vitamins C, D, and E have been reported. Numerous epidemiological, ecological, and clinical studies have shown the effects of fluoride on humans. Millions of people live in endemic fluorosis areas. A review of fluoride interactions from molecules to disease is necessary for a sound scientific assessment of health risks, which may be linked to the chronic intake of small doses of fluoride and aluminum from environmental and artificial sources.

How to clear fluoride from the brain

- **RK protocol and manual brain compressions** “SophiaFlow cream”daily (connect at the Booth for Bravo Yoghurt to get on the waiting list. Brief video will be at: www.KlinghardtInstitute.com).
- Stop drinking US water, unless you know the source. Get the best fluoride removing water filter (“Berkey” or RO, but only if it drains into an open tank)
- Rao, Mandava V., and Hemlata Tiwari. "Amelioration by **melatonin** of chromosomal anomalies induced by arsenic and/or **fluoride** in human blood lymphocyte cultures." *Fluoride* 39.4 (2006): 255.
- Kao, Wei-Fong, Jou-Fang Deng, Shu-Chiung Chiang, Kennon Heard, David HT Yen, Mei-Chun Lu, Benjamin Ing-Tiau Kuo, Chien-Chun Kuo, Tsung-Yun Liu, and Chen-Hsen Lee. "A simple, **safe, and efficient way to treat severe fluoride poisoning**—oral calcium or magnesium." *Journal of Toxicology: Clinical Toxicology* 42, no. 1 (2004): 33-40.
- Heard, Kennon, et al. "Calcium neutralizes fluoride bioavailability in a lethal model of fluoride poisoning." *Journal of Toxicology: Clinical Toxicology* 39.4 (2001): 349-353.

After mobilizing Fluoride and aluminum from the tissues with the RK protocol, the toxins are eliminated via the biliary system. To provent re-absorption, binders have to be lining the intestinal tract: **chlorella and zeolite**

Hiremath, Poornima G., and Thomas Theodore. "Modelling of Fluoride Biosorption by Calcium-doped Algae Using Response Surface Methodology." *Indian Chemical Engineer* 60.1 (2018): 37-57.
The present investigation deals with the biosorption of fluoride from aqueous solutions using calcium-doped Chlorella vulgaris, Chlorella protothecoides, and Nannochloropsis oculata. Effects of the factor variables (initial fluoride concentration, pH, biosorbent dose, and contact time) and their interactions on sorption of fluoride ion were investigated by response surface methodology based on central composite design. **Maximum fluoride biosorption of 78.7% was obtained** from calcium-doped ***C. vulgaris*** **biomass**. The fluoride biosorption followed pseudo-second-order kinetics. The fluoride sorption capacity was found to be 7.554, 7.549, and 7.827 mg/g for C. vulgaris, C. protothecoides, and N. oculata species, respectively. The biosorbent was characterised using scanning electron microscope with energy-dispersive spectroscopy and Fourier transform infrared. This study provides the efficiency of modified algal biomass for biosorption of fluoride. The positive results encourage extending the usage of the naturally abundant or waste algal biomass to remove other toxic heavy metals from the environment.

Hiremath, Poornima G., and Thomas Theodore. "**Biosorption of Fluoride** from Synthetic and Ground Water Using **Chlorella vulgaris** Immobilized in Calcium Alginate Beads in an Upflow Packed Bed Column." *Periodica Polytechnica Chemical Engineering* 61.3 (2017): 188-199.

Pitre, Danaé, Amiel Boullemant, and Claude Fortin. "**Uptake and sorption of aluminium and fluoride** by four green algal species." *Chemistry Central Journal* 8.1 (2014): 8.

Lava Vitae is a high Silica zeolite (klinoptiolite): **Zeolite removes Fluoride**

Structural forms of fluorides in bone tissue of animals under chronic fluoride intoxication Journal of Structural Chemistry
March 2006, Volume 47, Issue 2,
pp 258-266
S. P. Gabuda, A. A. Gaidash,S. G. Kozlova, N. L. Allan

Abstract

High-resolution solid-state ^{19}F NMR method has been used to examine the chemical structural forms of fluorine introduced into the bone tissue of experimental animals (Vistar rats) exposed to 30 day fluoride inhalation. It is shown that in the bone tissue three structural forms of fluorides are deposited: a solid phase of F-apatite and mobile nanoparticles of CaF_2 and MgF^2 (or $KMgF_3$) with the ratio of fluorine concentration in these three forms ~2:2:1. During the following 30 day rehabilitation this ratio remains constant, the total fluorine content in the bone tissue decreases ~3 times and the F-apatite phase transforms into disordered (F, OH)-apatite.

A **protective effect of the zeolitic** enterosorbent (klinoptilolite) on fluorine binding in the intoxication process was found, as well as the promotional effect of this enterosorbent on **fluorine excretion** during the postfluoride rehabilitation.

Toxin#2: Aluminum
It has a devastating synergistic effect with fluoride, glyphosate and mercury

The recent publication from Prof.C.Exley proved that autism and high brain-levels of aluminium go along with each other. Since nanonized aluminium can be only poorly detected with current lab methods, indirect tests and clinical indicators (such as oxalate sensitivity) have to be used to confirm the presence.

Prof.B.Haley showed that **aluminium has a devastating synergistic effect with testosterone and mercury**. D.Geier MD showed years ago dramatic results by lowering testosterone in affected persons.

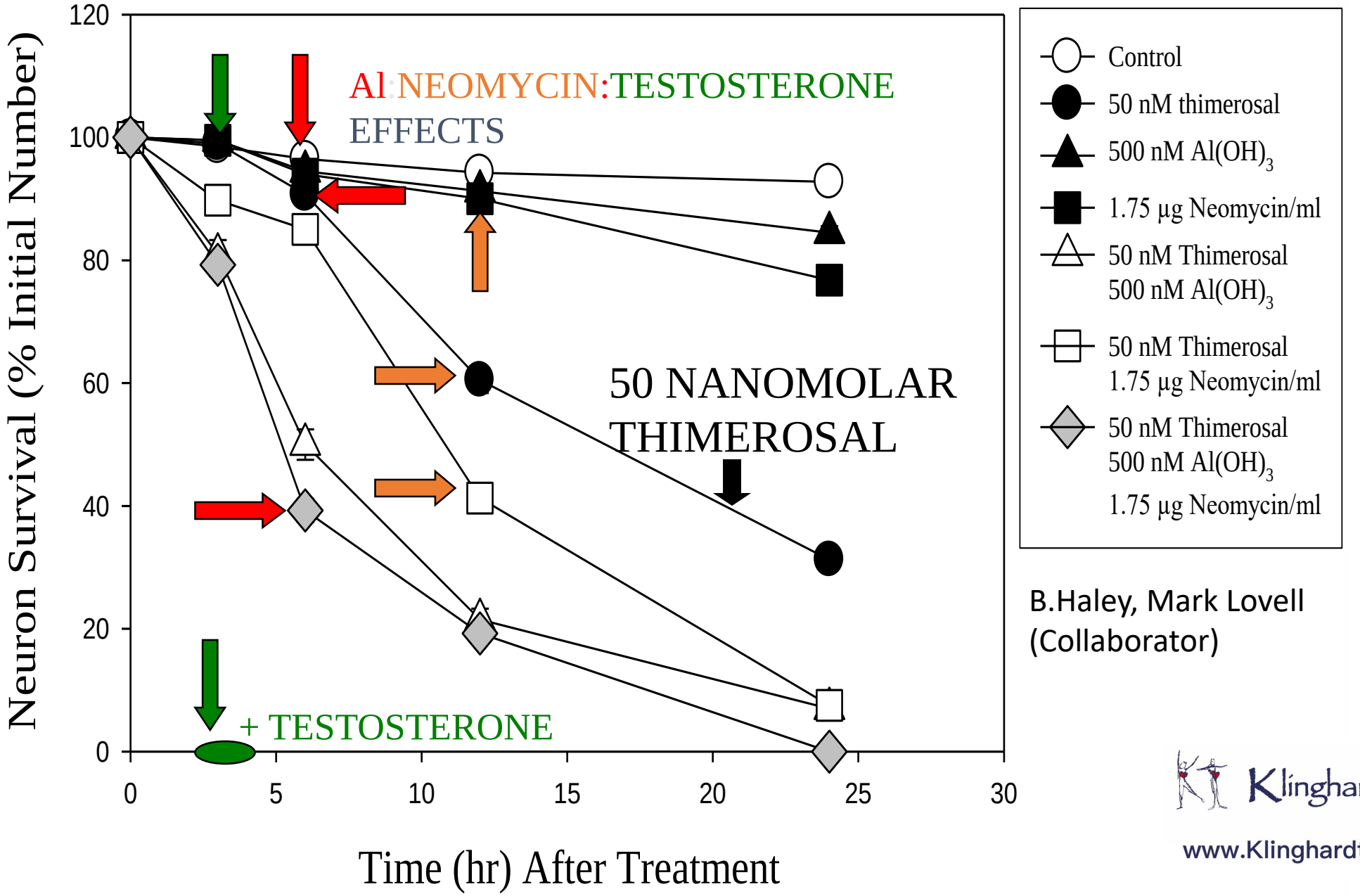
Prof. S.Seneff demonstrated the **synergy between glyphosate and aluminium**. In this lecture we will discuss effective diagnostic tools and biological toxicant lowering methods.

Journal of Neuroimmune Pharmacology; Dec 2008, Volume 3, Issue 4, pp 286-295; Lei Chen et al

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

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**.

SYNERGISTIC TOXICITIES of Hg and Al



B.Haley, Mark Lovell
(Collaborator)

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We observed that in ASD we have to consider the synergistic effects between several toxins and stressors, not just the individual levels of a particular toxin.

McLachlan, D. R., Bergeron, C., Alexandrov, P. N., Walsh, W. J., Pogue, A. I., Percy, M. E., ... & Zhao, Y. (2019). Aluminum in Neurological and Neurodegenerative Disease. *Molecular neurobiology*, 1-8.

This article finds AL elevated in AD,but not in ASD (compared to “controls”), but ignores the synergistic effects with Hg.

Alexandrov, Peter N., Aileen I. Pogue, and Walter J. Lukiw. "Synergism in aluminum and mercury neurotoxicity." *Integrative food, nutrition and metabolism* 5.3 (2018).

From the abstract: This is the first report on the neurotoxic effects of **aluminum sulfate and/or mercury sulfate on the initiation of inflammatory signaling** in human brain cells in primary culture. The effects aluminum+mercury together on other neurologically important signaling molecules or the effects of other combinations of common environmental metallic neurotoxins to human neurobiology currently remain not well understood

Allagui, M. S., et al. "Effects of melatonin on **aluminium-induced neurobehavioral and neurochemical changes** in aging rats." *Food and chemical toxicology* 70 (2014): 84-93.

Abstract

This study aimed to investigate the potential protective effects of melatonin (Mel) against aluminium-induced neurodegenerative changes in aging Wistar rats (24–28 months old). Herein, aluminium chloride ($AlCl_3$) (50 mg/kg BW/day) was administered by gavage, and melatonin (Mel) was co-administered to a group of Al-treated rats by an intra-peritoneal injection at a daily dose of 10 mg/kg BW for four months. The findings revealed that aluminium administration induced a significant decrease in body weight associated with marked mortality for the old group of rats, which was more pronounced in old Al-treated rats. Behavioural alterations were assessed by 'open fields', 'elevated plus maze' and 'Radial 8-arms maze' tests. The results demonstrated that Mel co-administration alleviated neurobehavioral changes in both old and old Al-treated rats. Melatonin was noted to play a good neuroprotective role, reducing lipid peroxidation (TBARs), and enhancing enzymatic (SOD, CAT and GPx) activities in the brain organs of old control and old Al-treated rats. Mel treatment also reversed the decrease of AChE activity in the brain tissues, which was confirmed by histological sections. Overall, the results showed that **Melatonin administration can induce beneficial effects for the treatment of Al-induced neurobehavioral and neurochemical changes in the central nervous system (CNS).**

Aluminium and Glyphosate

Aluminium has a synergistic effect with fluoride, mercury, aluminium and glyphosate

Seneff, Stephanie, Nancy Swanson, and Chen Li. "Aluminum and glyphosate can synergistically induce pineal gland pathology: connection to gut dysbiosis and neurological disease." *Agricultural Sciences* 6.01 (2015): 42.

Abstract Many neurological diseases, including autism, depression, dementia, anxiety disorder and Par- kinson's disease, are associated with abnormal sleep patterns, which are directly linked to pineal gland dysfunction. The pineal gland is highly susceptible to environmental toxicants. Two pervasive substances in modern industrialized nations are aluminum and glyphosate, the active ingredient in the herbicide, Roundup®. In this paper, we show how these two toxicants work synergistically to induce neurological damage. Glyphosate disrupts gut bacteria, leading to an overgrowth of Clostridium difficile. Its toxic product, p-cresol, is linked to autism in both human and mouse models. p-Cresol enhances uptake of aluminum via transferrin. Anemia, a result of both aluminum disruption of heme and impaired heme synthesis by glyphosate, leads to hypoxia, which induces increased pineal gland transferrin synthesis. Premature birth is associated with hypoxic stress and with substantial increased risk to the subsequent development of autism, linking hypoxia to autism. Glyphosate chelates aluminum, allowing ingested aluminum to bypass the gut barrier. This leads to anemia-induced hypoxia, promoting neurotoxicity and damaging the pineal gland. Both glyphosate and aluminum disrupt cytochrome P450 enzymes, which are involved in melatonin metabolism. Furthermore, melatonin is derived from tryptophan, whose synthesis in plants and microbes is blocked by glyphosate. We also demonstrate a plausible role for vitamin D3 dysbiosis in impaired gut function and impaired serotonin synthesis. This paper proposes that **impaired sulfate supply to the brain mediates the damage induced by the synergistic action of aluminum and glyphosate on the pineal gland and related midbrain nuclei**.

