

Impact of Nutrition, Infection and Environmental Toxins on Learning Disabilities: Evaluation and Treatment.

Star Academy
September 17th, 2010

Julie A. Griffith, M.D., M.S., C.M.T.
Pediatric and Adult Neurologist/Behavioral Neurologist
1099 D. St., Suite 208
San Rafael, CA 94901
415 925-1616

www.mybrainhealth.org, mybrainhealth@comcast.net

Outline

- 1. Learning disorders, define
- 2. Learning disorders, list
- 3. Learning disorders, underlying medical causes
- 4. Treatable underlying medical problems
 - a. testing
 - b. treatment
- 5. Resources

Learning disabilities

- According to the regulations for Public Law (P.L.) 101-476 which is entitled The Individuals with Disabilities Education Act (IDEA), the definition of *Learning Disability* is “a disorder in one or more of the basic psychological processes involved in understanding or in using spoken or written language, which may manifest itself in an imperfect ability to listen, think, speak, read, write, spell or to do mathematical calculations.”

Learning disabilities

- Alexia
- Attention Deficit Disorder (ADD)
- Attention Deficit Hyperactivity Deficit (ADHD)
- Dyslexia
- Math disorder
- Memory difficulties
- Nonverbal learning disorder
- Reading Comprehension Disorder
- Sensory Processing Disorders

Learning difficulties can be secondary to any of the following:

- Inattention
 - Low Energy level
 - Psychological stressors
 - Seizures (coordination difficulties, inattention, memory difficulties, weakness)
 - Sleep disorder (inattention, fatigue)
- Sleep. 2010 Mar 1;33(3):319-25.

- [Gozal D](#), [Serpero LD](#), [Kheirandish-Gozal I](#), [Capdevila OS](#), [Khalyfa A](#), [Tauman R](#), Pritzker School of Medicine, University of Chicago
- TNF-alpha levels are increased in pediatric obstructive sleep apnea, and the TNF-alpha levels are primarily driven by sleep fragmentation and increased body mass index, and are closely associated with the degree of sleepiness, as measured by Multiple Sleep Latency Test.

Learning disabilities, some common underlying medical problems

Pediatrics. 2009 Aug;124(2):837-44.

- ADD/ADHD
- Autism spectrum disorders
- Dyslexia
- Math disorder
- Nonverbal learning disorder
- Sensory processing disorders
- Speech and language disorders
- Writing disorders

ADD / ADHD

- Allergies
- hearing loss
- heavy metal poisoning (Sci Total Environ. 2010 Sep 6), lead (level 5 caused ADHD and cognitive deficits in Korean children)
- hormonal deficiencies (hypocholesterolemia, hypothyroidism)
- nutrititional deficiencies (aa, B vitamins, lithium, omega 3)
- infections
- pain
- seizures J Learn Disabil. 2008 May-Jun;41(3):195-207
- sleep disorders (obstructive; seizures, fungal infx, tick born d)

Snoring-induced ADHD

Sleep, 20(12):1185–1192

© 1997 American Sleep Disorders Association and Sleep Research Society

Pediatrics and Sleep

Symptoms of Sleep Disorders, Inattention, and Hyperactivity in Children

*Ronald D. Chervin, †James E. Dillon, *Claudio Bassetti, *Dara A. Ganoczy
and ‡Kenneth J. Pituch

*Departments of *Neurology, †Psychiatry, and ‡Pediatrics and Communicable Diseases,
University of Michigan Medical Center, Ann Arbor, Michigan, U.S.A.*

Summary: Children with sleep disorders are often inattentive or hyperactive, and some carry a diagnosis of attention deficit/hyperactivity disorder (ADHD) until their sleep disorder is detected. However, the potential behavioral impact of undiagnosed sleep disorders is not known. We sought to determine whether children with higher levels of inattention and hyperactivity more frequently have symptoms of sleep-related breathing disorders (SRBDs) or periodic limb movement disorder (PLMD). We surveyed parents of 2–18-year-old patients at a child psychiatry clinic ($n = 70$) and a general pediatrics clinic ($n = 73$) to assess the children's behavior, snoring, complaints of restless legs at night, and daytime sleepiness. A validated pediatric sleep questionnaire provided the explanatory variables, and a scale for inattention and hyperactivity, derived from the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV), provided the dependent variable. Habitual snoring was more frequent (33%) among children who carried a diagnosis of ADHD than among the other children at the psychiatry or general pediatric clinics (11 and 9%, respectively, chi-square test, $p = 0.01$). Snoring scores, derived from six snoring- and SRBD-related question items, were associated with higher levels of inattention and hyperactivity. The complaint of restless legs and a composite score for daytime sleepiness showed some evidence, though less consistent, of an association with inattention and hyperactivity. The association of snoring with inattention and hyperactivity suggests that SRBDs and perhaps other sleep disorders could be a cause of inattention and hyperactivity in some children. If a causal effect is present, our data suggest that 81% of habitually snoring children who have ADHD—25% of all children with ADHD—could have their ADHD eliminated if their habitual snoring and any associated SRBD were effectively treated. **Key Words:** Snoring—Sleep—Hyperactivity—Attention deficit/hyperactivity disorder—

Snoring children

- 70 children 2-18 yrs of age
snoring occurred in 33% kids with ADHD
compared to 10% of normal kids
- 81% children with snoring and ADHD could
have their ADHD eliminated if cause of snoring
is addressed, this would resolve 25% kids with
ADHD

Autisms

Term coined by Dr. Martha Herbert, MGH

- Genetic and Environmental Factors
- Genetic weakness in the detoxification system, the immune system, or both

5% genetic/metabolic syndrome, such as a mitochondrial disorder, or neurocutaneous disorders (Neurofibromatosis, Tuberous sclerosis), Acta Neurobiol Exp 2010, 70: 141–146, The review of most frequently occurring medical disorders

- related to aetiology of autism and the methods of treatment
- Magdalena Cubała-Kucharska^{1,2,3}

- Environmental Factors

- _ Allergies

- _ Chronic infection, often polyinfected

- _ EMF stress (electromagnetic field stress)

- _ Nutritional deficiencies

- _ Toxins

-

Autisms, environmental factors

- Environmental Factors

- _ allergies--food and environmental (mold, dust, cat and dog dander, plants)

- _ Chronic infection

- bacterial

- clostridia

- Streptococcal

- Tick born disease (Bartonella, Borrelia, Erlichia)

- fungus infections (Candida, Rhodotorula, others)

- parasitic infections (Babesia of tick born disease, blastocystis hominis, other)

- viral infections (HHV6, EBV, HSV Types 1 and 2 and others)

- **Toxins**

- Heavy metal poisoning, most common toxin, most recognized (80% children improve with removal of heavy metals, if elevated (i.e. Aluminum, Arsenic, cadmium, lead, mercury)

- Dis Model Mech. 2010 May-Jun;3(5-6):366-76. **Neuroligin-deficient mutants of *C. elegans* (nematode) have sensory processing deficits and are hypersensitive to oxidative stress and mercury toxicity.**, Our results suggest a possible link between genetic defects in synapse formation or function, and sensitivity to environmental factors in the development of autism spectrum disorders)

- Journal of Pediatric Health Care (20)5: P. 350-352, 2006. J.C. Dale.

