Chronic Illness Handout

**Retroviruses**

What are retroviruses? The more familiar DNA viruses such as those from the “herpes family” - and many others - work their way from the DNA over to the RNA and from there to the manufacture of viral proteins. Retroviruses work their way backwards – from the RNA to the DNA – and then forward again from there. The most well-known and published retrovirus is HIV. However, there are countless others, grouped together in various “classes”. The generally accepted key contributors to chronic illness are inflammation, oxidative stress and microbial infection. All of these are known triggers for retroviral activity, and in turn also known causes of retroviral activity.

Both human and animal retroviruses can infect the CNS. These are associated with many diseases of the CNS causing neurological disease directly through infection of immune cells which traffic to the brain and indirectly through increases in proinflammatory cytokines and chemokines or in the absence of detectable brain inflammation indirect effects known as “bystander effects”- causing chronic retroviral replication of immune cells.

A retrovirus works via the enzyme “reverse transcriptase”. Once inside the cell, it uses the enzyme to force the cell to create viral DNA. This viral DNA becomes integrated into the host-cell DNA. A retrovirus integrated into our genome may be passed from mother to child during pregnancy (Sakuma et al.,2012). Only 2 % or our DNA is protein-coding, but 6-8% of our DNA is retroviral DNA – passed down to us from our ancestors as battle-scars from our constant encounter with an often hostile microbial and virus-rich environment (Stoyle.,2006, Mayer et al.,2011; Li et al.,2001). These viruses are referred to as Human Endogenous Retroviruses or HERVs. An unintended source of retroviruses are some vaccines (Frontiers in Microbiology, January 2011).

The retroviruses are subdivided in different-lettered classes: Beta Retroviruses: HERV -K. Gamma Retroviruses: HERV -H and HERV-W. Human endogenous retroviruses (HERVs) make up part of our genome (4-8%) and represent footprints of previous retroviral infection (length of HERV in a single patient: 200 000 times round the earth). HERVs possess a similar genomic organisation to present-day exogenous retroviruses (from tick and insect bites, vaccines, etc.).The HERV-K “superfamily” represents one of the most active HERVs and is capable of reproducing retroviral particles, i.e symptoms. HERVs may be of benefit to the host (p53, placenta; “Beneficial role of human endogenous retroviruses: facts and hypotheses”. Scan J Immunol 1998; 48:329-38), but can also be harmful, involved in cancer, autoimmune disease, fatigue and many other debilitating symptoms. Retroviruses can also be aquired – usually in the company of bacterial or other viral infections ( Borrelia, Bartonella) or viral infections: flavi viruses (FSME, zika, some flus, dengue, etc.), EBV, HSV-1/2. Transmission from insect bites, contact, vaccines etc.

References: “The viruses in all of us: characteristics and biological significance of human endogenous retrovirus sequences”; Proc Natl Acad Sci USA 1996; 93: 5177-84

“Demystified: Human endogenous retroviruses”; Mol Pathol 2003 Feb; 56(1): 11-18; PN Nelson et al

Diagnosis:

Curently no PCR test or definitive other test (IgG/IgM etc.) test is available. The following markers - based on the currently available science - are what I use at the Sophia Health Institute (SHI): **IL-8** (upregulated); elevated extracellular **ATP and ADP** from cell danger response, **CD39 and CD73** elevated (Ectonucleotidases to break down ATP, ADP and AMP)**, CD26** (Maina, Ayub K., et al. "The Potential for DPP-4/CD26 usage as a surrogate marker for Antiretroviral Therapy Efficacyin HIV Infected populations." *African Journal of Pharmacology and Therapeutics* 5.4 (2017)). **low wbc; elevated nagalase** (Bradstreet, James Jeffrey, Emar Vogelaar, and Lynda Thyer. "Initial observations of elevated alpha-N-acetylgalactosaminidase activity associated with autism and observed reductions from Gc protein--macrophage activating factor injections." *Autism Insights* 4 (2012): 31.)

Elevated **RANTES** (Gordon, Cynthia J., et al. "Enhancement of human immunodeficiency virus type 1 infection by the CC-chemokine RANTES is independent of the mechanism of virus-cell fusion." *Journal of virology* 73.1 (1999): 684-694.)

Treatment to reduce retroviral activity:

**1. Broccoli sprouts:**

[Singh K, Zimmerman AW. Sulforaphane Treatment of Young Men with Autism Spectrum Disorder. CNS Neurol Disord Drug Targets. 2016;15(5):597-601.](https://www.ncbi.nlm.nih.gov/pubmed/27071786)

Singh, K., Connors, S. L., Macklin, E. A., Smith, K. D., Fahey, J. W., Talalay, P., & Zimmerman, A. W. (2014). Sulforaphane treatment of autism spectrum disorder (ASD). *Proceedings of the National Academy of Sciences*, October 2014; *111*(43), 15550-15555. from the text: . “Sulforaphane, which showed negligible toxicity, was selected because it **upregulates genes that protect aerobic cells against oxidative stress, inflammation, and DNA-damage**, all of which are prominent and possibly mechanistic characteristics of ASD”

Tester, Jodie, and Tessa Finney-Brown. "Sulforaphane treatment of autism spectrum disorder." *Australian Journal of Herbal Medicine* 27.1 (2015): 41.