Problem: Aluminium is orally too poorly absorbed to cause significant damage. It has to be injected or inhaled to do maximum damage.

Mold, Matthew, et al. "**Aluminium in brain tissue in autism.**" *Journal of Trace Elements in Medicine and Biology* 46 (2018): 76-82.

1. Introduction

Autism spectrum disorder (ASD) is a group of neurodevelopmental conditions of unknown cause. It is highly likely that both genetic [1] and environmental [2] factors are associated with the onset and progress of ASD while the mechanisms underlying its aetiology are expected to be multifactorial [3], [4], [5], [6]. Human exposure to aluminium has been implicated in ASD with conclusions being equivocal [7], [8], [9], [10]. To-date the majority of studies have used hair as their indicator of human exposure to aluminium while aluminium in blood and urine have also been used to a much more limited extent. Paediatric vaccines that include an aluminium adjuvant are an indirect measure of infant exposure to aluminium and their burgeoning use has been directly correlated with increasing prevalence of ASD [11]. Animal models of ASD continue to support a connection with aluminium and to aluminium adjuvants used in human vaccinations in particular [12]. Hitherto there are no previous reports of aluminium in brain tissue from donors who died with a diagnosis of ASD. We have measured aluminium in brain tissue in autism and identified the location of aluminium in these tissues.

5. Conclusions

We have made the first measurements of **aluminium in brain tissue in ASD and we have shown that the brain aluminium content is extraordinarily high**. We have identified aluminium in brain tissue as both extracellular and intracellular with the latter involving both neurones and non-neuronal cells. The presence of aluminium in inflammatory cells in the meninges, vasculature, grey and white matter is a standout observation and could implicate aluminium in the aetiology of ASD.

frontiers in
NEUROLOGY

REVIEW ARTICLE
published: 05 February 2015
doi: 10.3389/fneur.2015.00004

Biopersistence and brain translocation of aluminum adjuvants of vaccines

Romain Kroum Gherardi *, Housam Eidi, Guillemette Crépeaux, François Jerome Authier and Josette Cadusseau

"Thus alum and other poorly biodegradable materials taken up at the periphery by phagocytes circulate in the lymphatic and blood circulation and can enter the brain using a Trojan horse mechanism similar to that used by infectious particles."

German MDs found, that in vulnerable children the injection of Desferal into the vaccine site asap after the vaccination can greatly reduce the amount of aluminum transported into the CNS. (In addition doctors have given the child (or adult) high silica "Polmolo" tincture from KiScience for a few weeks plus higher doses of NAC plus 300 mg melatonin as transdermal crème)

- **Shaw, C. A., and L. Tomljenovic. "Aluminum in the central nervous system (CNS): toxicity in humans and animals, vaccine adjuvants, and autoimmunity." *Immunologic research* 56.2-3 (2013): 304-316.**
- We have examined the neurotoxicity of aluminum in humans and animals under various conditions, following different routes of administration, and provide an overview of the various associated disease states. The literature demonstrates clearly negative impacts of aluminum on the nervous system across the age span. In adults, aluminum exposure can lead to apparently age-related neurological deficits resembling Alzheimer's and has been linked to this disease and to the Guamanian variant, ALS–PDC. Similar outcomes have been found in animal models. In addition, injection of aluminum adjuvants in an attempt to model Gulf War syndrome and associated neurological deficits leads to an ALS phenotype in young male mice. **In young children, a highly significant correlation exists between the number of pediatric aluminum-adjuvanted vaccines administered and the rate of autism spectrum disorders.** Many of the features of aluminum-induced neurotoxicity may arise, in part, from autoimmune reactions, as part of the ASIA syndrome.

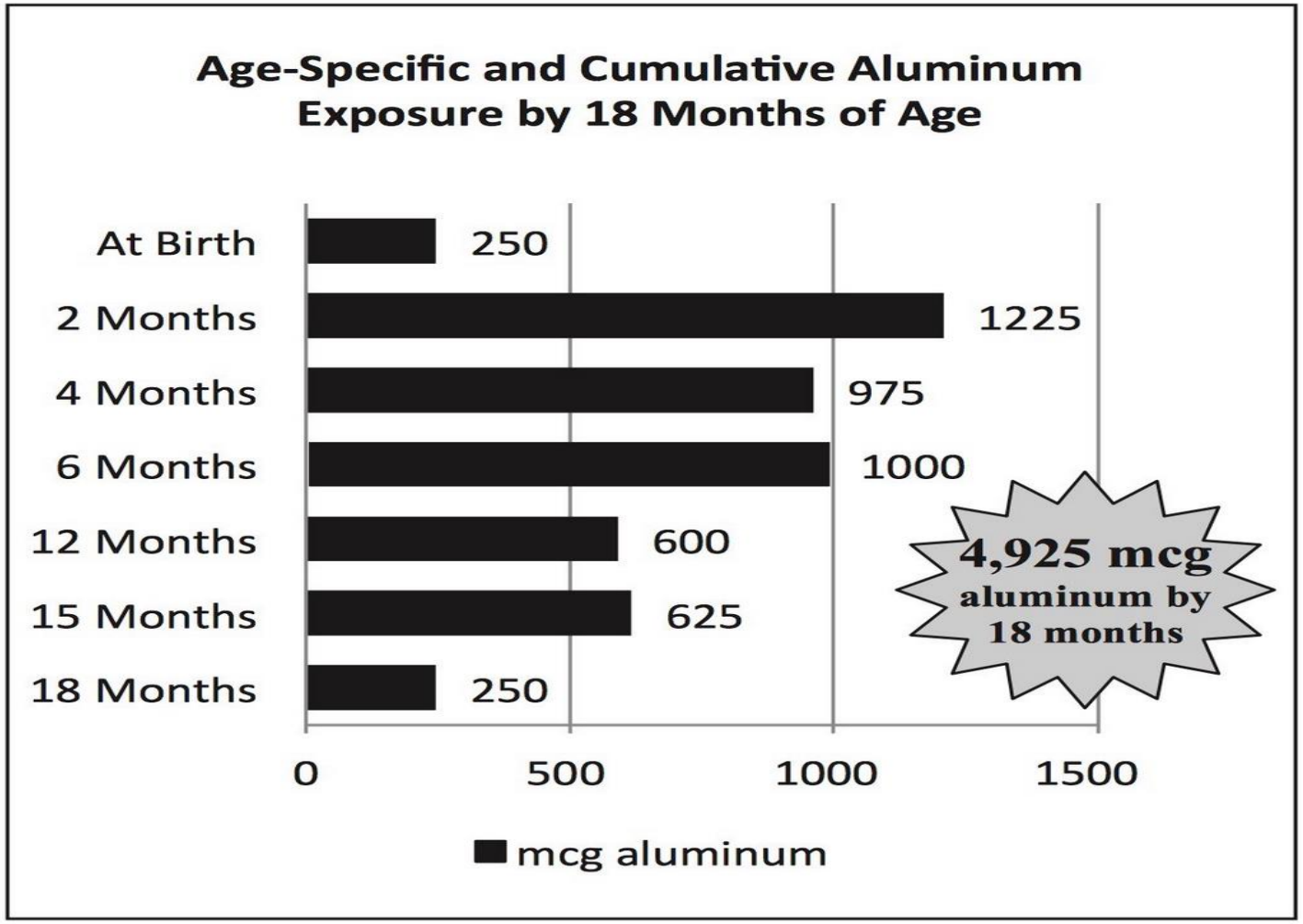
PK Vaccination schedule and parental exposure

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

Cumulative aluminum exposure from childhood vaccines*



*Source: The vaccine manufacturers' product inserts and the CDC's 2016 vaccination schedule

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Aluminum Sky – why? Does it stay up? were does it end up?

source: flikr.com, Pandoozy Photos, South Downs, Woodingdean

Toxic Rain in the US – Intentional poisoning of a Country

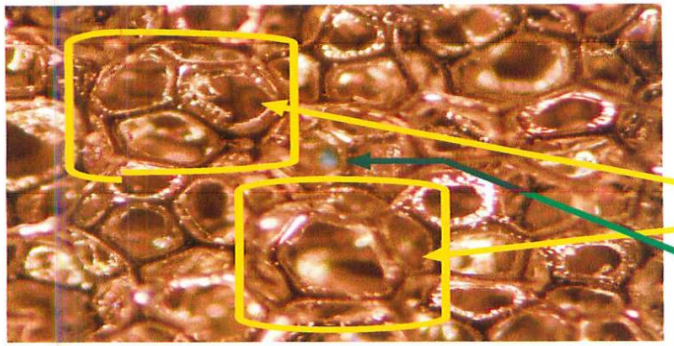
Normal aluminium levels in rain: 0 - 0.5 µg/l

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

Monsanto in your genes: Aluminum adducts blocking life (ALS patient)

Fluorescence-probe study of fatty acids in mitochondrial and plasma membranes

Please note that only abnormalities are illustrated

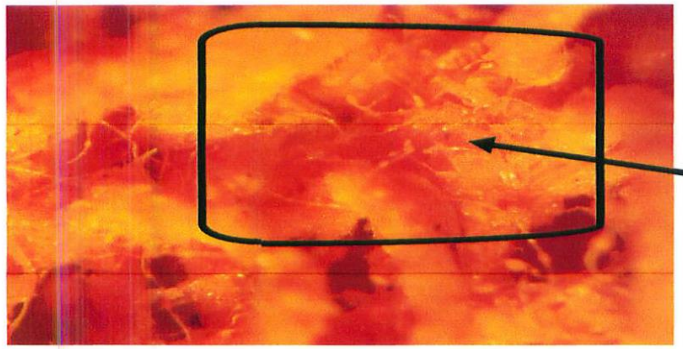


Layered polarization/fluorescence image
Outer surface of plasma membrane

Oxidative damage – white areas

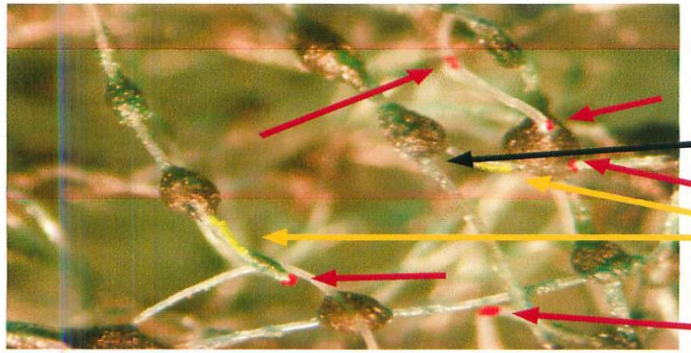
Large fatty acid structures
mostly at approx 0.4um spacing

Aluminium – green probe



Very high magnification fluorescence
Arachidonic acid/other fatty acids

Lower edge of one of the **large fatty acid structures**: weak interaction with normal 18- and 20-carbon PUFAs



The cytoskeleton (layered fluorescence)
actin fibrils & mitochondria

Rather unusual linking between some actin fibrils & mitochondria

Aluminium on some actin fibrils (Al = yellow probe)

Ca-actin binding (Ca = red probe)

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Military Operations: outfitting capability