- Chlorine (stillbirth, congenital cardiac defects, neural tube defects)

- PCB (polychlorinated biphenyl hydrocarbons) gas stations, landfill

Dyslexia

- Genetic
- Chromosome 6 (early gray hair, autoimmune, L-handed)
- FOXP2 truncation as a novel cause of developmental speech and language deficits, Am J Hum Genet. 2005 Jun;76(6):1074-80.

[MacDermot KD](#), [Bonora E](#), [Sykes N](#), [Coupe AM](#), [Lai CS](#), [Vernes SC](#), [Vargha-Khadem F](#), [McKenzie F](#), [Smith RL](#), [Monaco AP](#), [Fisher SE](#).

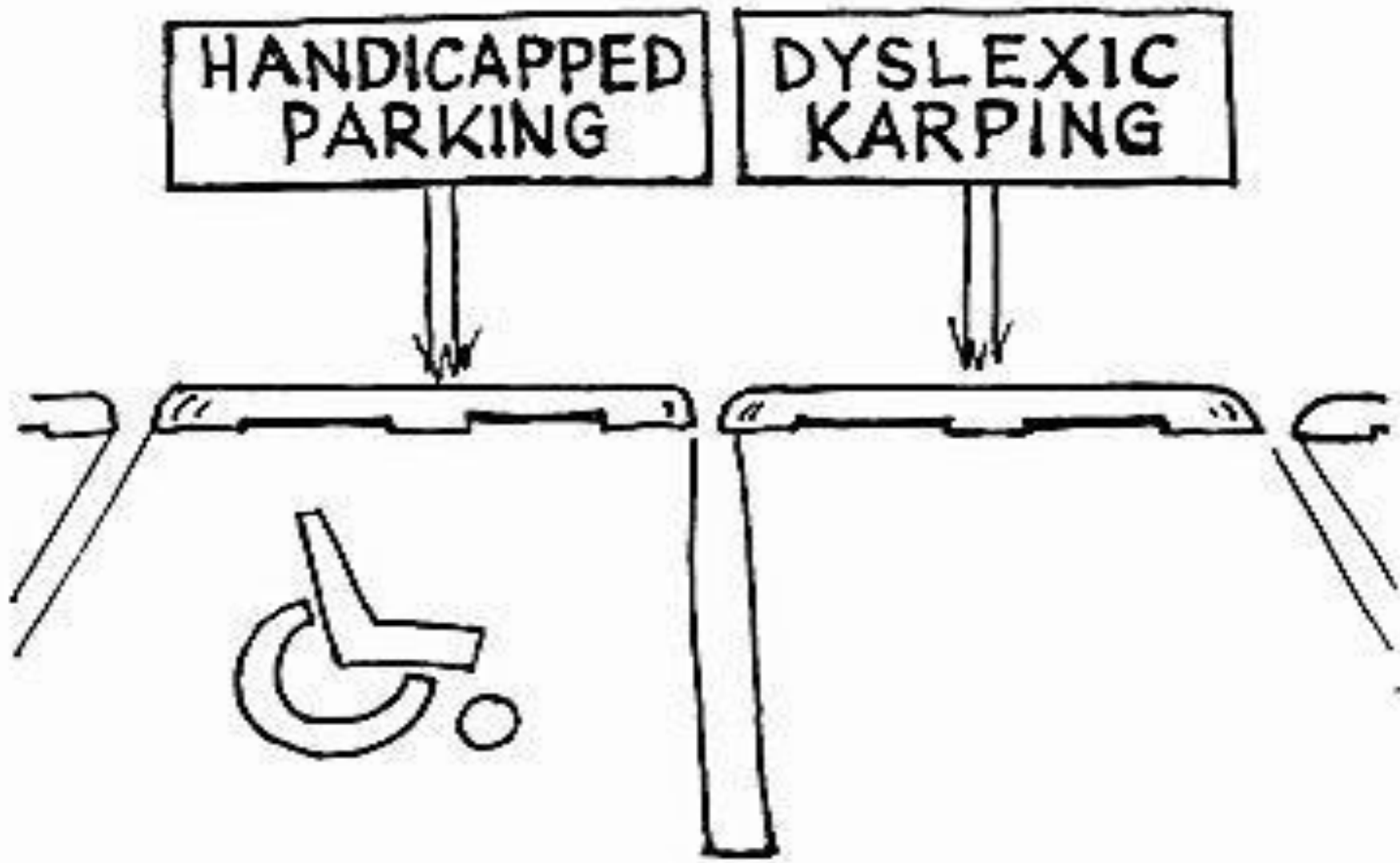
FOXP2, the first gene to have been implicated in a developmental communication disorder, offers a unique entry point into neuromolecular mechanisms influencing human speech and language acquisition. In multiple members of the well-studied family, a heterozygous missense mutation in FOXP2 causes problems in sequencing muscle movements required for articulating speech (developmental verbal dyspraxia), accompanied by wider deficits in linguistic and grammatical processing.

Alexia (cannot read)- acquired dyslexia

— stroke (Neurorehabil Neural Repair. 2010
Sep 9)

-Tick born disease

<http://www.townsendletter.com/July2010/savechildren0710.html>



SEARCH ID: JRA0210

© Original Artist
Reproduction rights obtainable from

Math disorder

- Genetic

Neurofibromatosis 1- Dev Disabil Res Rev.
2009;15(1):45-51.