[Armah CN, Traka MH, Dainty JR, Defernez M, Janssens A, Leung W, Doleman JF, Potter JF, Mithen RF. A diet rich in high-glucoraphanin broccoli interacts with genotypeto reduce discordance in plasma metabolite profiles by modulating **mitochondrial function**. Am J Clin Nutr. 2013 Sep;98(3):712-22.](https://www.ncbi.nlm.nih.gov/pubmed/23964055)

[Yang L, Palliyaguru DL, Kensler TW. Frugal chemoprevention: targeting **Nrf2** with foods rich in sulforaphane. Semin Oncol. 2016 Feb;43(1):146-153.](https://www.ncbi.nlm.nih.gov/pubmed/26970133)

[Heiss E, Herhaus C, Klimo K, Bartsch H, Gerhäuser C. Nuclear factor kappa B is a molecular target for sulforaphane-mediated **anti-inflammatory** mechanisms. J Biol Chem. 2001 Aug 24;276(34):32008-15.](https://www.ncbi.nlm.nih.gov/pubmed/11410599)

[Gan N, Wu YC, Brunet M, Garrido C, Chung FL, Dai C, Mi L. Sulforaphane activates **heat shock response** and **enhances proteasome activity** through up-regulation of Hsp27. J Biol Chem. 2010 Nov 12;285(46):35528-36.](https://www.ncbi.nlm.nih.gov/pubmed/20833711)

**2. Sardinian Cistus Incanus Tea** (source: [www.KiScience.com](http://www.kiscience.com/)) as powerful treatment strategy

A study in Scientific Reports from 2016 shows Ci to not only be effective in a brilliant invitro study against the worst of the retroviruses, HIV, but to be effective in the most drug resistant cases:

*Rebensburg, S., Helfer, M., Schneider, M., Koppensteiner, H., Eberle, J., Schindler, M., ... & Brack-Werner, R. (2016).* ***Potent in vitro antiviral activity*** *of Cistus incanus extract against* ***HIV and Filoviruses*** *targets viral envelope proteins. Scientific reports, 6, 20394.*

It has also been shown to be effective against **Ebola, the flu virus and Lyme/Borrelia**

Labdanum from Mediterranean Cistus species:

*GC-MS fingerprints and relative quantification of* ***Antispirochaetal*** *manoyloxides. Planta Medica, 78(11), PA10 Kuchta, K., Grötzinger, K., Birkemeyer, C., & Rauwald, H. W. (2012).*

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](http://dx.doi.org/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

**RetroV:** activated herbal powder anti-retroviral mix ([www.KiScience.com](http://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.

Zhao, Qing, Yang Zhang, Gang Wang, Lionel Hill, Jing-Ke Weng, Xiao-Ya Chen, Hongwei Xue, and Cathie Martin. "A specialized flavone biosynthetic pathway has evolved in the medicinal plant, Scutellaria baicalensis." *Science advances* 2, no. 4 (2016): e1501780.

 **b. ST John’s Wort**

Meruelo, Daniel, Gad Lavie, and David Lavie. "Therapeutic agents with dramatic antiretroviral activity and little toxicity at effective doses: aromatic polycyclic diones hypericin and pseudohypericin." *Proceedings of the National Academy of Sciences* 85.14 (1988): 5230-5234.

[Therapeutic agents with dramatic antiretroviral activity and little toxicity at effective doses: aromatic polycyclic diones hypericin and pseudohypericin.](http://www.pnas.org/cgi/pmidlookup?view=long&pmid=2839837&utm_source=TrendMD&utm_medium=cpc&utm_campaign=Proc_Natl_Acad_Sci_U_S_A_TrendMD_0)

D Meruelo et al., Proc Natl Acad Sci U S A

 **c. Green Tea**

Nakane, H., Hara, Y., & Ono, K. (1994). Tea polyphenols as a novel class of inhibitors for human immunodeficiency virus reverse transcriptase.

 **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.

 **e. Stinging Nettle**

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**.**

 **f. Olive Leaf**

Lee-Huang, S., Zhang, L., Huang, P. L., Chang, Y. T., & Huang, P. L. (2003). Anti-HIV activity of olive leaf extract (OLE) and modulation of host cell gene expression by HIV-1 infection and OLE treatment. *Biochemical and Biophysical Research Communications*, *307*(4), 1029-1037.

 **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.

 4 Ng T B, Wong C M, Li W W, Yeung H W. [Isolation and characterization of a galactose binding lectin with insulinomimetic activites from the seeds of the bitter gourd *Momordica charantia* (Cucurbitaceae).](https://www.thieme-connect.com/products/ejournals/linkout/10.1055/s-2001-17359/id/4)  International Journal of Peptide and Protein Research. 1986; 28 163-72

 **Suramin** (Homepathic “homaccord” given orally or intravenous Medical Suramin)

Ruprecht, R. M., Rossoni, L. D., Haseltine, W. A., & Broder, S. (1985). Suppression of retroviral propagation and disease by suramin in murine systems. *Proceedings of the National Academy of Sciences*, *82*(22), 7733-7737.

De Clercq, Erik. "Suramin in the treatment of AIDS: mechanism of action." (1987): 1-10.

Balzarini, J., Mitsuya, H., De Clercq, E., & Broder, S. (1986). Comparative inhibitory effects of suramin and other selected compounds on the infectivity and replication of human T‐cell lymphotropic virus (HTLV‐III)/lymphadenopathy‐associated virus (LAV). *International journal of cancer*, *37*(3), 451-457.

Hamidpour, Rafie, Soheila Hamidpour, Mohsen Hamidpour, Marriam Zarabi, Mahnaz Sohraby, and Mina Shalari. "**Antipurinergic therapy with suramin as a treatment for autism spectrum disorder.**" *Journal of Biomedical Sciences* 5, no. 2 (2016).