The Boeing Factory – clandestine off-label use of high flying planes

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

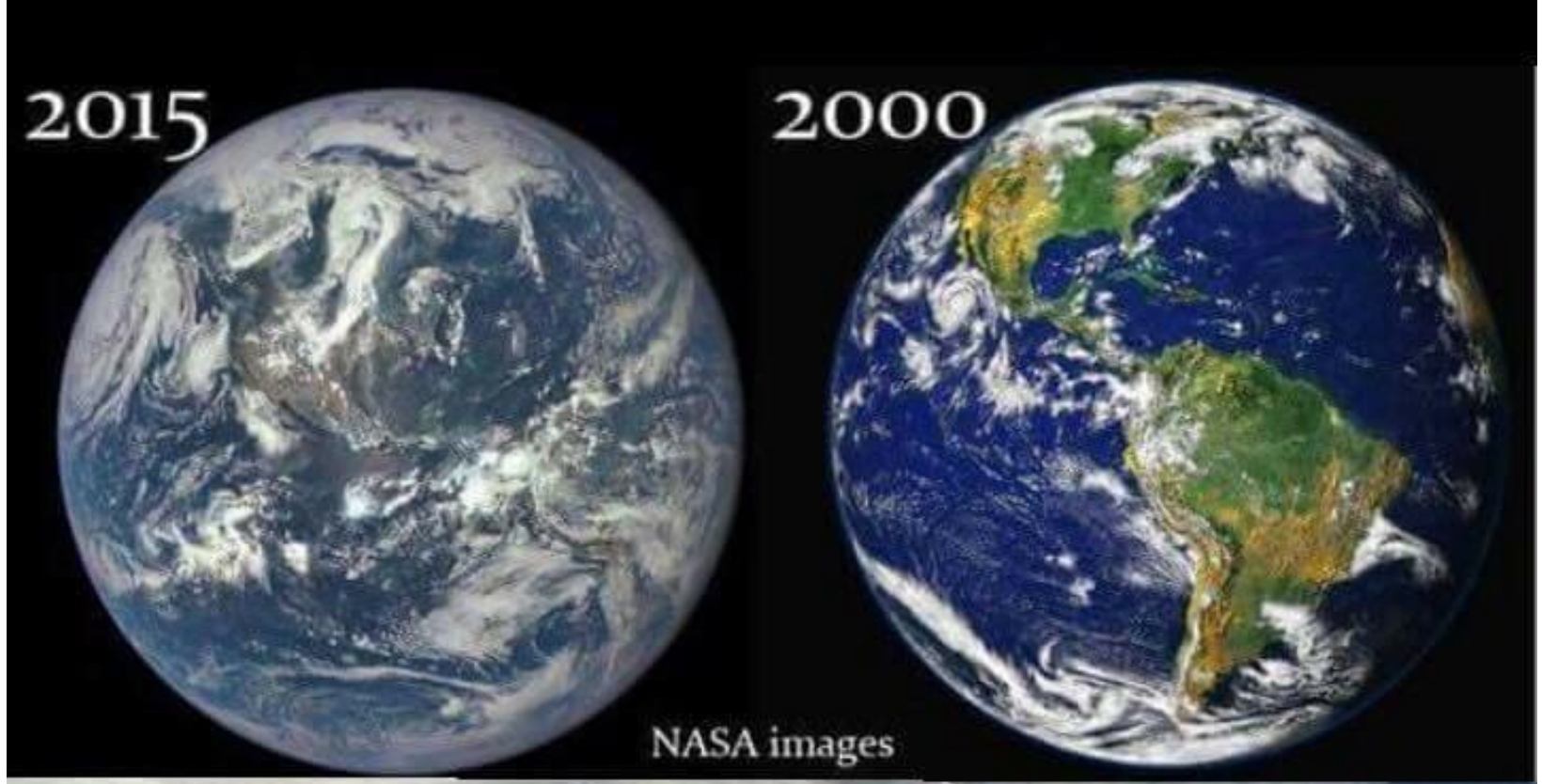
J. Marvin Herndon, ***Received: 29 June 2015 / Accepted: 5 August 2015 / Published: 11 August 2015***

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.

Wilker EH, Preis SR, Beiser AS, Wolf PA, Au R, Kloog I, Li W, Schwartz J, Koutrakis P, DeCarli C, Seshadri S, Mittleman MA. "Long-Term Exposure to Fine Particulate Matter, Residential Proximity to Major Roads and Measures of Brain Structure." Stroke. 2015 Apr 23.

"**Exposure to elevated levels of ambient fine particulate matter** was **associated with smaller total cerebral brain volume**, a marker of age-associated brain atrophy, and with higher odds of covert brain infarcts," the study authors submit that: "These findings suggest that air pollution is associated with insidious effects on structural brain aging even in dementia- and stroke-free persons."



Aluminum Toxicity: towards a solution

Zimnik, Paul R., and Joseph Sneddon. "Binding and removal of aluminum ions in waters by an algal biomass." *Analytical letters* 21.8 (1988): 1383-1396. A preliminary study on the binding and **removal of trace concentrations of aluminum** ions in waters by two species of algae, Chlorella Pyrenoidosa and **Chlorella Vulgaris**, were investigated. Binding by the former was minimal over all pH ranges, but binding by the latter was effective with a maximum binding of 68% occurring at pH 5. Binding was lowered drastically below pH 2, and this may be used to remove aluminum from the algae. Optimum binding occurred after 20 minutes exposure time of algae to aluminum solution and 450 mg algae mass to 100 mL solution. Binding was reproducible and more efficient in waters with low suspended solids. High salt concentrations interfere with binding, and the **Chlorella Vulgaris** could be reused 7 times with washings between each binding before a noticeable decrease in binding efficiency was found.

Rao, Mandava V., and Anshita R. Purohit. "Neuroprotection by **melatonin** on **mercury** induced toxicity in the rat brain." *Pharmacology & Pharmacy* 2.04 (2011): 375.

El-Sokkary, Gamal H., Esam S. Kamel, and Russel J. Reiter. "Prophylactic effect of **melatonin** in reducing **lead**-induced neurotoxicity in the rat." *Cellular & molecular biology letters* 8, no. 2 (2003): 461-470

Aluminium toxicity: towards a solution

Millán-Plano, S., García, J. J., Martínez-Ballarín, E., Reiter, R. J., Ortega-Gutiérrez, S., Lázaro, R. M., & Escanero, J. F. (2003). Melatonin and pinoline prevent aluminium-induced lipid peroxidation in rat synaptosomes. *Journal of trace elements in medicine and biology*, *17*(1), 39-44.

Abstract

The serum concentrations of aluminum, a metal potentially involved in the pathogenesis of Alzheimer's disease, increase with age. Also, intense and **prolonged exposure to aluminum may result in dementia.** Melatonin and pinoline are two well known antioxidants that efficiently reduce lipid peroxidation due to oxidative stress. Herein, we investigated the effects of melatonin and pinoline in preventing aluminum promotion of lipid peroxidation when the metal was combined with $FeCl_3$ and ascorbic acid in rat synaptosomal membranes. Lipid peroxidation was estimated by quantifying malondialdehyde (MDA) and 4-hydroxyalkenal (4-HDA) concentrations in the membrane suspension. Under the experimental conditions used herein, the addition of aluminum (0.0001 to 1 mmol/L) enhanced MDA + 4-HDA formation in the synaptosomes. **Melatonin and pinoline reduced**, in a concentration-dependent manner**, lipid peroxidation due to aluminum**, $FeCl_3$ and ascorbic acid in the synaptosomal membranes.

These results suggest that the indoleamine **melatonin** and the β-carboline pinoline **may potentially act as neuroprotectant agents in the therapy of those diseases with elevated aluminum concentrations in the tissues.**

Albendea, C. D., Gómez-Trullén, E. M., Fuentes-Broto, L., Miana-Mena, F. J., Millán-Plano, S., Reyes-Gonzales, M. C., ... & García, J. J. (2007). **Melatonin reduces** lipid and protein oxidative damage in synaptosomes due to **aluminium**. *Journal of Trace Elements in Medicine and Biology*, *21*(4), 261-268.

Abstract

Prolonged exposure to excessive aluminium (Al) concentrations is involved in the ethiopathology of certain dementias and neurological disorders. Melatonin is a well-known antioxidant that efficiently reduces lipid peroxidation due to oxidative stress. Herein, we investigated in synaptosomal membranes the effect of melatonin in preventing Al promotion of lipid and protein oxidation when the metal was combined with $FeCl_3$ and ascorbic acid. Lipid peroxidation was estimated by quantifying malondialdehyde (MDA) and 4-hydroxyalkenals (4-HDA) concentrations in the membrane suspension and protein carbonyls were measured in the synaptosomes as an index of oxidative damage. Under our experimental conditions, the addition of Al (0.0001–1 mmol/L) enhanced MDA+4-HDA formation in the synaptosomes. In addition, Al (1 mmol/L) raised protein carbonyl contents. Melatonin reduced, in a concentration-dependent manner, lipid and protein oxidation due to Al, $FeCl_3$ and ascorbic acid in the synaptosomal membranes. These results show that melatonin confers protection against Al-induced oxidative damage in synaptosomes and suggest that this indoleamine may be considered as a neuroprotective agent in Al toxicity because of its antioxidant activity.

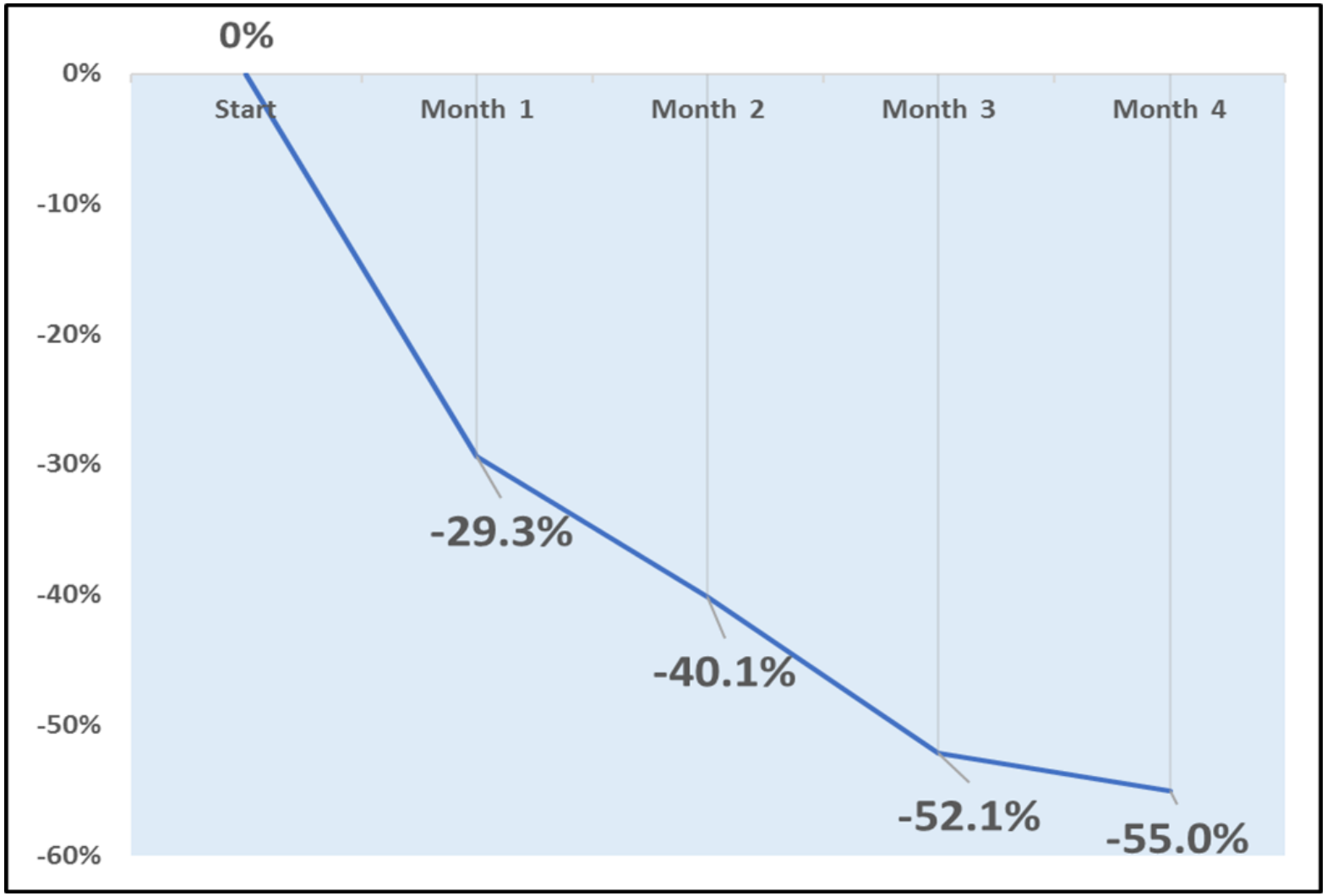
Aluminium and Mercury Detox

- Transdermal **Melatonin** (80-300 mg): Abd-Elghaffar, Sary Kh, Gamal H. El-Sokkary, and Ahmed A. Sharkawy. "**Aluminum-induced neurotoxicity** and oxidative damage in rabbits: protective effect of melatonin." *Neuroendocrinology Letters* 26.5 (2005): 609-616.
- **Coriandolo** tincture - specially grown organic cilantro + 7 flower stem cell extracts: slowly titrate from 5 drops twice daily to 2 dropperfull (=30 drops) 3 times daily (t.i.d) 30 min before each meal (mobilizes toxic metals, also increases bile flow) available from www.KiScience.com
- **Renolo** - allium ursinum, cistus incanus, 7 flower stem cell extracts (protects white and red bloodcells and nephrons from oxidative damage caused by mobilized metals) available from www.KiScience.com
- **Polmolo** – enula root, coriandolo plant & seeds, bardana root and equiseto. 10 to 15 drops twice daily. Available from www.KiScience.com

Binders:

- **Ki-Chlorella Vulgaris or Pyrenoidosa:** start with medium dose of 8 tablets (200mg each) t.i.d 5 min after taking Cilantro. Increase: during times of crisis, Herxheimer reaction or if no change is perceived. Max dose: 40 tablets. 3 times daily. Available from www.KiScience.com; www.BioPureUS.com
- **High Silica Zeolite**: in addition to chlorella (or instead, if chlorella is not yet tolerated): start ¼ teaspoon twice daily between meals, slowly increase to 4 times/day away from all food or vitamins. Available from www.KiScience.com
- **Ki Science Detox Footbath:** twice weekly for 30 minutes (www.KiScience.com). Main metal mobilization is on day 3 after the treatment. Increase binders on that day. www.KiScience.com
- High Organic Silica **Acilis Water**: 1 quart/day www.KiScience.com
- Kruck Protocol for AD: 500 mg Desferal s.c. once weekly for 18 months

The Ionic Footbath (KiScience, IonCleanse)



Average ATEC reduction was 55%!