Potential influences on mathematical difficulties in
children and adolescents with neurofibromatosis, type 1.,
[Moore BD.](#)

-polygenetic and polyenvironmental

-seizures (J Learn Disabil. 2008 May-Jun;41(3):195-207.)

Memory difficulties

- Hydrocephalus
- Seizures
- Sleep disorder
- Tick born Disease
- Viral infection (Herpes simplex virus)

Hydrocephalus and learning disabilities

- Neuropsychology. 1998 Oct;12(4):578-89.
- **Memory functions in children with early hydrocephalus.**
- [Scott MA](#), [Fletcher JM](#), [Brookshire BL](#), [Davidson KC](#), [Landry SH](#), [Bohan TC](#), [Kramer LA](#), [Brandt ME](#), [Francis DJ](#).
- Department of Pediatrics, University of Arkansas for Medical Sciences, Springdale 72765-0768, USA. mscott3@aol.com
- **Abstract**
- Children with arrested, shunted, and no hydrocephalus were compared on verbal and nonverbal memory tasks assessing multiple components of memory. A gradient of severity was hypothesized, with the shunted hydrocephalus group expected to exhibit the most significant memory impairments and the arrested group expected to perform more poorly than children with no hydrocephalus. Etiologies of prematurity, spina bifida, and aqueductal stenosis were represented by 157 participants. Results supported the hypothesis; the shunted hydrocephalus group performed poorer on all memory measures. Differences for the arrested group were less frequently statistically significant relative to children with no hydrocephalus. Irrespective of etiology, the shunted hydrocephalus group exhibited a pattern of performance suggestive of encoding and retrieval deficits on both verbal and nonverbal tasks, showing a pervasive disturbance of memory processes.

Nonverbal learning disorder needs head MRI and EEG

- Heavy metal poisoning
- Hydrocephalus
- Neurofibromatosis Curr Neurol Neurosci Rep. 2003 Mar;3(2):129-36. Review.
- Seizures in 15%
- Tick born disease

Sensory processing disorders

- Heavy metal poisoning (i.e. mercury)
- Skin-brain condition-neurocutaneous disorder, café au lait spots, large head circumference, Lisch nodules of eyes, math difficulties, visual spatial difficulties ADHD
Dev Disabil Res Rev. 2009;15(1):45-51.

Potential influences on mathematical difficulties in children and adolescents with neurofibromatosis, type 1.

- Tick born disease (sensory hyperarousal, Columbia website; Dr. Leventhal and O'Shea's neuropsych lecture, LIA 2009)

Speech and language disorders

- Hearing loss
- Heavy metal poisoning
- Hypothyroidism
- Stroke-perinatal injury (dysphagia) Clin Pediatr (Phila).
2009 Apr;48(3):247-51
- Tick born disease
- Traumatic brain injury

Writing disorders

- heavy metals poisoning,
-seizures J Learn Disabil. 2008 May-
Jun;41(3):195-207
- tick born disease

Genetics

- More difficult to change. Can do gene therapy, but this is for a handful of disorders. For most disorders, there are clinical trials, but not definitive proven benefits

Epigenetics

- Up to 30,000 different factors which may influence a gene (i.e. nutrition, radiation, toxins, herbs, medications, exercise, electromagnetic forces, etc) Dr. Bruce Lipton, previously from Stanford

3 common learning disabilities are
multifactorial—
polygenetic and polyenvironmental

J Dev Behav Pediatr. 2010 Sep;31(7):533-44

- ADD/ADHD
- Math disorder
- Reading disorder



Treatable common underlying medical problems (epigenetic factors):

Allergens

Infections

Nutritional deficiencies

Toxins

Allergies



Clearing allergies

- Improves attention
- Lessens cognitive fog (per my practice, not in the literature)
- Lessens fatigue
- Lessens snoring/hypoxemia at night
- Improves memory during the day (if snoring)
- Lessens anxiety/panic disorder
- Lessens conductive hearing loss

Allergies can cause anxiety

- Australian and New Zealand Journal of Psychiatry
 -
 - Association between allergy and anxiety disorders in youth
 - 2001, Vol. 35, No. 6 , Pages 815-821
 -
 - [Pavel A Kovalenko1, Christina W Hoven2, Ping Wu2, Judith Wicks2, Donald J Mandell2 and Quyen Tiet2](#)
 - 1Virginia Institute for Psychiatric and Behavioural Genetics, Virginia Commonwealth University, 800 East Leigh Street, PO Box 980126, Richmond, VA, 23298–0126, USA kovalenp@yahoo.com
 - 2Department of Child Psychiatry, Columbia University and New York State Psychiatric Institute, New YorkUSA
 -
- Conclusions:** Findings suggest that in some patients panic disorder may be associated with hypersensitivity of immune system. Panic disorder should be considered in anxious children reporting allergy when no organic cause of allergy is found, and likewise allergy should be considered in children with panic disorder.

Allergies can cause brain fog

- Cerebral hypoperfusion can cause cognitive difficulties: brain fog (histamine release causes low blood pressure/leaky vessels, theoretically causing lack of brain perfusion/leading to suboptimal brain functioning)

Allergies, congestion, and sleep

- Curr Allergy Asthma Rep. 2010 Mar;10(2):113-21.
- **Congestion and sleep impairment in allergic rhinitis.**
- [Craig TJ](#), [Sherkat A](#), [Safaei S](#).
- Hershey Medical Center, Penn State University, 500 University Drive, Hershey, PA, 17033-0850, USA. tcraig@psu.edu
- **Abstract**
- Allergic rhinitis is a prevalent disease in developed nations, and its prevalence has been increasing throughout the world. Nasal congestion is the most common and bothersome symptoms of rhinitis. Congestion is associated with sleep-disordered breathing and is thought to be a key cause of sleep impairment in individuals with rhinitis. The end result is a decrease in quality of life and productivity and an increase in daytime sleepiness. Treatment with intranasal corticosteroids has been shown to reduce nasal congestion. Data on sleep-related end points from clinical trials of intranasal corticosteroids indicate that this reduction is associated with improved sleep, reduced daytime fatigue, and improved quality of life. Other therapies, such as montelukast, also have a positive influence on congestion and sleep. This review examines nasal congestion and the associated sleep impairment of allergic rhinitis patients. It explores the adverse effects of disturbed sleep on quality of life and how these conditions can be reduced by therapies that decrease congestion.