Mahoney, C. W., A. Azzi, and K. P. Huang. "Effects of suramin, an anti-human immunodeficiency virus reverse transcriptase agent, on protein kinase C. Differential activation and inhibition of protein kinase C isozymes." *Journal of Biological Chemistry* 265.10 (1990): 5424-5428.

Ruprecht, Ruth M., et al. "Suppression of retroviral propagation and disease by suramin in murine systems." *Proceedings of the National Academy of Sciences* 82.22 (1985): 7733-7737

**Pantethine** (fat soluble form of vitamin B5)

**Pantethine** activates gene acetylation and slows replication of retroviral DNA
Kristensson, Krister. "Microbes' roadmap to neurons." *Nature Reviews Neuroscience* 12.6 (2011): 345. Abstract: The nervous system is protected by barriers that restrict the invasion of pathogens. Nevertheless, mechanisms have evolved by which microbes can pass these barriers, enter and exit neurons and target various regions of the nervous system. In the brain, immune responses to pathogens are generally not robust, so microbes can hide and survive or, conversely, cause severe uncontrolled infections. Depending on their sites of entry and the regions that they target, microbes can cause diverse nervous system dysfunctions and even influence host behaviour to their own advantage. This Review discusses routes by which microbes can reach the nervous system and cause persistent or life-threatening infections.

From the text: … dysfunctions in the neurovascular units. Administration of a low-molecular-weight thiol, **pantethine**, interrupts overproduction of microparticles and **prevents signs of cerebral malaria** in a mouse model 26

**Luteolin** (in GliaLia)

Ko, Yeon-Ju, et al**. "Flavonoids as potential inhibitors of retroviral enzymes**." *Journal of the Korean Society for Applied Biological Chemistry* 52.4 (2009): 321-326.Abstract

Flavonoids are abundant in plants as secondary metabolites where they have antioxidant activities. The diverse and non-specific inhibition of several enzymes, including avian myeloblastosis virus reverse transcriptase (RT), human immunodeficiency virus (HIV) RT, murine leukemia virus RT, HIV-1 protease, trypsin and elastase, were tested for by observing the effects of several flavonoid compounds. Some of the flavonoids were reconfirmed as antiretroviral agents as previously shown by others. Flavonoids belonging to the flavonol or flavone group simultaneously inhibited reverse transcriptases and proteases. On the contrary, flavonoids belonging to the flavanol, isoflavone or flavanone group were less likely to inhibit reverse transcriptase or protease. Vegetables and fruits rich in flavonoids have become integral and even necessary in the animal diet, suggesting that flavonoids may have developed to play beneficiary roles for not only the plants themselves but also the predator animals.

Mehla, Rajeev, Shalmali Bivalkar-Mehla, and Ashok Chauhan. "A flavonoid, **luteolin, cripples HIV-1** by abrogation of tat function." *PLoS One* 6.11 (2011): e27915. Abstract

Despite the effectiveness of combination antiretroviral treatment (cART) against HIV-1, evidence indicates that residual infection persists in different cell types. Intensification of cART does not decrease the residual viral load or immune activation. cART restricts the synthesis of infectious virus but does not curtail HIV-1 transcription and translation from either the integrated or unintegrated viral genomes in infected cells. All treated patients with full viral suppression actually have low-level viremia. More than 60% of treated individuals also develop minor HIV-1 –associated neurocognitive deficits (HAND) due to residual virus and immune activation. Thus, new therapeutic agents are needed to curtail HIV-1 transcription and residual virus.

In this study, **luteolin, a dietary supplement, profoundly reduced HIV-1 infection in reporter cells and primary lymphocytes.** HIV-1inhibition by luteolin was independent of viral entry, as shown by the fact that wild-type and VSV–pseudotyped HIV-1 infections were similarly inhibited. Luteolin was unable to inhibit viral reverse transcription. Luteolin had antiviral activity in a latent HIV-1 reactivation model and effectively ablated both clade-B- and -C -Tat-driven LTR transactivation in reporter assays but had no effect on Tat expression and its sub-cellular localization. We conclude that luteolin confers anti–HIV-1 activity at the Tat functional level. Given its biosafety profile and ability to cross the blood-brain barrier, luteolin may serve as a base flavonoid to develop potent anti–HIV-1 derivatives to complement cART.

Paterniti, Irene, et al. **"Neuroprotection by association of palmitoylethanolamide with luteolin** in experimental Alzheimer’s disease models: the control of neuroinflammation." *CNS & Neurological Disorders-Drug Targets (Formerly Current Drug Targets-CNS & Neurological Disorders)* 13.9 (2014): 1530-1541. Alzheimer’s disease (AD) is the most common neurodegenerative disorder. Its neuropathological hallmarks include deposition of beta amyloid (Aβ) fibrils in senile plaques. Numerous biochemical events, leading to Aβ neurotoxicity in AD, have been proposed and it seems that neuroinflammation plays a prominent role among these. Thus, since inflammatory processes and oxidative stress are considered to play an important role in neuroinflammatory disorders and in AD pathology, in the present work we decided to test a new composite, which is a formulation constituted of an anti-inflammatory compound such as palmitoylethanolamide (PEA) and the well recognized antioxidant flavonoid luteolin (Lut), subjected to an ultra-micronization process, here designated co-ultraPEALut. We investigated the effect of co-ultraPEALut in both an in vitro and ex vivo organotypic model of AD. For the in vitro model, we used human neuronal cells, obtained by differentiating SH-SY5Y neuroblastoma cells into sustainable neuronal morphology. These welldifferentiated cells express features specific to mature neurons, such as synaptic structures and functional axonal vesicle transport, making this new concept for in vitro differentiation valuable for many neuroscientific research areas, including AD. Differentiated SH-SY5Y cells were pre-treated with co-ultraPEALut (reference concentrations: 27, 2.7 and 0.27 µM PEA) for 2 h. AD features were induced by Aβ1-42 stimulation (1 µM). Twenty-four hours later cell vitality was evaluated by the colorimetric MTT assay, whereas the neuroinflammation underling AD was observed by Western blot analysis for IΚBα degradation and nuclear factor-ΚB traslocation, as well as glial fibrillary acidic protein expression. For the organotypic model of AD, hippocampal slice cultures were prepared from mice at postnatal day 6 and after 21 days of culturing the slices were pre-treated with co-ultraPEALut (reference concentrations: 27, 2.7 and 0.27 µM PEA) for 2 h and then incubated with Aβ1-42 (1 µg/ml) for 24 h. Pre-treatment with co-ultraPEALut significantly reduced inducible nitric oxide synthase and glial fibrillary acidic protein expression, restored neuronal nitric oxide synthase and brainderived neurotrophic factor and reduced the apoptosis. Taken together our results clearly showed that co-ultra PEALut is able to blunt Aβ-induced astrocyte activation and to exert a marked protective effect on glial cells. These findings suggest that the association of co-ultraPEALut may provide an effective strategy for AD.