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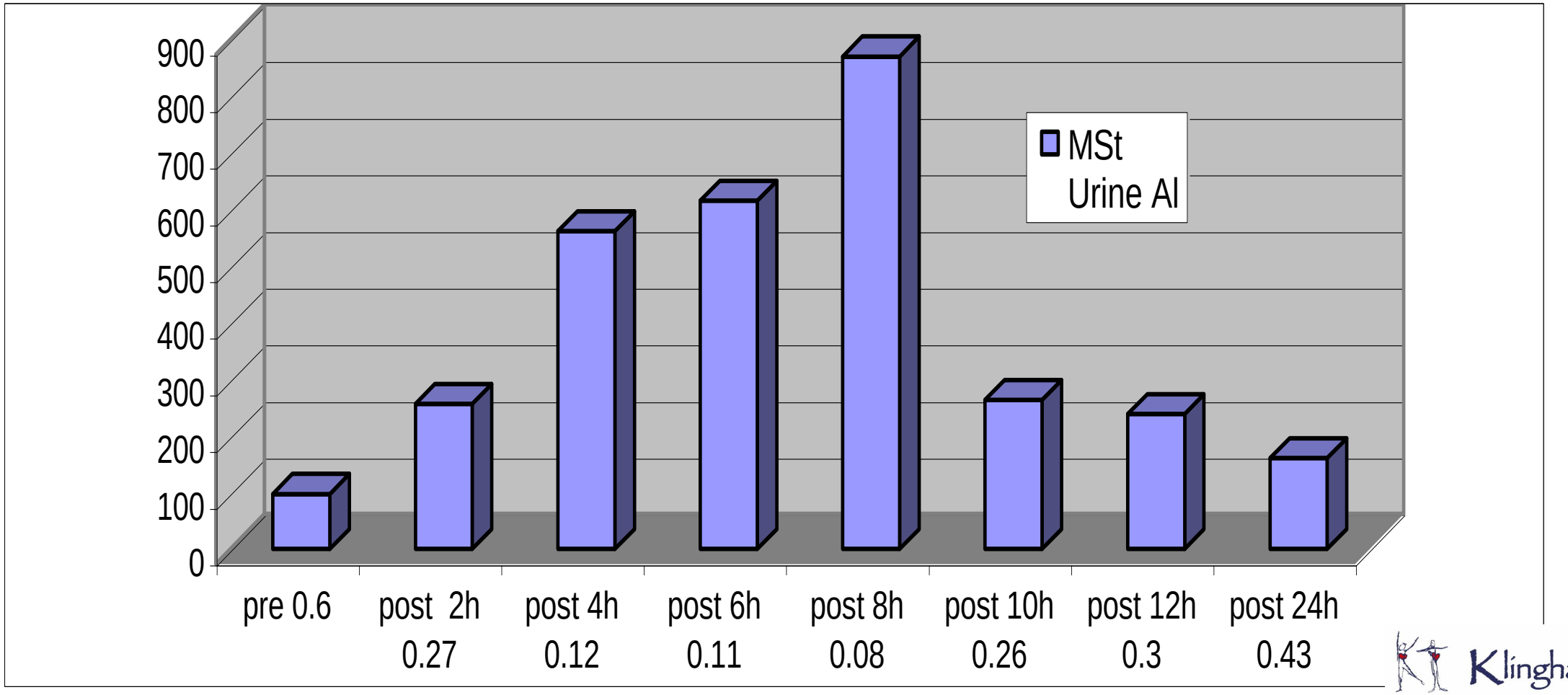
Over 900% increase of aluminum excretion 8 hrs post 30- minute ionic footbath (we used model from www.kiScience.com)



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Toxin #3:
Glyphosate,
Roundup and
GMO Crops

GMO Roundup-Ready corn, soy, canola, sugar beets, cotton, tobacco and alfalfa

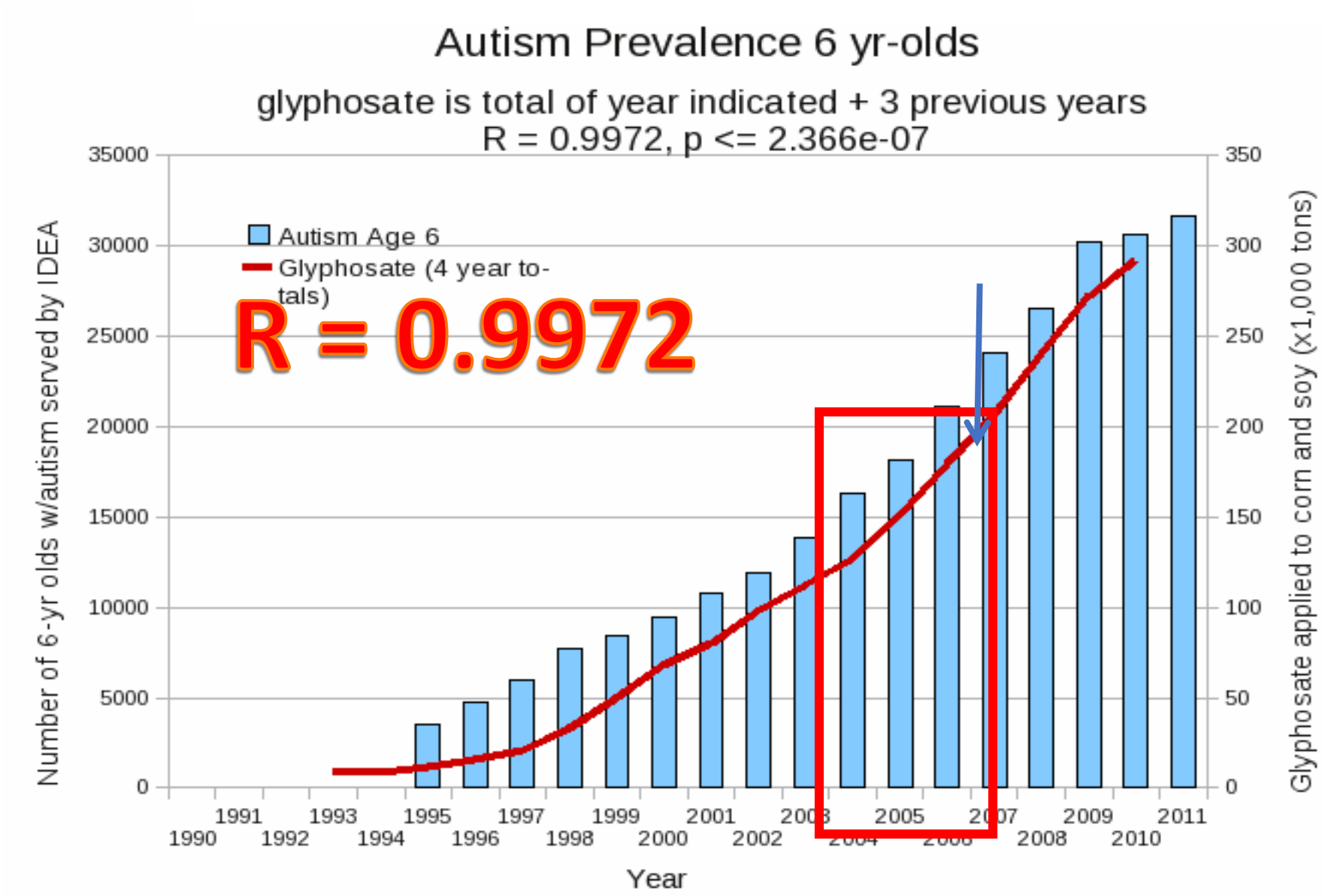
A little discourse on Glyphosate (from Stephanie Seneff PhD)

- Glyphosate is the most-used herbicide and harvest-drying agent used worldwide. It is inexpensive to produce, out of patent for decades (and therefore produced by many companies worldwide). It produces higher crop yields in the first few years before the soil is dead and becomes non-productive.
- Glyphosate selectively destroys our healthy gut microbes and fosters the growth of pathogens. It also destroys the microbes in our soil, the very foundation of life on the planet.
- It is also a chelating agent and renders microminerals in our food non-absorbable. It works as a shuttle agent for aluminium in the food and blood and transports it straight into the brain.
- When it enters the interstitium, its highly positive charge changes the membrane potential of all cells in the body and interferes with almost every healthy biological function of ionic channels, receptors and pumps.
- The damage to the aminoacid producing gut microbes makes us starved and **deficient for tryptophane, serotonin and melatonin.**

Journal of Organic Systems, 9(2), 2014
The Pearson correlation coefficients are highly significant between the percentage of GE corn and soy planted in the US and

- Hypertension, stroke
- diabetes prevalence
- diabetes incidence
- obesity
- lipoprotein metabolism disorder
- **Alzheimer's, senile dementia**
- **Parkinson's**
- **multiple sclerosis**
- **autism**
- inflammatory bowel disease
- intestinal infections
- end stage renal disease
- acute kidney failure
- cancers of the
  - Thyroid
  - Liver
  - Bladder
  - Pancreas
  - Kidney
  - and myeloid leukaemia

Autism Prevalence: 6 year olds*



* Figure 15, Seneff et al., Agricultural Sciences, 2015, 6, 42-70

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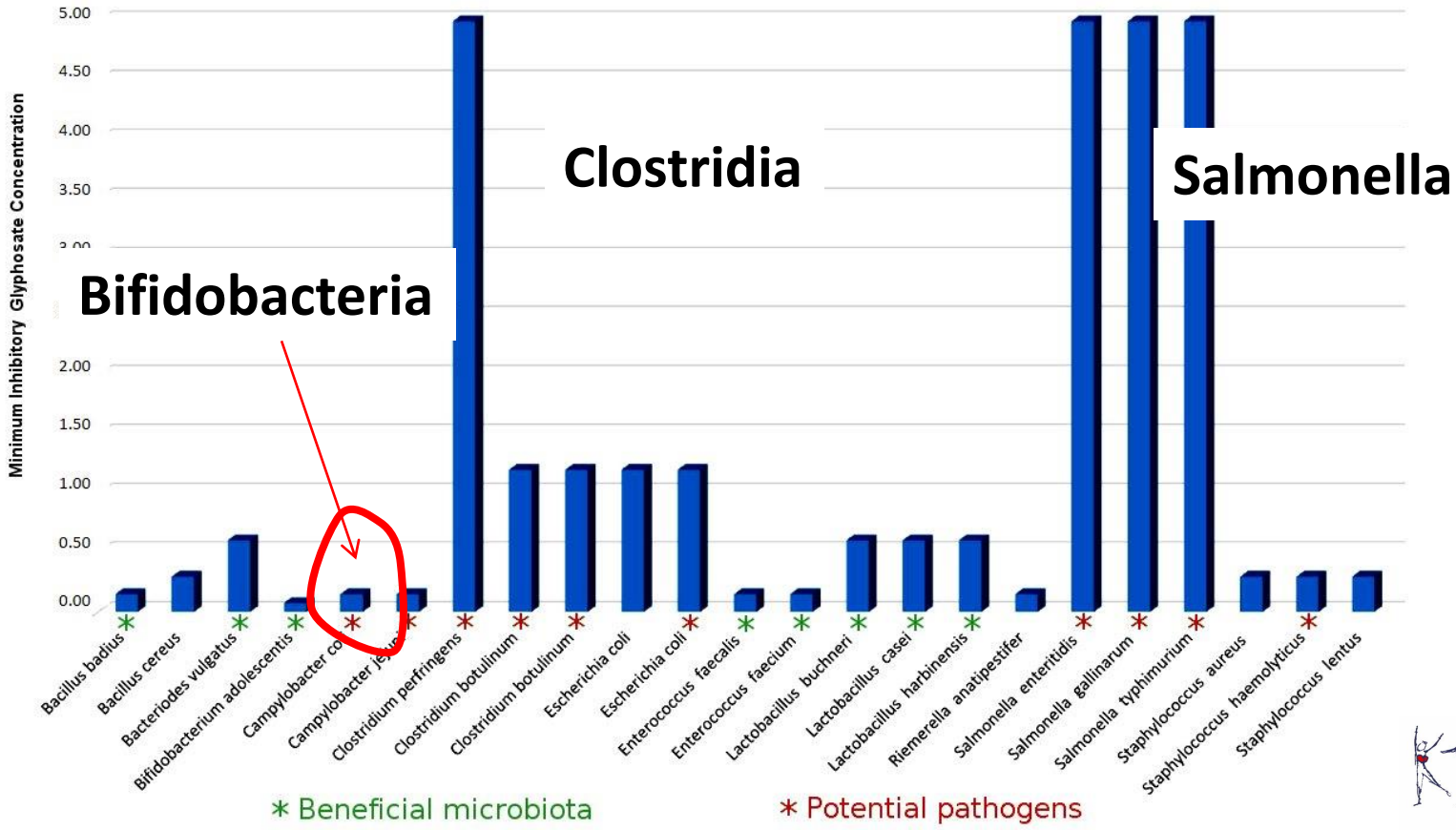
Pathogen Overgrowth in Poultry Microbes Exposed to Glyphosate*

Shehata AA, Schrödl W, Aldin AA, Hafez HM, Krüger M. The effect of glyphosate on potential pathogens and beneficial members of poultry microbiota in vitro. Curr Microbiol. 2013 Apr;66(4):350-8.