Bacteria and viruses in gut may contribute to development of allergies

- Adv Drug Deliv Rev. 2004 Apr 19;56(6):795-807.
- **Intestinal epithelial tight junctions as targets for enteric bacteria-derived toxins.**
- [Fasano A, Nataro JP.](#), Division of Pediatric Gastroenterology and Nutrition, School of Medicine, University of Maryland, Baltimore 21201, USA. afasano@umaryland.edu
- Ann N Y Acad Sci. 2000;915:214-22.
- **Regulation of intercellular tight junctions by zonula occludens toxin and its eukaryotic analogue zonulin.**
- [Fasano A.](#)
- Division of Pediatric Gastroenterology and Nutrition, Gastrointestinal Pathophysiology Section, Center for Vaccine Development, Department of Physiology, University of Maryland School of Medicine, Baltimore, MD 21201, USA. afasano@umaryland.edu
- **Abstract**
- Alteration of epithelial tj is a recently described property for infectious agents. Clostridium difficile toxin A and B and influenza and vesicular stomatitis viruses have been shown to loosen tight junctions in tissue culture monolayers. These changes appear to be irreversible and are associated with destruction of the tj complex. We were able to identify an intestinal Zot analogue, which we named zonulin. It is conceivable that the zonulins participate in the physiological regulation of intercellular tj not only in the small intestine, but also throughout a wide range of extraintestinal epithelia as well as the ubiquitous vascular endothelium, including the blood-brain barrier. Dysregulation ...may contribute to disease states that involve ... developmental and intestinal disorders, and tissue inflammation...
- **Comment::** when you affect the blood brain barrier, you increase the risk of neuroimmune disease and also increase the risk to chemical sensitivity.

Mold severely exacerbates allergies

- Allergy. 2003 Apr; 58(4):363-5.
- Does systemic exposure to aflatoxin B(1) cause allergic sensitization?
- Kocabaş CN, Sekerel BE.
- Department of Pediatric Allergy and Astma, Faculty of Medicine, Hacettepe University, Sıhhiye, Ankara, Turkey.

Allergy testing

- At least 2 immune reactions
 - 1) IgE mediated (immediate hypersensitivity)
 - 2) IgG Type 4 (delayed hypersensitivity)
 - 3) Sources
 - a) Environmental (cat, dog, dust, mold, plant)
 - b) Food
 - 4) Testing: Blood, consider Meridian Valley serum measures IgG(Type 4) and IgE through ELISA.
Also, other labs include Alcat or IBT (both can measure individual antibody reactions.)

Treatment of allergies

- For significant antibody response, remove foods for 4 months
- For moderate antibody response, rotate foods (once every 4 days)
- Caveat: casein and gluten sensitivity may need to go on to further testing for difficulties in digestion, if no immune reaction (urine peptide testing)
- Antiinflammatory diet

Allergies-inhalant

- Blood IgE and IgG Type 4

Cat, dog, dust, mold, weeds, trees and grasses

Antiinflammatory diet

- Increase PGE3: Good foods: omega 3 (fish oils, seaweed), 6 (nuts, avocado), 9 (cooking with olive oil)

fish oils: 80 mg/kg (mercury and PCB free), frig

Nordic naturals, Carlson possibilities of brands

Wild fish, free range meat and poultry

- Avoid (decrease PGE2): dairy, grain fed/corn fed meats and poultry

Infections



Infections-chronic and/or recurrent

- Bacterial
 - Lyme disease (Borrelia related complex, BRC, Helicobacter pylori) clostridia
 - PANDAS (Strep) -Intermittent flares of anxiety and/or tics
 - Fungal (Candida, Rhodotorula, Aspergillus)
 - Parasitic (blastocystis hominis, Giardia, others)
 - Viral- EBV, HHV6, HSV, measles, Rubella
- Less likely, but possible: CMV, human gamma retrovirus (co-infection of Tick born disease)

Infections, testing

- Blood

- _ IgE and IgG can assist identifying mold pathogens

- Tick born disease – may begin to use CD57, but T cells may not fully mature until 18 yrs

- Igenex western blot for Borrelia, titers for Babesia, Bartonella, and Erlichia

- Neuroscience testing interleukins, may be pd for by insurance

- Viral titers - HHV6, EBV full panel, HSV Type 1, urine neopterin (screen for virus, and identifies inflammation)

Stool testing

(possible labs Genova, Great Plains, Doctor's Data, DiagnosTechs)

- Cultures
Bacteria - Level of healthy bacteria lactobacillus acidophilus and bifidobacter
-pathogenic bacteria
- fungi

Big advantage: better able to identify and grow fungi than other local hospital labs or local national labs, cultures specific organism (if bacteria or fungus), identifies which medications or herbs actually effective in clearing organism

- parasites
- Inflammation (calprotectin)
- Insufficient digestion (pancreatic elastase 1)
- Bile acids
- pH

Lyme-induced autism

- Med Hypotheses. 2008;70(5):967-74. Epub 2007 Nov 5.
- **The association between tick-borne infections, Lyme borreliosis and autism spectrum disorders.**
- [Bransfield RC](#), [Wulfman JS](#), [Harvey WT](#), [Usman AI](#).
- Department of Psychiatry, Riverview Medical Center, 225 State Route 35, Red Bank, NJ, United States. bransfield@comcast.net
- **Abstract**
- Chronic infectious diseases, including tick-borne infections such as *Borrelia burgdorferi* may have direct effects, promote other infections and create a weakened, sensitized and immunologically vulnerable state during fetal development and infancy leading to increased vulnerability for developing autism spectrum disorders. A dysfunctional synergism with other predisposing and contributing factors may contribute to autism spectrum disorders by provoking innate and adaptive immune reactions to cause and perpetuate effects in susceptible individuals that result in **inflammation**, molecular mimicry, kynurenine pathway changes, increased quinolinic acid and decreased serotonin, **oxidative stress**, **mitochondrial dysfunction** and **excitotoxicity** that impair the development of the amygdala and other neural structures and neural **networks** resulting in a partial Klüver-Bucy Syndrome and other deficits resulting in autism spectrum disorders and/or exacerbating autism spectrum disorders from other causes throughout life. Support for this hypothesis includes **multiple cases of mothers with Lyme disease and children with autism spectrum disorders**; fetal neurological abnormalities associated with tick-borne diseases; **similarities between tick-borne diseases and autism spectrum disorder regarding symptoms, pathophysiology, immune reactivity, temporal lobe pathology, and brain imaging data**; positive reactivity in several studies with autistic spectrum disorder patients for *Borrelia burgdorferi* (22%, 26% and 20-30%) and 58% for *mycoplasma*; similar geographic distribution and improvement in autistic symptoms from antibiotic treatment. It is imperative to research these and all possible causes of autism spectrum disorders in order to prevent every preventable case and treat every treatable case until this disease has been eliminated from humanity.