**Selenium** to silence retroviruses

Bologna, Rosa, et al. "Selenium and immunity in HIV-1 infected pediatric patients." *Journal of Nutritional immunology* 3.1 (1994): 41-49.

Baum, Marianna K., et al. "High risk of HIV-related mortality is associated with selenium deficiency." *JAIDS Journal of Acquired Immune Deficiency Syndromes* 15.5 (1997): 370-374.

Campa, Adriana, et al. "Mortality risk in selenium-deficient HIV-positive children." *Journal of acquired immune deficiency syndromes and human retrovirology: official publication of the International Retrovirology Association* 20.5 (1999): 508-513.

Cirelli, Augusto, et al. "Serum selenium concentration and disease progress in patients with HIV infection." *Clinical biochemistry* 24.2 (1991): 211-214.

Dworkin, Brad M. "Selenium deficiency in HIV infection and the acquired immunodeficiency syndrome (AIDS)." *Chemico-biological interactions* 91.2-3 (1994): 181-186.

Schrauzer, Gerhard N., and Juliane Sacher. "Selenium in the maintenance and therapy of HIV-infected patients." *Chemico-Biological Interactions* 91.2-3 (1994): 199-205.

Baeten, Jared M., et al. "Selenium deficiency is associated with shedding of HIV-1--infected cells in the female genital tract." *Journal of acquired immune deficiency syndromes (1999)* 26.4 (2001): 360-364.

***The streamlined Ki- approach to silence the retroviruses:***

1. ***Cistus tea (6-8 cups/day)***
2. ***Broccoli sprout extract (1 heaping tsp twice daily, best in yoghurt. Has to be chewed for enzymatic conversion to sulforaphane)***
3. ***RetroV powder: 1 heaping tsp twice daily together with broccoli extract)***
4. ***Aminoacid-bound selenium (SelenoCysteine,SelenoMethionine) - adult dose: 800 mcg/day***
5. ***Pantethine: 500-1000 mg twice daily (higher dosage in advanced neurological disease)***
6. ***In autism: add Glialia, 1 sachet/day***
7. ***KiVita (herbal antiviral, anti-Lyme and coinfection mix): 2 pipettes 3 times/day***

**Lyme Disease**

Borrelia never comes alone: it is always paired with filoviruses, retroviruses and the more well-known co-infection: mycoplasma, Bartonella, Babesia and Rickettsia

Diagnosis: Armin Labs

Treatment

To treat chronic persistent infections, appropriate biochemistry and good biophysics should be combined in the individual patient protocol. Before engaging with Lyme and Co, we treat the human endogenous retroviruses (HERV) and acquired retroviruses. In our experience the activity of retroviruses creates the amazing difference between a symptomatic Lyme patient and an asymptomatic carrier of Lyme and co-infections.

Step 1: The retroviral treatment + detoxification should be the foundation of a solid Lyme treatment

Binders when the HERV treatmentawakens the immune system, mild to moderate Herxheimer reactions are to be expected, also the release of toxins that were entrapped before. Progress can be facilitated or symptoms can be minimalized by using colonics, lymph drainage and the oral use of agents that bind mobilized toxins and prevent re-absorption.

**Zeolite (Lava Vitae)**: 1/2 tsp 2-3 times daily between meals and/or at bedtime

**Chlorella**: 8-16 tablets (250 mg each) 3 times per day 30 min before meals or at bedtime

As soon as any improvement is noticed, stay with that dose without further increase. If a die-off effect or worsening occurs, go back to the last tolerated dose

Step 2: Taming Borrelia, mould, biofilm and retroviruses with **Stevia sweetened Cistus tea**

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.

Also against mould and other bacteria: 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*

*Step 3: The treatment of persistent Lyme and co-infections*

**Hyaluronic Acid Liquid:** teases microbes out of protected hiding places; nutrient for joints and connective tissue. The KiScience product has the exact Dalton molecular size for complete sublingual uptake. Take 2 dropperfull 3 times daily. If joint pain is the main presenting issue, consider a third dose.