*Plot provided by Dr. Martin Michener

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Glyphosate Contamination in Vaccines (Parts Per Billion)*

Merck	ZOSTAVAX	0.62	Shingles
Merck	MMR-II	3.74	Measles, Mumps and Rubella
Merck	VARIVAX	0.56	Varicella, Chicken Pox
MERCK	PNEUMOVAX	ND	Pneumococcal 18
MERCK	PROQUAD	0.66	Measles, Mumps, Rubella, Varicella
GSK	ENERGIX-B	0.34	Heptatitis B

*A Samsel and S Seneff, Journal of Biological Physics and Chemistry 2017;17:8-32.

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Roundup as a Desiccant/ Ripener just before Harvest

- Eliminating Glyphosate, Pesticides, Phthalates, Bisphenol A, wood preservatives, petrochemicals
- Humic acid (from peat) Shehata, Awad A., et al. "Distribution of glyphosate in chicken organs and its reduction by humic acid supplementation." *The Journal of Poultry Science* 51.3 (2014): 333-337. ("**Austrian Peat extract**" from www.KiScience.com))
- High dose **transdermal melatonin** de Almeida, Lécio Leone, et al. "Effects of melatonin in rats in the initial third stage of pregnancy exposed to sub-lethal doses of **herbicides.**" *Acta histochemica* 119.3 (2017): 220-227.*Conclusion: These results reveal that melatonin is a protective agent against experimentally induced maternal/embryo toxicity with herbicides and favoring normalization of reproductive parameters and hepatic.*
- **Glycine** (adult dose: 5 grams twice daily – Glypohsate is a glycine derivative)
- Homeopathic auto-urine therapy (AUT): H-Series of 1:5 dilutions. Secuss 50 times. After step 6 (=H6) test with ART. The dilution which gives the strongest yang state is used. 8 drops hourly for 2 days, then qid. Renew weekly, since antigens in urine will change rapidly (more: see protocol handout) – or: Homeopathic "simile" (i.e. glyphosate 12 C)
- Binders: **Zeolite** 1-2 caps tid, **chlorella vulgaris**: 8 tbl tid, between meals and/or at bedtime (all from KiScience)
- **Ionic Foot/handbath** ("FootSPa" from KiScience.com) – we found glyphosate in the water post tx
- Sauna therapy and/or infrared therapy (many devices)
- Eat organic only!

Toxin #4: man made electromagnetic fields (microwave etc.)

W*e are exposed to Microwave – everywhere we go - it opens the blood brain barrier, so that fluoride, aluminium and glyphosate can enter. It also destroys our DNA and that of our children.*

- Kesari, Kavindra Kumar, Sanjay Kumar, and Jitendra Behari. "Pathophysiology of microwave radiation: effect on rat brain." *Applied biochemistry and biotechnology* 166.2 (2012): 379-388.
- Reiter, R. J. "Electromagnetic fields and melatonin production." *Biomedicine & pharmacotherapy* 47.10 (1993): 439-444.
- Adey, W. Ross. "Biological effects of electromagnetic fields." *Journal of cellular biochemistry* 51.4 (1993): 410-416.
- Rudolph, Klaus, et al. "Static magnetic fields decrease nocturnal pineal cAMP in the rat." *Brain Research* 446.1 (1988): 159-160.
- Magnetosensitivity of the rat's pineal cyclic adenosine monophosphate (cAMP) system was investigated. During their dark phase, rats were exposed for one hour to a static magnetic field (MF) inverting the horizontal component of the natural MF. MF-exposed animals showed a 38% decrease in pineal cAMP content (1.21 pmol/pineal gland) compared to a non-exposed control group (1.96 pmol/pineal gland).
- Nazıroğlu, Mustafa, Sümeyye Tokat, and Seda Demirci. "Role of melatonin on electromagnetic radiation-induced oxidative stress and Ca2+ signaling molecular pathways in breast cancer." *Journal of Receptors and Signal Transduction* 32.6 (2012): 290-297.

A little look at our relevant anatomy: our most vulnerable part of your brain is the Pineal gland (and the least studied)

What do we know about it? Wikipedia says:

The pineal gland is involved in several functions of the body including:

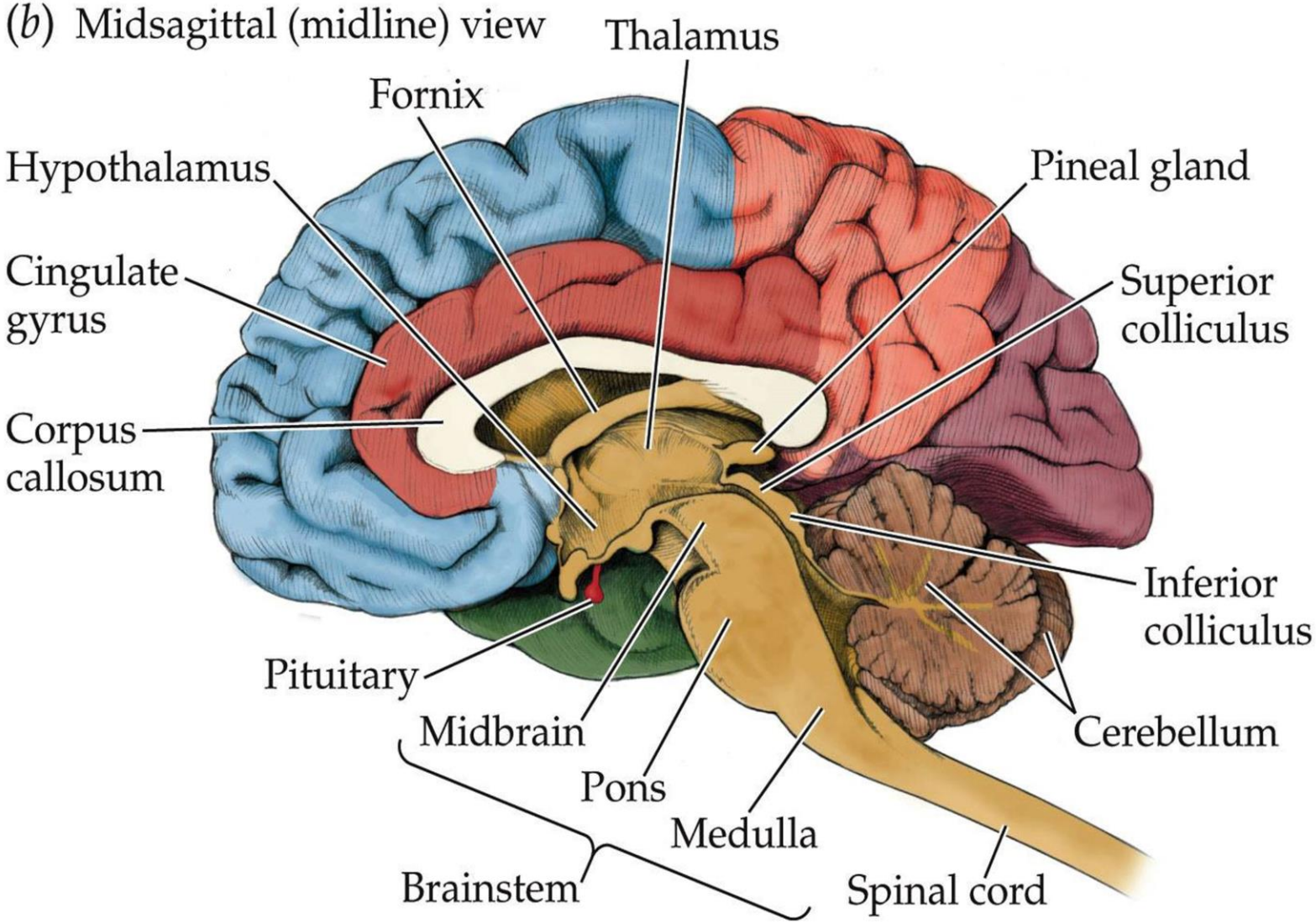
- secretion of the hormone **melatonin** with its multiple roles in our health
- Regulation of many if not all endocrine functions
- Conversion of nervous system signals to endocrine signals
- Causes sleepiness
- Influences sexual development
- Influences immune system function.

In humans, however, its full function remains somewhat unknown. It is **located in the anatomical center of the brain**.

The projection zone of the pineal gland to the forehead is often referred to as the third eye (also called the mind's eye, or inner eye). It is mostly believed to be a mystical and esoteric concept of a speculative invisible eye which provides perception beyond ordinary sight. In certain dharmic spiritual traditions the third eye refers to the ajna (or brow) chakra.

Experiment Prof. Y.Omura: if the 3rd eye is covered with aluminum foil, a dowser can no longer dowse, a kinesiologist can no longer muscle test and a psychic can no longer retrieve higher information

Pineal Gland activation: 936Hz

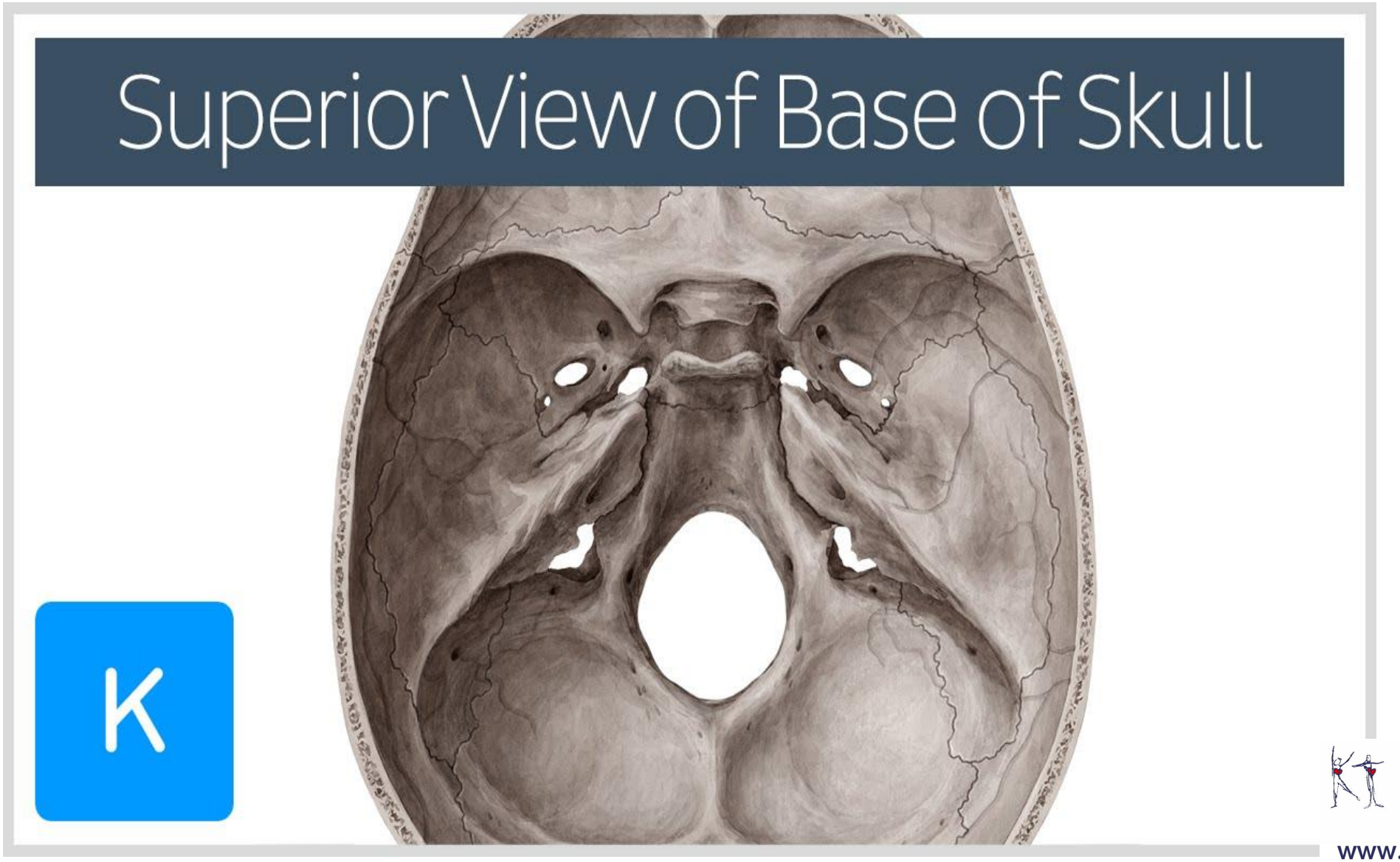


***Biological Psychology 6e*, Figure 2.12 (Part 2)**

If the dish would be the base of the skull, the receiver would be exactly at the location of the pineal gland. **The skull and pineal gland are a perfect satellite dish - tuned for the reception of light**

Klinghardt Institute
The Heart Of Healing
www.KlinghardtInstitute.com

The base of the skull is shaped like 4 satellite dishes slightly turned towards a common point: the exact location of the **pineal gland**



Sahai, Ashok, and Raj Kumari Sahai. "Pineal gland: A structural and functional enigma." *Journal of the Anatomical Society of India* 62, no. 2 (2013): 170-177.

Abstract

The structures and **functions of neuroendocrine pineal gland remains an enigma to both philosophers and scientists** alike since time immemorial. Some of the structural and functional mysteries of pineal gland are unfolded to some extent in this article by reviewing the work of various researchers. Recently a neuronal circuit consisting of seven neurons between retina and pineal gland have been established to relate the effect of light and other rays on its secretion.