Bacteria endotoxin association with autism

- Neurosci Lett. 2010 Mar 8;471(3):162-5. Epub 2010 Jan 25.
- **Low-grade endotoxemia in patients with severe autism.**
- [Emanuele E](#), [Orsi P](#), [Boso M](#), [Broglia D](#), [Brondino N](#), [Barale F](#), [di Nemi SU](#), [Politi P](#).
- Department of Health Sciences, Section of Psychiatry, University of Pavia, Via Bassi, 21, I-27100, Pavia, Italy. enzo.em@libero.it <enzo.em@libero.it>
- **Abstract**
- The objective of this study was to examine whether levels of endotoxin and other markers of immuno-inflammatory activation are altered in adult patients with severe autism. We determined circulating serum endotoxin levels, its soluble receptor (sCD14), and markers of immuno-inflammatory activation (IL-1beta, IL-6, and IL-10) in 22 adult patients with severe autism and 28 age- and gender-matched healthy controls. Compared with healthy subjects, serum levels of endotoxin were significantly higher in autistic patients and inversely and independently correlated with Socialization scores on the Vineland Adaptive Behavior Scales (VABS) and ADI-R Domain A score (social). Whether increased endotoxin may contribute to the pathophysiology of inflammation and impaired reciprocal social interaction in autism should be further explored in future studies.

Proinflammation in autism

- Brain Behav Immun. 2010 Jan;24(1):64-71. Epub 2009 Aug 8.
- **Differential monocyte responses to TLR ligands in children with autism spectrum disorders.**
- [Enstrom AM](#), [Onore CE](#), [Van de Water JA](#), [Ashwood P](#).
- Departments of Medical Microbiology and Immunology, University of California at Davis, CA 95817, USA.
- **Abstract**
- Autism spectrum disorders (ASD) are characterized by impairment in social interactions, communication deficits, and restricted repetitive interests and behaviors. Recent evidence has suggested that impairments of innate immunity may play an important role in ASD. To test this hypothesis, we isolated peripheral blood monocytes from 17 children with ASD and 16 age-matched typically developing (TD) controls and stimulated these cell cultures in vitro with distinct toll-like receptors (TLR) ligands: TLR 2 (lipoteichoic acid; LTA), TLR 3 (poly I:C), TLR 4 (lipopolysaccharide; LPS), TLR 5 (flagellin), and TLR 9 (CpG-B). Supernatants were harvested from the cell cultures and pro-inflammatory cytokine responses for IL-1beta, IL-6, IL-8, TNFalpha, MCP-1, and GM-CSF were determined by multiplex Luminex analysis. After in vitro challenge with TLR ligands, differential cytokine responses were observed in monocyte cultures from children with ASD compared with TD control children. In particular, there was a marked increase in pro-inflammatory IL-1beta, IL-6, and TNFalpha responses following TLR 2, and IL-1beta response following TLR 4 stimulation in monocyte cultures from children with ASD ($p < 0.04$). Conversely, following TLR 9 stimulation there was a decrease in IL-1beta, IL-6, GM-CSF, and TNFalpha responses in monocyte cell cultures from children with ASD compared with controls ($p < 0.05$). These data indicate that, monocyte cultures from children with ASD are more responsive to signaling via select TLRs. As monocytes are key regulators of the immune response, dysfunction in the response of these cells could result in long-term immune alterations in children with ASD that may lead to the development of adverse neuroimmune interactions and could play a role in the pathophysiology observed in ASD

Fungal infection- treatment improves:

- Attention
- diarrhea or constipation
- lessens urinary and stool incontinence
- clears cognition
- lessens night time sweating and insomnia

Fungal infections

- Autism-antifungals is proposed to relieve both gastrointestinal symptoms and helps cognitive functions as well (Sandler et al.,2000)

Fungal infection-examination and testing

- Thrush
- Ringworm
- Athlete's foot, nail infection, groin infection
- Stool culture and antifungal sensitivities (both to medications and herbs)
- Blood IgE and IgG screening against mold antigens

Dust mite endotoxins cause inflammation

- Int Arch Allergy Immunol. 2010;152(3):279-87. Epub 2010 Feb 12.
- **Indoor determinants of endotoxin and dust mite exposures in Hong Kong homes with asthmatic children.**
- [Leung TF](#), [Wong YS](#), [Chan IH](#), [Yung E](#), [Wong CK](#), [Lam CW](#), [Wong GW](#).
- Department of Pediatrics, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong, SAR, China.
- **Abstract**
- **BACKGROUND:** Domestic endotoxin enhances airway inflammation and increases asthma severity in Caucasian children, but little data are published on indoor endotoxin exposure in Asian countries. This study investigated house dust endotoxin and Der p 1 levels in Hong Kong families with asthmatic children, and their effects on asthma severity.
- **METHODS:** 115 asthmatics from a pediatric clinic underwent fractional exhaled nitric oxide (FeNO) and spirometric measurements. Home visits were then made within 2 weeks, during which parents completed the International Study of Asthma and Allergies in Childhood questionnaire. Settled dust was collected from patients' mattresses, bedroom floors and living room floors. Endotoxin and Der p 1 were measured by limulus ameocyte lysate and immunoassay, respectively.
- **RESULTS:** Endotoxin was detectable in all locations from all families, whereas Der p 1 was detectable in 58-70% of indoor sites. Floors of both bedroom and living rooms had higher endotoxin but lower Der p 1 levels than mattresses ($p < 0.001$ for both). Mattress endotoxin level correlated inversely with Der p 1 level ($r = -0.308$, $p = 0.001$). Household smoker, feather bedding and vacuum cleaning were independent determinants of indoor endotoxin. Timing of last bedding change was associated with Der p 1 levels at all sites. Mattress endotoxin level was associated with frequency of wheezing episodes ($p = 0.044$), but neither endotoxin nor Der p 1 was associated with FeNO and spirometric parameters.
- **CONCLUSIONS:** Domestic endotoxin levels are associated with frequency of wheezing episodes in asthmatic children but not their FeNO or spirometric measurements.