You need a 250 or 500 ml glass jam jar with a lid. Fill with a cup (6 oz) of clean water (or use the daily dose of cistus tea - as outlined above) and add the tinctures into it). Add the entire daily dose of herbals and 1-3 teaspoons (15 ml) **MicroPhos** (phospholipids - known effects: increases cell wall integrity, increases parasympathetic tone, helps anti-microbials to penetrate into biofilm and cross membranes and barriers). Shake vigorously for 1 minute. Take half the daily dose in the morning just before breakfast (to prevent nausea), the rest in divided doses throughout the day; nothing past sunset. It may take months to reach the full treatment dosage. Source: BioPureUS.com

*To increase the depth of penetration of the mix, an ultrasound jewellery cleaning device can be used (costs less than 30.-). Put water in the chamber, place the closed jar with the mixed herbal tinctures into it - after the shaking - and vibrate for 12 minutes*.

*If you suspect* ***mycoplasma****, add 2*-3 tbsp of organic olive oil.

*Slowly titrate doses to tolerance for each item below, in the following order:*

**Artemisia annua** (most important for **Babesia**. Known effects: anti-protozoal, anti-viral, anti-fungal, anti-inflammatory): 1 drop twice daily each, double every 3 days until improvement of any symptom is noticed. If a dose of 2 pipettes twice daily is reached and has to be increased, start a 3rd dose in the middle of the day. If a die-off effect or worsening is noticed, go back to the last tolerated dose and stay there for at least 10 days before attempting to increase the dose again. Do not exceed 2 full pipettes three times per day. Stay on that dose if no further change is noticed and add the next item. Alternatively we use the injection of 60-120 mg artesunate i.v or s.c 2-3 times/week for the more severely ill patient. Treatment can also be intensified by adding artemisinin powder (**artemisia forte** from KiScience - NobelPrize in Medicine 2015): 100-600 mg/day

**KiVita (liposomal anti-Lyme cocktail)** - known effects of the ingredients: anti-bacterial, anti-viral, neuro-protective and neuro-regenerative, anti-aging: start with 1 drop twice daily, titrate to tolerated and effective dose. Full dose> 2-3 pipettes 3 times/day

**Coriandolo** (KiScience - known effects: removal of toxic metals, normalizes serum lipids, increases bile flow): start with 1 drop twice daily, increase to full dose of 2 dropperful 3 times per day as tolerated. This item can also be used separate from the cocktail in a cup of water. Best if combined with 20-30 min ionic foot bath twice weekly.

**Propolis Plus** (KiScience -known effects: anti-viral, radioprotective, anti-bacterial, anti-fungal. In our experience: powerful anti-**Bartonella** effects): titrate carefully to tolerated dosage. Some patients might be allergic to propolis which should be picked up early during initial titration). Full dose: 2 dropperful 3 times per day. We like to add **Calendula** tincture in the treatment of Bartonella (full dose> 1 pipette 3 times/day)

Good additional treatment options:

**Ag23**: this is a unique low potency homeopathic preparation of nanonized silver with a multitude of documented broad spectrum anti-microbial properties. It does not cause silver storage problems (such as skin discoloration) as many colloidal silver products would do, if taken for long periods of time. Silver has been shown to work on its own, but it also potentizes the effect of other anti-microbials. Like other homeopathics, Ag23 is taken straight and undiluted away from all other items, including food, water or other drinks. Dosage: 2 tablespoons 2-3 times per day

Most patients benefit from decreasing the aluminium burden of lungs and brain. Aluminum has been shown to enhance the destructiveness of Lyme: add **Polmolo** to the mix and titrate the optimal dose in the same manner (somewhere between 2-3 dropperful 2-3 times/day). This can be done independent of the presence of Lyme or other related infections. Use in addition BioSil, 10-15 drops/day, consider malic acid or magnesium malate and 1 lemon/day in 1 quart of water (citric acid)

Many Lyme patients have undiagnosed kidney problems since the inner lining of the kidney, ureter, bladder, and urethra are favourite hiding and feasting places of Lyme and Co. **Renolo tincture** (Source: www.KiScience.com): take separately from the cocktail in a glass of water and titrate to the effective dose. Full dose: 3 dropperful 3 times per day in a full glass of clean water. Can also be squirted on food (salad dressing, cooked veggies, etc.)

**Mold**: many Lyme patients suffer from mould illness as well. The home has to be mould free, as well as the body. In addition to Cistus use **Rizol Gamma** (ozonated plant oils). After the tolerated dosages of the cocktail are established, add Gamma and slowly increase to the optimal dose of 15 drops 3 times per day. These can be added to the cocktail or be taken independently. Rizol Kappa and Lambda often test for Bartonella (same dose).

*The dosages recommended are for a 70 kg person. Other suppliers can be used as long as the products are sourced carefully and meet the sensitive exclusion criteria. Herbs may have the same name but may not have the same amount of biological activity. During the initial active treatment phase antioxidant vitamins have been shown to be an obstacle rather than helpful. Most chronic Lyme patients start feeling better after 3-4 months on this protocol and reach a profound level of recovery after 18 months. Some patients who were severely ill for many years and have taken antibiotics for more than 3 years may need a safe, simple, inexpensive and well tolerated maintenance dose of some of these liposomal herbs for the rest of their life.*

**Biophysics**: at SHI we use acupuncture, neural therapy, microcurrent, sound and light therapy, pulsating magnetic fields, homeopathy, massage and ultrasound to enhance remedy uptake and recovery of the autonomic nervous system. But clearly neural therapy stands out in its effectiveness, simplicity and versatility.

**References**: S.Buhner’s books (i.e “Healing Lyme”, “Herbal Antibiotics”, etc.), + handout Lyme Bastyr University 2017. The below references with the kind permission from klinghardt Institute. We borrow much of the literature from my work with autistic children. Research on autism has been well funded in recent years – because of pressure from parents, not because of government willingness to change course. However, to date there has been no such funding for Lyme-related research, other than finding a marketable, patentable antibiotic that can be sold for high profits. We realized that the same environmental factors that contribute to autism are the ones also responsible for the severity of Lyme related symptoms. The successful treatment options for autism also work for the most severe cases of Lyme. For the truly biological treatment of Lyme we borrow insights gained painfully from the treatment experience gained in the Autism epidemic.