The various physical properties such as piezoelectricity, piezoluminescence, electromagnetic field, solar flare, infrared energy are also explained and correlated with the structural and secretional components of the gland. The neurosecretion of pineal gland such as melatonin play an important role in sleep-wake patterns, timings and release of reproductive hormones along with temperature control.

The presence of all enzymes needed for the synthesis of di-methyl-tryptamine (DMT) in pineal gland explains the **near death experience** (NDE) phenomenon. The various audio-visual hallucinations in NDE phenomenon occur due **to massive increase of DMT in pineal gland before death**. A very high concentration of di-methyl-tryptamine (DMT), presence of retinal proteins in 5–10% of pinealocytes, its role in thermoregulation and a **possible role as magnetoreceptor in blind men** and highest deposits of fluoride in the body are not only interesting but significant for the future research. Hence a lot of further research on pineal gland is still required to correlate its unique properties with its structural components.

Exposing all of us, especially the foetus, to destructive radiation 24/7

"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.

Herbert, Martha R., and Cindy Sage. "**Autism and EMF**? Plausibility of a pathophysiological link–Part I." *Pathophysiology* 20.3 (2013): 191-209.

Although autism spectrum conditions (ASCs) are defined behaviorally, they also involve multileveled disturbances of underlying biology that find striking parallels in the physiological impacts of electromagnetic frequency and radiofrequency exposures (EMF/RFR). Part I of this paper will review the critical contributions pathophysiology may make to the etiology, pathogenesis and ongoing generation of core features of ASCs. We will review pathophysiological damage to core cellular processes that are associated both with ASCs and with biological effects of EMF/RFR exposures that contribute to chronically disrupted homeostasis. Many studies of people with ASCs have identified oxidative stress and evidence of free radical damage, cellular stress proteins, and deficiencies of antioxidants such as glutathione. Elevated intracellular calcium in ASCs may be due to genetics or may be downstream of inflammation or environmental exposures. Cell membrane lipids may be peroxidized, mitochondria may be dysfunctional, and various kinds of immune system disturbances are common. Brain oxidative stress and inflammation as well as measures consistent with blood–brain barrier and brain perfusion compromise have been documented. Part II of this paper will review how behaviors in ASCs may emerge from alterations of electrophysiological oscillatory synchronization, how EMF/RFR could contribute to these by de-tuning the organism, and policy implications of these vulnerabilities. Changes in brain and autonomic nervous system electrophysiological function and sensory processing predominate, seizures are common, and sleep disruption is close to universal. All of these phenomena also occur with EMF/RFR exposure that can add to system overload ('allostatic load') in ASCs by increasing risk, and worsening challenging biological problems and symptoms; conversely, reducing exposure might ameliorate symptoms of ASCs by reducing obstruction of physiological repair. Various vital but vulnerable mechanisms such as calcium channels may be disrupted by environmental agents, various genes associated with autism or the interaction of both. With dramatic increases in reported ASCs that are coincident in time with the deployment of wireless technologies, we need aggressive investigation of potential ASC – EMF/RFR links. The evidence is sufficient to warrant new public exposure standards benchmarked to low-intensity (non-thermal) exposure levels now known to be biologically disruptive, and strong, interim precautionary practices are advocat

Pall, Martin L. "Electromagnetic fields act via activation of voltage-gated calcium channels to produce beneficial or adverse effects." *Journal of cellular and molecular medicine* 17.8 (2013): 958-965.

The direct targets of extremely low and microwave frequency range electromagnetic fields (EMFs) in producing non-thermal effects have not been clearly established. However, studies in the literature, reviewed here, provide substantial support for such direct targets. Twenty-three studies have shown that voltage-gated calcium channels (VGCCs) produce these and other EMF effects, such that the L-type or other VGCC blockers block or greatly lower diverse EMF effects. Furthermore, the voltage-gated properties of these channels may provide biophysically plausible mechanisms for EMF biological effects. Downstream responses of such EMF exposures may be mediated through Ca^{2+}/calmodulin stimulation of nitric oxide synthesis. Potentially, physiological/therapeutic responses may be largely as a result of nitric oxide-cGMP-protein kinase G pathway stimulation. A well-studied example of such an apparent therapeutic response, EMF stimulation of bone growth, appears to work along this pathway. However, pathophysiological responses to EMFs may be as a result of nitric oxide-**peroxynitrite-oxidative stress pathway** of action. A single such well-documented example, EMF induction of DNA single-strand breaks in cells, as measured by alkaline comet assays, is reviewed here. Such single-strand breaks are known to be produced through the action of this pathway. Data on the mechanism of EMF induction of such breaks are limited; what data are available support this proposed mechanism. Other Ca^{2+}-mediated regulatory changes, independent of nitric oxide, may also have roles. This article reviews, then, a substantially supported set of targets, VGCCs, whose stimulation produces non-thermal EMF responses by humans/higher animals with downstream effects involving Ca^{2+}/calmodulin-dependent nitric oxide increases, which may explain therapeutic and pathophysiological effects.

Pall, Martin L. "Microwave frequency electromagnetic fields (EMFs) produce widespread neuropsychiatric effects including depression." *Journal of Chemical Neuroanatomy* 75 (2016): 43-51.

A key mechanism in autism is the generation of peroxynitrite in the neuron, leading to severe oxidative damage. Melatonin mitigates….

Sajdel-Sulkowska, Elizabeth M., Ming Xu, and Noriyuki Koibuchi. "Increase in cerebellar neurotrophin-3 and oxidative stress markers in autism." *The Cerebellum* 8.3 (2009): 366-372.

Abstract

Autism is a neurodevelopmental disorder characterized by social and language deficits, ritualistic–repetitive behaviors and disturbance in motor functions. Data of imaging, head circumference studies, and Purkinje cell analysis suggest impaired brain growth and development. Both genetic predisposition and environmental triggers have been implicated in the etiology of autism, but the underlying cause remains unknown. Recently, we have reported an increase in 3-nitrotyrosine (3-NT), a marker of oxidative stress damage to proteins in autistic cerebella. In the present study, we further explored oxidative damage in the autistic cerebellum by measuring 8-hydroxydeoxyguanosine (8-OH-dG), a marker of DNA modification, in a subset of cases analyzed for 3-NT. We also explored the hypothesis that oxidative damage in autism is associated with altered expression of brain neurotrophins critical for normal brain growth and differentiation. The content of 8-OH-dG in cerebellar DNA isolated by the proteinase K method was measured using an enzyme-linked immunosorbent assay (ELISA); neurotrophin-3 (NT-3) levels in cerebellar homogenates were measured using NT-3 ELISA. Cerebellar 8-OH-dG showed trend towards higher levels with the increase of 63.4% observed in autism. Analysis of cerebellar NT-3 showed a significant ($p = 0.034$) increase (40.3%) in autism. Furthermore, there was a significant positive correlation between cerebellar NT-3 and 3-NT ($r = 0.83$; $p = 0.0408$). These data provide the first quantitative measure of brain NT-3 and show its increase in the autistic brain. Altered levels of brain NT-3 are likely to contribute to autistic pathology not only by affecting brain axonal targeting and synapse formation but also by further exacerbating oxidative stress and possibly contributing to Purkinje cell abnormalities.

Melatonin is – in higher doses - the **strongest antioxidant and peroxinitrite scavenger** in our system (Reiter, Russel J., et al. "Biochemical reactivity of melatonin with reactive oxygen and nitrogen species." *Cell biochemistry and biophysics* 34.2 (2001): 237-256.)

Electromagnetic Radiation (EMR)

- Internal protection:
  - KiScience tincture **RayWave** (Cilantro, Propolis, Rosemary + frequencies).
    Take 2-3 pipettes 2-3 times daily

- Daytime strategy:
  - E-Shield skin cream (KiScience)
  - Stetzer filters at home or workplace
  - Avoid use of cellphone – texting is ok
  - Switch off WiFi whenever not in use or get rid of WiFi – get Broadband/Ethernet
  - Consider Building Biology –measuring, considering Swiss Shield paint/earthing
  - wear radiowave protective clothing

- Nightime strategy:
  - melatonin transdermal crème (after dinner) – 80-500 mg (to obtain all benefits)
  - Reiter, Russel J., et al. "Melatonin as a radioprotective agent: a review." *International Journal of Radiation Oncology* Biology* Physics* 59.3 (2004): 639-653.
  - sleep sanctuary (only if you ground the net and switch off the fuses for the bedroom)
  - switch off WiFi, if possible switch off all fuses
  - Samina bed system. Elevate bedposts at head end 12 cm for better lymphatic drainage
  - Russian “Torsion Field Corrector” (plug in version)
  - no computer use after sunset - or get red filter:
    Download “Iris-mini-0.3.0-Installer-Windows”iris-mini-0.3.0-installer.exe

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

Toxin #4: pathogens (diagnose or assume they are all there)

Pathogens are with us whenever our interstitium has become toxic

In autistic children we almost always find:

- Parasites: treat with Rizol Gamma 10 drops tid, consider also as enema
- Mold: Cistus tea, 6-8 cups/day
- Lyme and co-infections acquired in the womb (gestational): treat with KiVita: 3 dropperful 3 times/day, Cistus tea sweetened with Stevia (6-8 cups/day)
- HERV (human endogenous retroviruses) and acquired retroviruses: treat with Cistus, Baikalin, Broccoli sprout extract and RetroV powder (see recommendations)
- Amyloid and prions: Briotech HOCL as inhalation, oral and nasal drops - especially with PANS/PANDAS

We are losing generations of children to ill health and neurological illness. The Elephant in the Living Room: Autism, chronic illness and Retroviral activity

- **Human endogenous retroviruses** (HERVs) have been in our DNA for millenia – but silenced by methylation, acetylation and histone modification. However, the current environmental influences are crippling these wonderful mechanisms, activating this sleeping giant: radiowaves from WiFi, agrochemicals, heavy metals. WiFi has been shown by Prof.emeritus M.Pall to drive calcium into the cells (especially the neurons) causing a cascade of inflammatory events with the formation of destructive peroxynitrite – explaining all features of autism: microglia activation, uncontrollable parasites and infections, inability to detox
- Torres, Anthony R., Alma Maciulis, and Dennis Odell. "The association of **MHC genes** with **autism**." Front Biosci 6 (**2001**): D936-43.
- Giles, T., Washington SJ Boyer-Lehnert, and Ohio P. Bassin. "Is **A Retrovirus The Root Cause Of The Autism** Epidemic?." (**2011**).
- Balestrieri, E., Arpino, C., Matteucci, C., Sorrentino, R., Pica, F., Alessandrelli, R., Coniglio, A., Curatolo, P., Rezza, G., Macciardi, F. and Garaci, E., **2012**. HERVs expression in autism spectrum disorders. PloS one, 7(11), p.e48831. From the discussion: "***To the best of our knowledge, this is the first evidence linking retrotransposon activity and ASD***"
- Balestrieri, E., Cipriani, C., Matteucci, C., Capodicasa, N., Pilika, A., Korca, I., ... & Coniglio, A. (**2016**). Transcriptional activity of human **endogenous retrovirus** in Albanian children with **autism spectrum disorders**. The new microbiologica, 39(3), 228.
- Cipriani, Chiara, Laura Ricceri, Claudia Matteucci, Alessia De Felice, Anna Maria Tartaglione, Ayele Argaw-Denboba, Francesca Pica et al. "***High expression of Endogenous Retroviruses from intra adulthood in two mouse models of Autism Spectrum Disorders***." Scientific reports 8, nc

MMR contains human fetal DNA and Retroviruses

Deisher, Theresa A., Ngoc V. Doan, Kumiko Koyama, and Sarah Bwabye. "Epidemiologic and molecular relationship between vaccine manufacture and autism spectrum disorder prevalence." Issues in law & Medicine [01 Jan 2015, 30(1):47-70] (PMID:26103708)

Abstract

OBJECTIVES: To assess the public health consequences of fetal cell line manufactured vaccines that contain residual human fetal DNA fragments utilizing laboratory and ecological approaches including statistics, molecular biology and genomics. METHOD: MMR coverage and autism disorder or autism spectrum disorder prevalence data for Norway, Sweden and the UK were obtained from public and government websites as well as peer reviewed published articles. Biologically, the size and quantity of the contaminating fetal DNA in Meruvax II and Havrix as well as the propensity of various cell lines for cellular and nuclear uptake of primitive human DNA fragments were measured and quantified using gel electrophoresis, fluorescence microscopy and fluorometry. Lastly, genomic analysis identified the specific sites where fetal DNA fragment integration into a child's genome is most likely to occur. RESULTS: The average MMR coverage for the three countries fell below 90% after Dr. Wakefield's infamous 1998 publication but started to recover slowly after 2001 until reaching over 90% coverage again by 2004. During the same time period, the average autism spectrum disorder prevalence in the United Kingdom, Norway and Sweden dropped substantially after birth year 1998 and gradually increased again after birth year 2000. Average single stranded DNA and double stranded DNA in Meruvax II were 142.05 ng/vial and 35.00 ng/vial, respectively, and 276.00 ng/vial and 35.74 ng/vial in Havrix respectively. The size of the fetal DNA fragments in Meruvax II was approximately 215 base pairs. There was spontaneous cellular and nuclear DNA uptake in HFF1 and NCCIT cells. Genes that have been linked to autism (autism associated genes; AAGs) have a more concentrated susceptibility for insults to genomic stability in comparison to the group of all genes contained within the human genome. Of the X chromosome AAGs, 15 of 19 have double strand break motifs less than 100 kilobases away from the center of a meiotic recombination hotspot located within an exon.