Nutritional deficiencies



Nutrients-purposes

- Antiinflammatory to brain
- Attention
- Energy
- Infection, prevent and resolve
- Neurotransmitter production
- Prevent depression/mood instability
- Prevent seizures

Nutritional deficiencies

- Amino acids (neurot, enzym, thyroid h, proteins, mus)
- Essential fatty acids (omega 3, dha)- attn, mood
- Vitamin A (vision, stop infections, stop acne)
- Vitamin B1 (stop nervous breakdown)
- Vitamins B2,3,5
- Vitamin B6 (prot, neurotrans, energy, sleep, dreams)
- Vitamin B12 (methyl dopamine, anemia, detox, lowering homocysteine)
- Folate-fully methylated form, helps to detoxify, attn

Nutritional deficiencies-testing

- Fasting blood and urine

Either local lab, or specialized lab (i.e. Metametrix, Genova, others) for functional needs

Generates amino acid and mvi/mmi recipes

- Hair-lithium deficiency

Nutritional deficiencies-treatment

- Best, excellent diet, but not enough, JAMA 1999 article, need to supplement with mvi/mmi, likely fish oils.
- Options, try not to include sugar in products
 - amino acid powder
 - mvi/mmi powder
 - specific supplements, mvi/mmi
 - Fish oils (liquid, chewable capsules, capsules)
 - lithium orotate pills

Nutritional deficiencies cont'd

- Methylcobalamine (+/- folic acid, +/- n-acetylcysteine) subcutaneous shots
- IV glutathione, vitamin C, Phospholipid
- Rarely, IV nutrition for severe Tick born disease, may also give IV antibiotics

Nutritional deficiencies-retest

- Retest blood (and possibly, urine, hair as needed) in approx 1-3 months to ensure nutrient levels are improving.

Toxins



Toxins

- Neurotox Res. 2007 Apr;11(3-4):203-18.
- **Glutamate antagonists are neurotoxins for the developing brain.**
- [Kaindl AM, Ikonomidou C.](#)
- Department of Pediatric Neurology, Charité, University Medical School, Campus Virchow-Klinikum, Augustenburger Platz 1, 13353 Berlin, Germany. angela.kaindl@charite.de
- **Abstract**
- Neurotransmitters and neuromodulators are essential for normal nervous system development. Disturbances in the expression timetable or intensity of neurotransmitter signalling during critical periods of brain development can lead to permanent damage. Neuroactive drugs and **environmental toxins** interfere with neurotransmitter signalling and may thereby provide one mechanism underlying neurological abnormalities. Glutamate is the main excitatory neurotransmitter in the mammalian central nervous system and mediates neurotransmission across **most excitatory synapses**. In this article we review the timely expression of the excitatory neurotransmitter glutamate and its receptors during brain development, briefly review glutamate receptor antagonists and present clinical and experimental evidence describing their adverse effects in the developing brain.

Toxins

- Biotoxins (bacteria, fungi) (common)
- EMF stress (common)
- Heavy metal poisoning (very common, most heavily studied and documented)
- PCB (polychlorinated biphenyl HC's) poisoning
- Screening from history of exposure, pesticides are common consider Metamatrix, US Biotek

Heavy metal poisoning

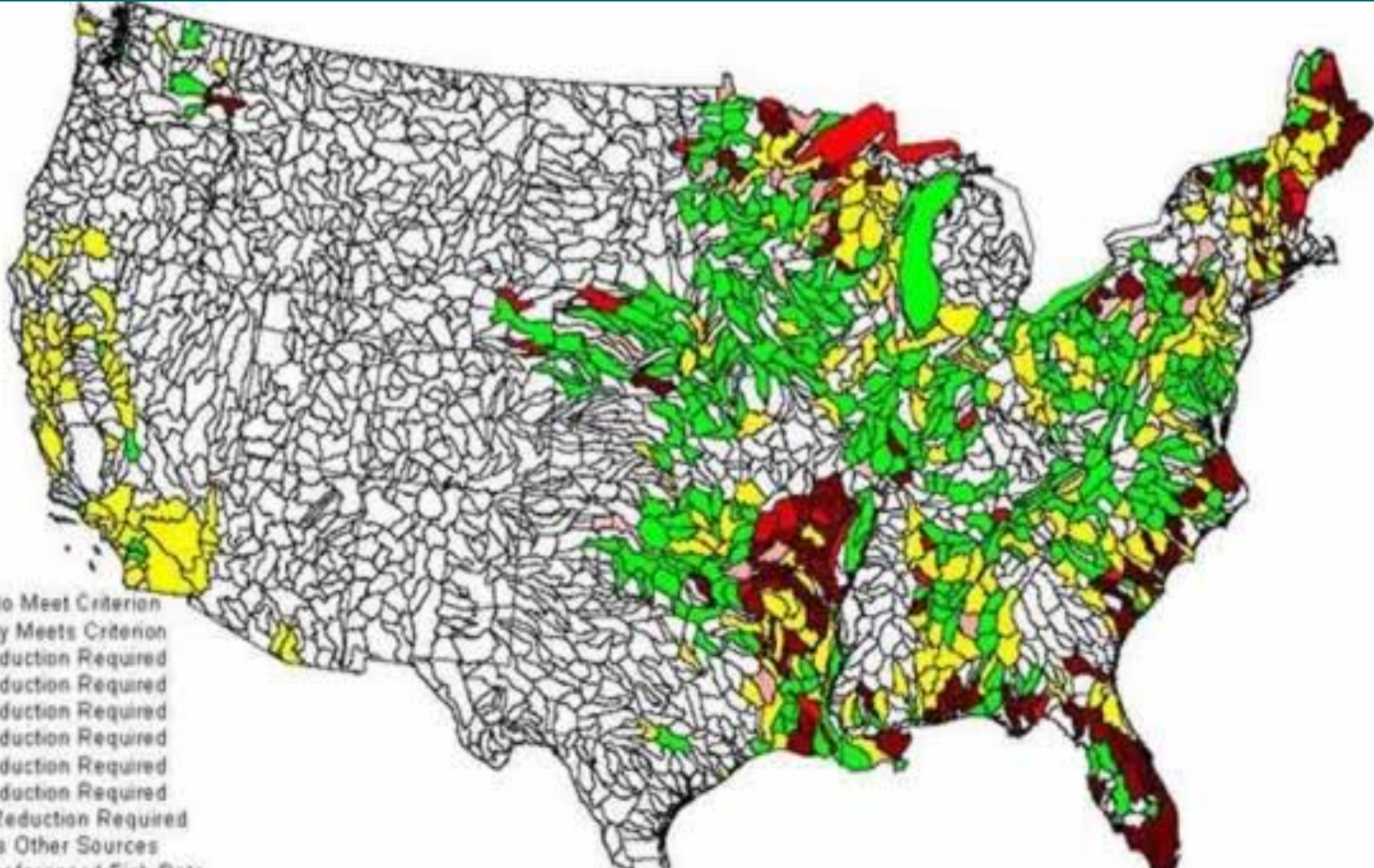
- Aluminum
- Arsenic
- Cadmium
- Lead
- Mercury
- Thallium
- tin