**Bee Venom Therapy:**

**Bee venom has been my personal favourite treatment that has kept me and many of my patients alive through many health challenges, including all stages of Lyme with many immunological, neurological and orthopaedic issues.**

Socarras, Kayla M., Sapi, E. et al. "*Antimicrobial Activity of Bee Venom and Melittin against Borrelia burgdorferi."* *Antibiotics* 6.4 (2017): 31. Abstract: Lyme disease is a tick-borne, multi-systemic disease, caused by the bacterium *Borrelia burgdorferi.* Though antibiotics are used as a primary treatment, relapse often occurs after the discontinuation of antimicrobial agents. The reason for relapse remains unknown, however previous studies suggest the possible presence of antibiotic resistant Borrelia round bodies, persisters and attached biofilm forms. Thus, there is an urgent need to find antimicrobial agents suitable to eliminate all known forms of *B. burgorferi*. In this study, natural antimicrobial agents such as *Apis mellifera* venom and a known component, melittin, were tested using SYBR Green I/PI, direct cell counting, biofilm assays combined with LIVE/DEAD and atomic force microscopy methods. The obtained results were compared to standalone and combinations of antibiotics such as Doxycycline, Cefoperazone, Daptomycin, which were recently found to be effective against Borrelia persisters. Our findings showed that both bee venom and melittin had significant effects on all the tested forms of *B. burgdorferi.* In contrast, the control antibiotics when used individually or even in combinations had limited effects on the attached biofilm form. These findings strongly suggest that whole bee venom or melittin could be effective antimicrobial agents for *B. burgdorferi;* however, further research is necessary to evaluate their effectiveness in vivo, as well as their safe and effective delivery method for their therapeutic use.

**Keywords:** Lyme disease; bee venom; melittin; biofilms; persisters; antibiotic resistance

***Bee Venom Therapy for Chronic Pain****: D Klinghardt, J. of Neurol and Orthop. Med and Surg., Vol. 11, Issue 9, Oct 1990, pp. 195-197*

Lubke, L.L., and Garon, C.F.: ***The Antimicrobial Agent Melittin Exhibits Powerful In Vitro Inhibitory Effects on the Lyme Disease Spirochete*.** Clinical Infectious Diseases, 1997;25 (Suppl 1): pp 48-51

***The streamlined Ki- approach to reduce the impact of Lyme on the system without causing severe collateral damage:***

1. ***Follow the retroviral treatment recommendation plus add Step 1 and Step 2 of this Lyme protocol. Rarely anything else is needed!***
2. ***If positive change, wait until the client flatlines again. If no change, include Step 3***
3. ***If the client needs a more aggressive approach, use bee venom therapy (according to the recommendations by M.Simics/Klinghardt). Depending on what country you live in and the legal restrictions, consider a one-time Medical trial with intravenous suramin (20 mg/kg body weight, dissolved in normal saline. Given over 20 minutes. Expect temporary mild skin rashes as side effect***

**Aluminium Toxicity**

Immunome Res 2013, 9:1http://dx.doi.org/10.4172/1745-7580.1000069  **Aluminum’s Role in CNS-immune System Interactions leading to Neurological Disorders**Shaw CA1,2,3\*, Kette SD4, Davidson RM5 and Seneff S6
Neural Dynamics Research Group, Department of Ophthalmology and Abstract

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

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](https://www.sciencedirect.com/topics/medicine-and-dentistry/autism-spectrum) (ASD) is a group of [neurodevelopmental conditions](https://www.sciencedirect.com/topics/medicine-and-dentistry/neurodevelopmental-disorder) of unknown cause. It is highly likely that both genetic [[1]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763) and environmental [[2]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763) factors are associated with the onset and progress of ASD while the mechanisms underlying its [aetiology](https://www.sciencedirect.com/topics/medicine-and-dentistry/etiology-medicine) are expected to be multifactorial [[3]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763), [[4]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763), [[5]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763), [[6]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763). Human exposure to aluminium has been implicated in ASD with conclusions being equivocal [[7]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763), [[8]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763), [[9]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763), [[10]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763). 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](https://www.sciencedirect.com/topics/medicine-and-dentistry/pediatrics) [vaccines](https://www.sciencedirect.com/topics/earth-and-planetary-sciences/vaccines) that include an aluminium [adjuvant](https://www.sciencedirect.com/topics/chemistry/adjuvant) are an indirect measure of infant exposure to aluminium and their burgeoning use has been directly correlated with increasing prevalence of ASD [[11]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763). Animal models of ASD continue to support a connection with aluminium and to aluminium adjuvants used in human [vaccinations](https://www.sciencedirect.com/topics/medicine-and-dentistry/vaccination) in particular [[12]](https://www.sciencedirect.com/science/article/pii/S0946672X17308763). 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](https://www.sciencedirect.com/topics/medicine-and-dentistry/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](https://www.sciencedirect.com/topics/medicine-and-dentistry/non-neuronal-cell). The presence of aluminium in [inflammatory cells](https://www.sciencedirect.com/topics/veterinary-science-and-veterinary-medicine/white-blood-cell) in the [meninges](https://www.sciencedirect.com/topics/veterinary-science-and-veterinary-medicine/meninges), vasculature, [grey and white matter](https://www.sciencedirect.com/topics/veterinary-science-and-veterinary-medicine/grey-matter) is a standout observation and could implicate aluminium in the [aetiology](https://www.sciencedirect.com/topics/medicine-and-dentistry/etiology-medicine) of ASD.