CONCLUSION: Vaccines manufactured in human fetal cell lines contain unacceptably high levels of fetal DNA fragment contaminants. The human genome naturally contains regions that are susceptible to double strand break formation and DNA insertional mutagenesis. The "Wakefield Scare" created a natural experiment that may demonstrate a causal relationship between fetal cell-line manufactured vaccines and ASD prevalence.

Vaccines today contain retroviral sequences with predictable adverse consequences

Issues Law Med. 2016 Fall;31(2):221-234.

Insertional mutagenesis and autoimmunity induced disease caused by human fetal and retroviral residual toxins in vaccines.

Jarzyna P[1], Doan NV[1], Deisher TA[1].

Article excerpt

Introduction

Major concern of vaccination regarding childhood diseases in terms of Insertional Mutagenesis and Autoimmunity

The potential consequences of injecting our children with human fetal DNA contaminants include two well-established pathologies:

1) Insertional mutagenesis in which fetal DNA incorporates into the child's DNA **causing mutations**.

2) **Autoimmune disease triggered by the human fetal DNA in vaccines** leading a child's immune system to attack his or her own body.

Vaccines: The United States manufacturing process and vaccine history of using human fetal cell lines: In January 1979, the rubella manufacturing switch from animal based to the human fetal cell line WI-38 was approved by the FDA

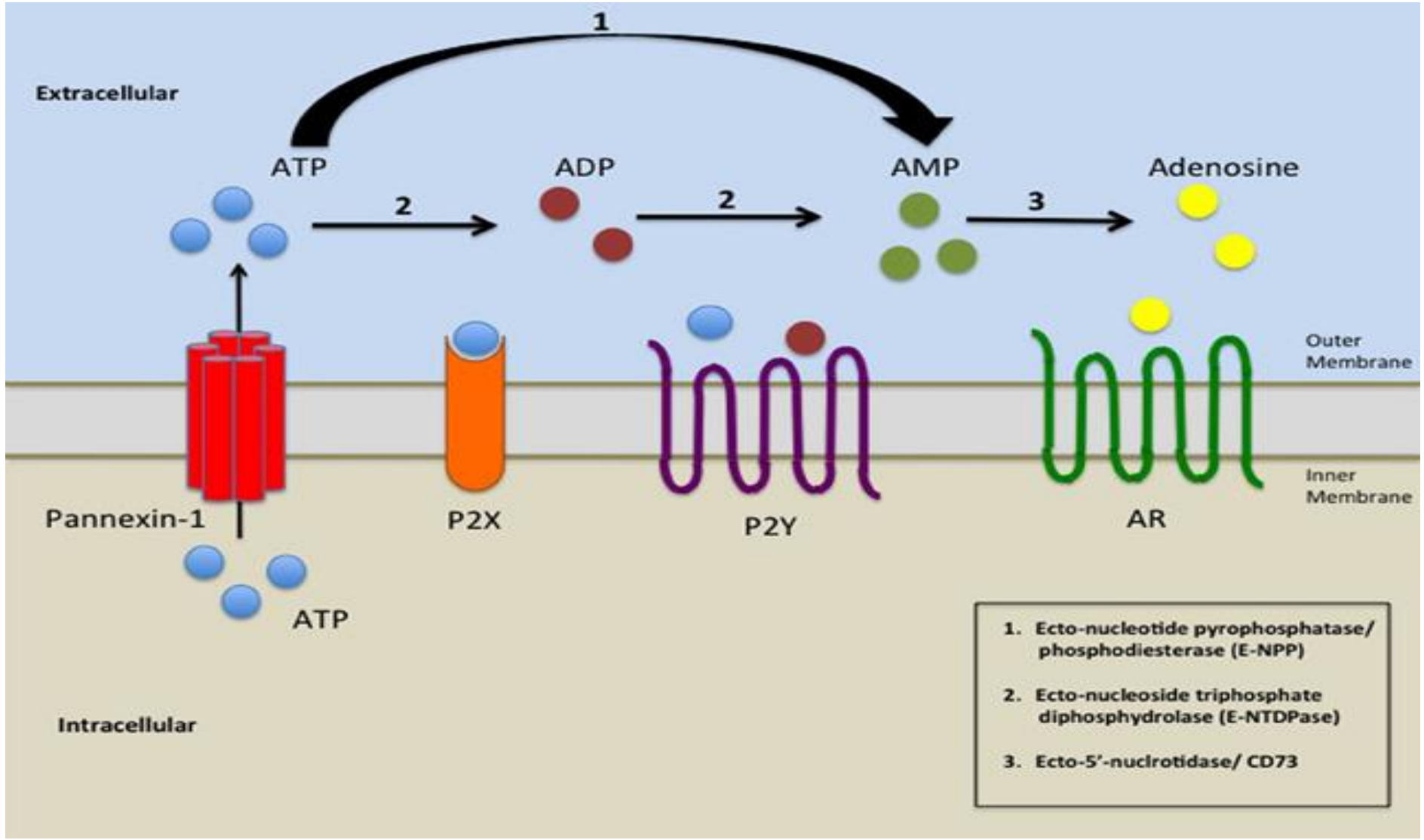
Retroviruses cause brain inflammation and elicit the “cell danger reponse”

Johnston, James B., et al. "Monocyte activation and differentiation augment **human endogenous retrovirus expression: implications for inflammatory brain diseases**." *Annals of neurology* 50.4 (2001): 434-442.

Abstract

Human endogenous retroviruses (HERVs) have been implicated as causative agents in diseases characterized by inflammation and macrophage activation, such as multiple sclerosis. Because monocyte activation and differentiation influence retroviral transcription and replication, we investigated the contribution of these processes to the expression of four HERV families (HERV-W, HERV-K, HERV-E, and HERV-H) in human monocytes and autopsied brain tissue from patients with brain diseases associated with increased macrophage activity. Reverse transcriptase-polymerase chain reaction analysis of primary macrophages and U937 monocytoid cells stimulated with phorbol-12-myristate-13-acetate or lipopolysaccharide revealed **three- to ninefold increases in HERV-W, HERV-K, and HERV-H RNA levels**. In addition, **elevated reverse transcriptase activity** and HERV RNA were detectable in supernatants from PMA-stimulated U937 cultures, properties that could be attenuated with the inhibitor of monocyte differentiation threonine-lysine-proline. In contrast, stimulation of monocytes decreased or had no effect on HERV-E expression. Compared with controls, HERV-W and HERV-K expression was increased in brain tissue from patients with multiple sclerosis or human immunodeficiency virus infection or AIDS, with concomitant elevated tumor necrosis factor-α levels. Similarly, elevated HERV-W levels were detected in patients with Alzheimer's dementia only when tumor necrosis factor-α expression was also evident (2 of 6 cases). The **detection of several HERVs in inflammatory brain diseases** and the capacity to augment HERV expression in monocytes with compounds that influence cellular activity suggest that increased expression of these viruses is a consequence of increased immune activity.

ATP from damaged cells as extracellular messenger signals the cell danger response. It is degraded to adenosin, which is anti inflammatory or immunosuppressive. Degradation of ATP by the ectonucleotidases (CD 39 and CD 73). The Purine receptor P2Y becomes dysregulated.



Autism and Prions/Beta Amyloid

The question, if prions might be involved in autism has never been disproven!

Taylor, J. Paul, John Hardy, and Kenneth H. Fischbeck. "Toxic proteins in neurodegenerative disease." *Science* 296.5575 (2002): 1991-1995.

Sokol, Deborah K., et al. "High levels of Alzheimer beta-amyloid precursor protein (APP) in children with severely autistic behavior and aggression." *Journal of child neurology* 21.6 (2006): 444-449.

Abstract: Autism is characterized by restricted, repetitive behaviors and impairment in socialization and communication. Although no neuropathologic substrate underlying autism has been found, the findings of brain overgrowth via neuroimaging studies and increased levels of brain-derived neurotrophic factor (BDNF) in neuropathologic and blood studies favor an anabolic state. We examined acetylcholinesterase, plasma neuronal proteins, secreted beta-amyloid precursor protein (APP), and amyloid-beta 40 and amyloid-beta 42 peptides in children with and without autism. Children with severe autism and aggression expressed secreted beta-amyloid precursor protein at two or more times the levels of children without autism and up to four times more than children with mild autism. There was a trend for children with autism to show higher levels of secreted beta-amyloid precursor protein and nonamyloidogenic secreted beta-amyloid precursor protein and lower levels of amyloid-beta 40 compared with controls. This favors an increased α-secretase pathway in autism (anabolic), opposite to what is seen in Alzheimer disease. Additionally, a complex relationship between age, acetylcholinesterase, and plasma neuronal markers was found. (*J Child Neurol* 2006;21:444—449; DOI 10.2310/7010.2006.00130).

Wild, Edward J., and Sarah J. Tabrizi. "The differential diagnosis of chorea." *Practical neurology* 7.6 (2007): 360-373.

From the text about PANDAS: that … **prion disease** caused by an 8-octapeptide repeat insertion in the gene encoding **prion** protein (PRNP) … It occurs in **children**, mainly girls

Misfolding infectious proteins (prions and amyloid) in autism

A. Diagnosis: Kuehn, Bridget M. "Rapid Prion Test." *JAMA* 305.4 (2011): 348-348.

B. Treatment:

1. Stabilized isotonic HOCL (from BrioTech):

Hughson, A. G., Race, B., Kraus, A., Sangaré, L. R., Robins, L., Groveman, B. R., ... & Manca, M.

Inactivation of prions and amyloid seeds with hypochlorous acid. *PLoS pathogens*, *12*(9), e1005914. (2016).

Hypochlorous acid (HOCl) is produced naturally by neutrophils and other cells to kill conventional microbes *in vivo*. Synthetic preparations containing HOCl can also be effective as microbial disinfectants. Here we have tested whether HOCl can also inactivate prions and other self-propagating protein amyloid seeds. Prions are deadly pathogens that are notoriously difficult to inactivate, and standard microbial disinfection protocols are often inadequate. Recommended treatments for prion decontamination include strongly basic (pH ≥~12) sodium hypochlorite bleach, ≥1 N sodium hydroxide, and/or prolonged autoclaving. These treatments are damaging and/or unsuitable for many clinical, agricultural and environmental applications. We have tested the anti-prion activity of a weakly acidic aqueous formulation of HOCl (BrioHOCl) that poses no apparent hazard to either users or many surfaces. For example, BrioHOCl can be applied directly to skin and mucous membranes and has been aerosolized to treat entire rooms without apparent deleterious effects. Here, we demonstrate that immersion in BrioHOCl can inactivate not only a range of target microbes, including spores of *Bacillus subtilis*, but also prions in tissue suspensions and on stainless steel. Real-time quaking-induced conversion (RT-QuIC) assays showed that BrioHOCl treatments eliminated all detectable prion seeding activity of human Creutzfeldt-Jakob disease, bovine spongiform encephalopathy, cervine chronic wasting disease, sheep scrapie and hamster scrapie; these findings indicated reductions of $\geq 10^3$- to 10^6-fold. Transgenic mouse bioassays showed that all detectable hamster-adapted scrapie infectivity in brain homogenates or on steel wires was eliminated, representing reductions of $\geq$~$10^{5.75}$-fold and >10^4-fold, respectively. Inactivation of RT-QuIC seeding activity correlated with free chlorine concentration and higher order aggregation or destruction of proteins generally, including prion protein. BrioHOCl treatments had similar effects on amyloids composed of human α-synuclein and a fragment of human tau. These results indicate that HOCl can block the self-propagating activity of prions and other amyloids.

2. Melatonin:

Lahiri DK , Chen D. , Ge YW , et al: Dietary supplementation with melatonin reduces levels of Amyloid beta-peptides in the murine cerebral cortex. J Pineal Res 2004;36:224—231.

Rossignol, Daniel A., and Richard E. Frye. "Melatonin in autism spectrum disorders: a systematic review and meta-analysis." *Developmental Medicine & Child Neurology* 53.9 (2011): 783-792.

The aim of this study was to investigate melatonin-related findings in autism spectrum disorders (ASD), including autistic disorder, Asperger syndrome, Rett syndrome, and pervasive developmental disorders, not otherwise specified.

Method Comprehensive searches were conducted in the PubMed, Google Scholar, CINAHL, EMBASE, Scopus, and ERIC databases from their inception to October 2010. Two reviewers independently assessed 35 studies that met the inclusion criteria. Of these, meta-analysis was performed on five randomized double-blind, placebo-controlled studies, and the quality of these trials was assessed using the Downs and Black checklist.