Heavy metal poisoning

- Lead-greatest toxicant in hx of world
- mercury-signif for children, vaccines, dental amalgams, fish, water (Marin mercury is 3-4 parts per trillion, too high per Dr. William Rea, Ctr for Env Health, Dallas, Tx)
- Bay area hot spot on the mercury map of US- San Jose mercury mine, gold mining in Sierras, power plants, oil refining

Mercury map of US

<http://water.epa.gov/type/watersheds/datait/maps/poster.cfm>



Heavy metal testing

- Urine fractionated porphyrins best
- Old std: rbc or 24 hour urine
- Hair, may be helpful if elevated vs artifact, also some children fail to excrete, therefore test may be false positive
- Other physicians: chelation flush of heavy metals, problematic if not have nutrients and if not stooling
- High MCV on blood cell count, defic zinc, magnesium, manganese, selenium and molybdenum

Heavy metal detoxification

- Stop exposure
- Not eat mercury containing fish, not eat out of aluminum cans, carpet free of cadmium, no thimerosal in vaccines
- Stooling 2-3x.day
- Epsom salt baths, 1 cup bath 20 minute soak
- Zinc, magnesium, manganese, molybdenum, selenium
- Amino acids
- Antioxidants (vitamins C, E, glutathione and after body cleared of heavy metal then alpha lipoic acid)
- Methylation (methylb12, 5tetramethylhydrofolate)
- Water filtration (distilled or reverse osmosis)
- Chelation may be needed if genetics of detoxification very weak or if degree of heavy metal poisoning is severe

Biotoxins

- **Bacteria**

- Clostridium difficile (antibiotic-induced bacterial overgrowth gut)

- PLoS One. 2010 Jun 11;5(6):e11071.

- **-Staph aureus Regulation of hemolysin expression and virulence of Staphylococcus aureus by a serine/threonine kinase and phosphatase.**

- [Burnside K](#), [Lembo A](#), [de Los Reyes M](#), [Iliuk A](#), [Binhtran NT](#), [Connelly JE](#), [Lin WJ](#), [Schmidt BZ](#), [Richardson AR](#), [Fang FC](#), [Tao WA](#), [Rajagopal L](#).

- Department of Pediatric Infectious Diseases, University of Washington and Seattle Children's Hospital Research Institute, Seattle, Washington, USA.

- **Abstract**

- Exotoxins, including the hemolysins known as the alpha (alpha) and beta (beta) toxins, play an important role in the pathogenesis of Staphylococcus aureus infections

Biotoxins

- - Mold

estrogenic mycotoxin produced by several fungi of Fusarium genera, a mold which can contaminate food Toxicol. 2010 Nov;56(6):956-63. Epub 2010 Jul 6. Int J Food Microbiol. 2010 May 15;139(3):210-3. Epub 2010 Feb 13.

- **Ochratoxin A production by Aspergillus** isolated from cereals and derived products, dried fruits, cacao, grape juice Food Chem Toxicol. 2009 Nov;47(11):2847-52. Epub 2009 Sep 9 and grapes Int J Food Microbiol. 2010 May 15;139(3):210-3. Epub 2010 Feb 13. ,

Biotoxins, mold, cont'd

- pistacio nuts Food Microbiol. 2008 Aug;25(5):683-9. Epub 2008 Mar 26. , corn, cornflakes Int J Food Microbiol. 2008 Mar 31;123(1-2):81-7. Epub 2007 Dec 23.
- Distribution of **fumonisin and aflatoxins in corn fractions** during industrial **cornflake** processing.
- pine nuts Lett Appl Microbiol. 2007 Jan;44(1):68-72., Contamination of pine nuts by **fumonisin (mycotoxin)** produced by strains of **Fusarium proliferatum** isolated from Pinus pinea.
- rice Distribution of total **aflatoxins** in milled fractions of hulled rice., Castells M, Ramos AJ, Sanchis V, Marín S. J Agric Food Chem. 2007 Apr 4;55(7):2760-4. Epub 2007 Mar 10.
- Colors of molds help to identify -golden staphyloxanthin of Staphylococcus aureus (bacteria), the blue-green pyocyanin of Pseudomonas spp., and the dark brown or black melanin pigments of Cryptococcus neoformans and Aspergillus spp. Trends Microbiol. 2009 Sep;17(9):406-13. Epub 2009 Aug 31.

Molds induce food allergies via mycotoxins

- Toxicol Appl Pharmacol. 2009 May 15;237(1):41-8.
- **The food contaminant deoxynivalenol, decreases intestinal barrier permeability and reduces claudin expression.**
- [Pinton P](#), [Nougayrède JP](#), [Del Rio JC](#), [Moreno C](#), [Marin DE](#), [Ferrier L](#), [Bracarense AP](#), [Kolf-Clauw M](#), [Oswald IP](#), Toulouse, France.
-
- Deoxynivalenol (DON) is a mycotoxin that commonly contaminates cereals and causes various toxicological effects. We demonstrated that, in intestinal epithelial cell lines from human (Caco-2) origin, DON decreases trans-epithelial electrical resistance (TEER) and increases in a time and dose-dependent manner the paracellular permeability to 4 kDa dextran and to pathogenic Escherichia coli across intestinal cell monolayers. These alterations of barrier function were associated with a specific reduction in the expression of claudins. In conclusion, DON alters claudin expression and decreases the barrier function of the intestinal epithelium. Considering that high levels of DON may be present in food or feed, consumption of DON-contaminated food/feed may induce intestinal damage and has consequences for human and animal health. (JG: food allergies)