Diagnosis:

Because the aluminium toxicity is from nanoparticles and the total amount in the system, however illness producing, is small and non-detectable with current blood tests or hair analysis. Both apheresis (Dr.Straube/Germany) and the OligoScan will show the problem

Treatment/Detox protocol:

Polmolo tincture: 3 pipettes 3 times/day. Add MicroPhos as soon as there is space in the bottle. Ionic footbath twice weekly. BioSil: 10 drops twice daily in apple juice. Add a whole lemon for citric acid. Use magnesium malate at night for better sleep and additional anti aluminium effect. Prof. Exley recommends drinking Volvic water ( high silica), others Fiji water. Always use homeopathic alumina C30 while pushing out Al. Use chlorella, zeolite or ecklonia cava as binder.

**Glyphosate:**

Please google MIT researcher Stephanie Seneff PhD and her work to understand the depth and breadth of this catastrophic issue.

Diagnosis: urine test (available in US, Germany)

Treatment:

Eat organic only. Grow your own food. Over 90% of the British population has glyphosate in the urine. It was in the system before - causing severe damage.

Use “Matrix minerals” (humic/fulvic acid from peet concentrate, in US [www.biopure.com](http://www.biopure.com)). 1 dropperful with each meal. In autistic children: use “Restore”

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.

Use LavaVitae as binder.

Oil pulling: after moderate exercise use 1 tablsp of olive oil or sesame oil, swiche and chew for 15 minutes. Spit out and brush teeth. Oil extracts fat-soluble toxic cofactors of glyphosate toxicity from blood stream.

Use post-exercise Korsakoff homeopathic urine therapy as discussed in ART (urine contains macro amounts of glyphosate)

**Dental Issues**

**Root canal treated teeth harbour pathogens**

More than half the teeth studied with apical periodontitis had bacteria in tubules all the way to the cemental junction. Peters et al JOE 2001 27:76-81.

205 of 256 species isolated from human dentinal tubules were obligate anaerobes. Ando et al Int Endod J. 1990 23:20-7.

Four of ten specimens with apical periodontitis were heavily invaded by yeasts.

Sen et al Endod Dent Traumatol 1995 11:6-9.

Bacteria and fungi in six of nine root end biopsies with therapy resistant periapical lesions. *Nair et al JOE 1990, 16:580-8.*

All endodontic pathogens tested were able to penetrate into dentinal tubules, though different species penetrated to different extents. *Siqueira et al JOE 1996 22:308-10.*

Bacterial recognition of factors such as collagen type, and interspecies interaction facilitates colonization of dentinal tubules. *Love et al Crit Rev Oral Biol Med 2002 13:171-83.*

J.F. Siqueira, et. al., Polymerase chain reaction-based analysis of microorganisms associated with failed endodontic treatment, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004; 97: 85-94

JF Siquiera, et. al.; A Scanning Electron Microscopic Evaluation of In Vitro Dentinal Tubules Penetration by selected Anaerobic Bacteria, *Journal of Endodontics, June 1996, Vol. 22, No. 6*

*The results indicated that all bacterial strains tested were able to penetrate into dentinal tubules….persistent infection may be caused by microorganisms that have invaded dentinal tubules before or during endodontic treatment. Root canal walls were heavily infected with E. faecalis…..*

**Jaw bone infections breed retroviruses, secreting RANTES into the system with severe consequences**

International Journal of General Medicine 2013:6 277–290

RANTES and fibroblast growth factor 2 in jawbone cavitations: triggers for systemic disease? Johann Lechner, Volker von Baehr

Abstract: Regulated upon activation, normal T-cell expressed, and secreted (RANTES) and fibroblast growth factor (FGF)-2 were found at high levels in the JCs tested. Other cytokines could not be detected at excessive levels. Discussion: The study confirms that JC is able to produce inflammatory messengers, primarily RANTES, and, secondarily, FGF-2. Both are implicated in many serious illnesses. The excessive levels of RANTES/FGF-2 in JC patients with amyotrophic lateral sclerosis, multiple sclerosis, rheumatoid arthritis, and breast cancer are compared to levels published in medical journals. Levels detected in JCs are higher than in the serum and cerebrospinal fluid of amyotrophic lateral sclerosis and multiple sclerosis patients and four-fold higher than in breast cancer tissue.

Conclusion: This study suggests that JC might serve as a fundamental cause of IDs, through RANTES/FGF-2 production. Thus, JC and implicated immune messengers represent an integrative aspect of IDs and serve as a possible cause. Removing JCs may be a key to reversing IDs. There is a need to raise awareness about JC throughout medicine and dentistry.

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**Poor oral health activates Retroviruses**[Journal of the American Veterinary Medical Association](https://avmajournals.avma.org/loi/javma); October 15, 2014, Vol. 245, No. 8, Pages 916-922 <https://doi.org/10.2460/javma.245.8.916>

Abstract: Association between oral health status and retrovirus test results in cats. Mathew R. Kornya, DVM Susan E. Little, DVM Margie A. Scherk, DVM William C. Sears, MSc Dorothee Bienzle, DVM, PhD. Address correspondence to Dr. Bienzle (dbienzle@uoguelph.ca).

Objective—To determine associations between oral health status and seropositivity for FIV or FeLV in cats. Animals—5,179 cats. Procedures—Veterinarians at veterinary clinics and animal shelters completed online training on oral conditions in cats and then scored oral health status of cats with no known history of vaccination against FIV. Age, sex, and results of an ELISA for retroviruses were recorded. Results were analyzed by means of standard logistic regression with binary outcome.