Results Nine studies measured melatonin or melatonin metabolites in ASD and all reported at least one abnormality, including an abnormal melatonin circadian rhythm in four studies, below average physiological levels of melatonin and/or melatonin derivates in seven studies, and a positive correlation between these levels and autistic behaviors in four studies. Five studies reported gene abnormalities that could contribute to decreased melatonin production or adversely affect melatonin receptor function in a small percentage of children with ASD. Six studies reported improved daytime behavior with melatonin use. Eighteen studies on melatonin treatment in ASD were identified; these studies reported improvements in sleep duration, sleep onset latency, and night-time awakenings. Five of these studies were randomized double-blind, placebo-controlled crossover studies; two of the studies contained blended samples of children with ASD and other developmental disorders, but only data for children with ASD were used in the meta-analysis. The meta-analysis found significant improvements with large effect sizes in sleep duration (73min compared with baseline, Hedge's *g* 1.97 [95% confidence interval {CI} CI 1.10–2.84], Glass's Δ 1.54 [95% CI 0.64–2.44]; 44min compared with placebo, Hedge's *g* 1.07 [95% CI 0.49–1.65], Glass's Δ 0.93 [95% CI 0.33–1.53]) and sleep onset latency (66min compared with baseline, Hedge's *g*−2.42 [95% CI −1.67 to −3.17], Glass's Δ−2.18 [95% CI −1.58 to −2.76]; 39min compared with placebo, Hedge's *g*−2.46 [95% CI −1.96 to −2.98], Glass's Δ−1.28 [95% CI −0.67 to −1.89]) but not in night-time awakenings. The effect size varied significantly across studies but funnel plots did not indicate publication bias. The reported side effects of melatonin were minimal to none. Some studies were affected by limitations, including small sample sizes and variability in the protocols that measured changes in sleep parameters.

Interpretation Melatonin administration in ASD is associated with improved sleep parameters, better daytime behavior, and minimal side effects. Additional studies of melatonin would be helpful to confirm and expand on these findings

The problem with the vaccines grown on human DNA: a desperate call for cleaner vaccines (Japan had them already, but was "discouraged" to continue producing them

- Jarzyna, Peter, Ngoc V. Doan, and Theresa A. Deisher. "Insertional mutagenesis and autoimmunity induced disease caused by human fetal and retroviral residual toxins in vaccines." *Issues L. & Med.* 31 (2016): 221.
- Objectives • Understand vaccine manufacturing and cell substrate residual contaminant levels. • Gain knowledge regarding species specific insertional mutagenesis and autoimmunity. • Understand the relationship of these pathological processes to current childhood disease epidemics including autistic disorder, leukemia, lymphoma, intellectual disability, schizophrenia and bipolar disorder. Introduction Major concern of vaccination regarding childhood diseases in terms of Insertional Mutagenesis and Autoimmunity The potential consequences of injecting our children with human fetal DNA contaminants include two well-established pathologies: **1) Insertional mutagenesis in which fetal DNA incorporates into the child's DNA causing mutations. 2) Autoimmune disease triggered by the human fetal DNA in vaccines leading a child's immune system to attack his or her own body.**
- Deisher, T. A., Doan, N. V., Koyama, K., & Bwabye, S. (2015). Epidemiologic and molecular relationship between vaccine manufacture and autism spectrum disorder prevalence. *Issues L. & Med.*, *30*, 47.
- Deisher, T. A., Doan, N. V., Omaiye, A., Koyama, K., & Bwabye, S. (2014). Impact of environmental factors on the prevalence of autistic disorder after 1979. *Journal of Public Health and Epidemiology*, *6*(9), 271-286.
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Naviaux, Robert K., et al. "**Low-dose suramin in autism spectrum disorder**: a small, phase I/II, randomized clinical trial." *Annals of clinical and translational neurology* 4.7 (2017): 491-505.

Abstract: No drug is yet approved to treat the core symptoms of autism spectrum disorder (ASD). Low-dose suramin was effective in the maternal immune activation and Fragile X mouse models of ASD. The Suramin Autism Treatment-1 (SAT-1) trial was a double-blind, placebo-controlled, translational pilot study to examine the safety and activity of low-dose suramin in children with ASD.

Methods: Ten male subjects with ASD, ages 5–14 years, were matched by age, IQ, and autism severity into five pairs, then randomized to receive a single, **intravenous infusion of suramin (20 mg/kg)** or saline. The primary outcomes were ADOS-2 comparison scores and Expressive One-Word Picture Vocabulary Test (EOWPVT). Secondary outcomes were the aberrant behavior checklist, autism treatment evaluation checklist, repetitive behavior questionnaire, and clinical global impression questionnaire.

Results: Blood levels of suramin were 12 ± 1.5 μmol/L (mean ± SD) at 2 days and 1.5 ± 0.5 μmol/L after 6 weeks. The terminal half-life was 14.7 ± 0.7 days. A self-limited, asymptomatic rash was seen, but there were no serious adverse events**. ADOS-2 comparison scores improved** by −1.6 ± 0.55 points (n = 5; 95% CI = −2.3 to −0.9; Cohen's d = 2.9; P = 0.0028) in the suramin group and did not change in the placebo group. Secondary outcomes also showed **improvements in language, social interaction, and decreased restricted or repetitive behaviors.**

Interpretation: The safety and activity of low-dose suramin showed promise as a novel approach to treatment of ASD in this small study. We hypothesized that **there is a conserved cellular response to metabolic perturbation or danger that is shared by all children with ASD**.

This is called the cell danger hypothesis.

Towards an affordable, safe and effective retroviral treatment

1. Melatonin

Zhang, Z., et al. *Immunology* 96.2 (1999): 291. "Prevention of immune dysfunction and vitamin E loss by dehydroepiandrosterone and **melatonin supplementation** during murine **retrovirus infection**."

ABSTRACT

Female C57BL/6 mice infected with the LP-BM5 leukaemia retrovirus developed murine acquired immune-deficiency syndrome (AIDS). Dehydroepiandrosterone (DHEA) and melatonin (MLT) modify immune dysfunction and prevent lipid peroxidation. We investigated whether DHEA and MLT could prevent immune dysfunction, excessive lipid peroxidation, and tissue vitamin E loss induced by retrovirus infection. Retrovirus infection inhibited the release of T helper 1 (Th1) cytokines, stimulated secretion of Th2 cytokines, increased hepatic lipid peroxidation, and induced vitamin E deficiency. Treatment with DHEA or MLT alone, as well as together, largely prevented the reduction of B- and T-cell proliferation as well as of Th1 cytokine secretion caused by retrovirus infection. Supplementation also suppressed the elevated production of Th2 cytokines stimulated by retrovirus infection. DHEA and MLT simultaneously reduced hepatic lipid peroxidation and prevented vitamin E loss. The use of DHEA plus MLT was more effective in preventing retrovirus-induced immune dysfunction than either DHEA or MLT alone. **These results suggest that supplementation with DHEA and MLT may prevent cytokine dysregulation, lipid oxidation and tissue vitamin E loss induced by retrovirus infection.** Similarly, hormone supplementation also modified immune function and increased tissue vitamin E levels in uninfected mice

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Transdermal Melatonin: VitaHealth Apothecary in New York, tel: 001-212 6281110.

- Adult dosing: initially 500 mg or more. Permanent protective dose: 125-250 mg
- Children: initial dosing 250 mg. Permanent dose: 80 mg.
- For the first 3-6 months strong detox reactions are to be expected and should be dealt with - with the help of a practitioner

2. Cistus Incanus Tea as anti-retroviral remedy (www.kiScience.com)

Stephanie Rebensburg, Markus Helfer, Martha Schneider, Herwig Koppensteiner, Josef Eberle, Michael Schindler, Lutz Gürtler, Ruth Brack-Werner. **Potent in vitro antiviral activity of Cistus incanus extract against HIV and Filoviruses targets viral envelope proteins**. *Scientific Reports*, 2016; 6: 20394 DOI: 10.1038/srep20394

Scientists at the Helmholtz Zentrum München discover that extracts of the medicinal plant *Cistus incanus* (Ci) prevent human immunodeficiency viruses from infecting cells. Active antiviral ingredients in the extracts inhibit docking of viral proteins to cells. Antiviral activity of *Cistus* extracts also targets Ebola- and Marburg viruses.

HIV: broad activity, no resistance

The Brack-Werner team found potent activity of Ci extracts acted against a broad spectrum of clinical HIV-1 and HIV-2 isolates. This also included a virus isolate resistant against most available drugs. "Antiviral ingredients of Ci extracts target viral envelope proteins on infectious particles and prevent them from contacting host cells," Brack-Werner explains. No resistant viruses were detected during long-term treatment (24 weeks) with Ci extract, indicating that Ci extract attacks viruses without causing resistance. Since antiviral activity of Ci extracts differs from all clinically approved drugs, Ci-derived products could be an important complementation to current established drug regimens,

Cistus for Lyme, mold and anti-biofilm

Rauwald, H. W., et al. "On the **antispirochaetal activity** of manoyloxides and carvacrol from the oleoresin labdanum of Cistus creticus L." Planta Medica 79.13 (2013): PN53.

Bouamama, H., et al. "Antibacterial and **antifungal activities** of **Cistus incanus** and C. monspeliensis leaf extracts." Therapie 54.6 (1999): 731-733..

Cistus is also a very effective **biofilm breaker**, unmasking persistent infections:

Hannig, Christian, et al. "Effects of **Cistus-tea** on bacterial colonization and enzyme activities of the in situ pellicle." Journal of dentistry 36.7 (2008): 540-545.

Whole leaf Stevia has been shown in a study by Northwest University in the US to be as effective or more effective in the treatment of Lyme disease than triple antibiotic therapy, including the use of Daptomycin:

Effectiveness of **Stevia Rebaudiana Whole Leaf Extract** Against the Various Morphological Forms of **Borrelia Burgdorferi** in Vitro. Eur J Microbiol Immunol (Bp). 2015 Dec ;5(4):268-80. Epub 2015 Nov 12; P A S Theophilus, M J Victoria, K M Socarras, K R Filush, K Gupta, D F Luecke, E Sapi

SophiaFlow: apply transdermal cream twice daily to the front of the neck (contains bioactive molecules; from www.SophiaNutrition.com)

3. “RetroV Powder” anti-retroviral mix (www.KiScience.com)

a. Baikalin extract from Scullcap root/Scutalaria

- Li, B. Q., T. Fu, Y. D. Yan, N. W. Baylor, F. W. Ruscetti, and H. F. Kung. "Inhibition of HIV infection by baicalin--a flavonoid compound purified from Chinese herbal medicine." *Cellular & molecular biology research* 39, no. 2 (1993): 119-124.
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b. ST John’s Wort

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- Therapeutic agents with dramatic antiretroviral activity and little toxicity at effective doses: aromatic polycyclic diones hypericin and pseudohypericin.
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c. Green Tea

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d. Reishi Mushroom

- Min, B. S., Nakamura, N., Miyashiro, H., BAE, K. W., & Hattori, M. (1998). Triterpenes from the spores of Ganoderma lucidum and their inhibitory activity against HIV-1 protease. *Chemical and Pharmaceutical Bulletin*, *46*(10), 1607-1612.

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- Balzarini, J., Neyts, J., Schols, D., Hosoya, M., Van Damme, E., Peumans, W., & De Clercq, E. (1992).The mannose-specific plant lectins from Cymbidium hybrid and Epipactis helleborine and the (N-acetylglucosamine) n-specific plant lectin from Urtica dioica are potent and selective inhibitors of human immunodeficiency virus and cytomegalovirus replication in vitro. *Antiviral research*, *18*(2), 191-207**.**

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g. Bitter Melon

- Trivedi, R. V., Wadher, K. J., Taksande, J. B., Mahore, J. G., & Umekar, M. J. (2011). Momordica charantia: A Natural and Safe Approach for the Treatment of HIV Infection. *Int J of Pharm Tech Research*, *3*, 1660-1666.
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4. Broccoli sprouts

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Other uses of higher dose melatonin

Melatonin is mostly known for ist mild sleep inducing effect at dosages of 0.25 - 3 mg

To induce more long lasting sleep, use transdermal melatonin (Aeschbach, D., et al. "Use of **transdermal melatonin delivery** to improve sleep maintenance during daytime." Clinical Pharmacology & Therapeutics 86.4 (2009): 378-38) zum anderen gibt es aber auch zahlreiche andere Heilwirkungen.

To minimize birthtrauma and decompression injury from divers (Aridas, James DS, et al. "Systemic and **transdermal melatonin administration prevents neuropathology in response to perinatal asphyxia** in newborn lambs." *Journal of pineal research* 64.4 (2018): e12479). This research has helped us understand why high dose TD Melatonin is a miracle for **spinal chord injuries** and infectious issues such as transverse myelitis from Bartonella or Lyme (300-500 mg)

Cancer: Blask, David E., Leonard A. Sauer, and Robert T. Dauchy. "Melatonin as a **chronobiotic/anticancer agent**: cellular, biochemical, and molecular mechanisms of action and their implications for circadian-based cancer therapy." *Current topics in medicinal chemistry* 2.2 (2002): 113-132.)

Jung-Hynes, Brittney, et al. "Melatonin, a novel Sirt1 inhibitor, imparts antiproliferative effects against **prostate cancer** in vitro in culture and in vivo in TRAMP model." *Journal of pineal research* 50.2 (2011): 140-149.

The ultimate repair agent for **mitochondrial damage** - that's all of us! (Andrabi, Shaida A., et al. "Direct inhibition of the mitochondrial permeability transition pore: a possible mechanism responsible for anti-apoptotic effects of melatonin." *The FASEB journal* 18.7 (2004): 869-871)

„Activation of GABA¬A receptor Protects Mitochondria and Reduces **Cerebral ischemia**". Neetu Tyagi et al., FASEB J)

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