Toxins cause oxidative stress, autonomic disease, and proinflammation

- Curr Opin Pediatr. 2009 Apr;21(2):222-9.
- **Moving towards making social toxins mainstream in children's environmental health.**
- [Wright RJ.](#)
- The Channing Laboratory, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, 181 Longwood Avenue, Boston, MA 02067, USA. rerjw@channing.harvard.edu
- **Abstract**
- **PURPOSE OF REVIEW:** Although traditional disciplinary research theory and methods have focused separately on how social and physical environmental factors affect children's health, evolving research underscores important integrated effects.
- **RECENT FINDINGS:** This review outlines the specific reasons why social determinants should be considered mainstream in children's environmental health research with particular focus on interactive effects between social and physical hazards. These include sensitivity of overlapping physiological systems, via epigenesis, programming, and plasticity to social and physical environmental moderation that may impact health across the life span; ways in which social environmental vulnerabilities moderate the effects of physical environmental factors, providing specific examples related to respiratory health and neurodevelopment; overlapping exposure distribution profiles; and relevance to pediatric health disparities.
- **SUMMARY:** Because of the covariance across exposures, and evidence that social stress and other environmental toxins (e.g., pollutants, tobacco smoke) may influence common physiological pathways (e.g., oxidative stress, proinflammatory immune pathways, autonomic disruption), understanding the potential synergistic effects promises to more completely inform children's environmental health risk. Although this discussion focuses around the **respiratory and neurological systems**, these concepts extend more broadly to children's psychological and physical development

Treatment of toxin elevation

- General principles:

Organic, fresh (lessens mold ingestion), whole foods (no preservatives)

Water filtration-distilled or reverse osmosis

Determine exposure and stop exposure

Retest to ensure recovering

Boost detoxification system of body, liver

Test phase 1 and phase 2 detox system (Genova oxid stress)

Antioxidants (urine lipid peroxides Genova oxid stress or DNA and RNA oxidative stress, Laboratoire Phillippe Auguste)

PCB poisoning

- Polychlorinated hydrocarbons, exposure from gasoline, before it was removed (old filling stations, soil)
- Test: urine fractionated porphyrins
- Treatment: general detoxification principles, stop PICA (provide minerals, nutrients, etc)

Antiinflammation improves brain health –whether inflammation is from allergy, infection or toxin

- Brain Res. 2010 Jun 21;1339:60-9. Epub 2010 Apr 24.
- **Curcumin protects pre-oligodendrocytes from activated microglia in vitro and in vivo.**
- [He LF](#), [Chen HJ](#), [Qian LH](#), [Chen GY](#), [Buzby JS](#).
- Shanghai Institute for Pediatric Research, Xinhua Hospital affiliated to Shanghai Jiao Tong University School of Medicine, Shanghai, PR China. waxflying@yahoo.com.cn
- **Abstract**
- Infection and inflammation leading to injury or death of pre-oligodendrocytes (preOLs) is one of the principal initiating mechanisms in the pathogenesis of preterm periventricular leukomalacia (PVL). The present study explores the possible protective effect of curcumin against the toxicity of lipopolysaccharide (LPS)-activated microglia on preOLs in vitro and in vivo. In vitro, preOLs in coculture with microglia exhibited increased apoptosis after exposure to LPS. LPS also induced significantly increased expression of inducible nitric oxide synthase (iNOS) and NADPH oxidase (NOX) subunits, p67-phox and gp91-phox in microglia. Our results suggest that iNOS and NOX contribute to the apoptosis of preOLs by activated microglia. The potential anti-inflammatory effects of curcumin were tested to determine if they could help to minimize microglia-mediated damage. Curcumin (10 microg/ml) was found to significantly inhibit the apoptosis of preOL and expression of either iNOS or NOX in the LPS-activated microglia. In vivo, curcumin was administered (50 mg/kg/day, i.p.) to two-day-old neonatal Sprague-Dawley rats subjected to intracerebral injection of LPS. Treatment with curcumin either 1h before or immediately after LPS injection significantly ameliorated white matter injury and loss of preOLs, decreased activated microglia, and inhibited microglial expression of iNOS and translocation of p67phox and gp91phox to the microglial cell membranes in neonatal rat brains following LPS injection. These results suggest that **curcumin has a protective effect on infection-driven white matter injury**, which is associated with suppression of iNOS and NOX activation. Consequently, curcumin may have potential as a protective agent against immature white matter injury.

Resources



Pharmacies

- Compounding pharmacies

Ross Valley , Greenbrae, Marin County, CA 415 924-2454

- Nutrient recipe based:

Amino acid recipe powder-HRI pharmacy, Warrenville, IL, 630 505-0300

Multivitamin/multimineral powder-HRI pharmacy

- Sterile injectables

Abbott's-Berkeley, California, 510 548-8777

Grandpa's pharmacy, Placerville, California, 530 622-2323

Hopewell, Hopewell, NJ, 800 792 6670

Labs

- Labs
- Doctor's Data West Chicago, IL, 800 323-2784, www.doctorsdata.com
- Genova Asheville, NC, 800 522-4762, www.GDX.net
- Great Plains, Lenexa, KS, 913 341-8949, www.greatplainslaboratory.com
- Igenex, Palo Alto, CA 800 832-3200, www.igenex.com
- Meridian Valley Lab, Renton, WA 425 271-8689
www.meridianvalleylab.com
- Metametrix, Duluth, GA, 800 221-4640, www.metametrix.com

Useful websites

- <http://www.autism.com>

The Autism Research Institute (ARI) is the hub of a worldwide network of parents and professionals concerned with autism

- <http://www.childneurologysociety.org/>

The Child Neurology Society is the preeminent non-profit professional association of pediatric neurologists in the United States

- <http://www.ldonline.org/questions/nonverbal>

Comprehensive site, lots of info, can ask questions of experts, glossary, definitions of LD

More websites

- <http://www.interdys.org/>

The International Dyslexia Association (IDA) is a non-profit organization dedicated to helping individuals with dyslexia, their families and the communities that support them

- <http://ilads.org/>

International Lyme And Associated Diseases Society
ILADS promotes understanding of Lyme and its associated diseases through research and education