Results—Of 5,179 cats, 237 (4.6%) and 186 (3.6%) were seropositive for FIV and FeLV, respectively, and of these, 12 (0.2%) were seropositive for FIV and FeLV. Of all 5,179 cats, 1,073 (20.7%) had gingivitis, 576 (11.1%) had periodontitis, 203 (3.9%) had stomatitis, and 252 (4.9%) had other oral conditions (overall oral disease prevalence, 2,104/5,179 [40.6%]). Across all age categories, inflammatory oral disease was associated with a significantly higher risk of a positive test result for FIV, compared with the seropositivity risk associated with other oral diseases or no oral disease. Stomatitis was most highly associated with risk of FIV seropositivity. Cats with any oral inflammatory disease were more likely than orally healthy cats to have a positive test result for FeLV. Increasing age was associated with a higher prevalence of oral disease in retrovirus-seronegative cats.

Conclusions and Clinical Relevance—**Inflammatory oral disease was associated with an increased risk of seropositivity for retroviruses** in naturally infected cats. Therefore, retroviral status of cats with oral inflammatory disease should be determined and appropriate management initiated.

**Mercury:** Metal Toxicity sets the stage for Lyme disease!

From the abstract: “These studies suggest that the detection of autoantibodies to NS (nervous system)-specific antigens may be used to monitor the development of neurotoxicity to environmental chemicals and that immune mechanisms may be involved in the progression of neuro-degeneration.” “Autoantibodies against neurofilament triplet proteins (NFs) as well as MBP and GFAP (glial fibrillary acidic protein) have been detected in sera and CSF of human subjects suffering from neurologic disorders, including Alzheimer’s disease, Parkinson dementia, ALS, Creutzfedt-Jakob disease, and kuru. Furthermore, autoantibodies to *neurotypic proteins have been demonstrated in animal models of allergic encephalomyelitis and electroconvulsive shock.*”

“Autoantibodies against NFs, MBP, GFAP, and GM1 ganglioside have been detected in sera and CSF of human subjects suffering from these diseases.  Anti-MBP antibodies are cytotoxic and are believed to play a key role in the pathogenesis of MS.  More recently the presence of autoantibodies to the dihydropyridine calcium channel (the L channel) has been demonstrated in sera of ALS and Lambert-Eaton syndrome patients.” “In the human studies, anti-NFs, IgG isotope, were the most frequently detected antibodies.  These were also the antibodies and the isotope best correlated with blood lead or urinary mercury and clinical scores of sensorimotor defects.”  “…the detection of GFAP titers in these studies supports **the targeting of the CNS and astrocytes by heavy metals**, since astrocytes are exclusively found in the CNS.”



*I consider the Beagle study below still the most convincing piece of work, demonstrating that* *correcting the occlusion is not an expensive hobby but a significant part of an integrative health* *protocol!*

J.Kyoto Pref. Univ. Med. 98(10). 1077-1085. October 1989.

**Systemic effects of the peripheral disturbance of the trigeminal system:** **Influences of the occlusal destruction in dogs.** Teruaki Sumioka. Department of Anesthesiology. Kyoto Prefectural University of Medicine

Abstract:

Although there is an increasing amount of information pertaining to intracranial pathways of the trigeminal nerve, its clinical significance still remains unclear in many ways. I assumed that dental disorders including malocclusion would lead to the disturbance of the central nervous system via the trigeminal nerve. Based on this belief, this study was conducted to find out systemic effects of the occlusal destruction by grinding teeth unilaterally in dogs. As the result: abnormal involuntary movement and symptoms of autonomic failure were observed. These experimental results indicate that the trigeminal nuclear complex contains not only the functions of the sensory relay in the face and the control of chewing movement, but it is likely that it modulates motor, especially postural control and autonomic system. It is believed that the dental aspects, especially occlusion, play an important role for the proper functioning of the trigeminal system.

Treatment:

* Correct the occlusion
* Remove all metals and dissimilar materials by “biological dentist”, certified by IAOMT (International Acad. of Oral Medicine and Toxicology)
* Cavitation surgery with specialized and recommended dentist

Electromagnetic Radiation

**“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.

Treatment

1. **Daytime:**
* No wireless in home, no chordless phones
* Child should wear radio protective clothing
* **Rosemary tincture**: “*highly significant protective anti-mutagenic activity”. “Even the most powerful water-soluble antioxidants lack the capacity to protect against gamma ray induced damage”. (***British Journal of Radiology***,* February 2 edition, 2015) (source: KiScience.com)
* Using the **Stetzer filters** throughout home or school to decrease “dirty electricity”

**2. Evening:**

* Use red filter on the computer screen after 4 pm
* Avoid computer work/cell phone after sunset. Read a book!
* Liposomal Melatonin(+ 50-100 mg DMSA for a few weeks)
* Trial with 5 HTP (adult dose: 200 mg)
* **Propolis tincture** 4-6 pipettes after dinner (a valid alternative to propolis). A propoliscompound (CAPE) also protects lymphocytes against radiation (2008 Journal of Biochemical and Molecular Toxicology). Best: use KiScience **“RayWave”** – 2 pipettes 3 times/day. At dinner time: KiScience **Sleep tincture**
* TD-Magnesium, **Epsom salt baths** twice daily, oral Mag.glycinate. Magnesium acts as calcium channel blocker. Voltage gate calcium channels are upregulated by EMR (M.Pall, 2013)

3. **Nights:**

* Sleep sanctuary, fuses off
* Consider Samina bed. Elevate head-end of bed 10 cm