AUTISM IS TREATABLE

KENNETH A. BOCK, MD, FAAFP, FACN, CNS

BOCK INTEGRATIVE MEDICINE

50 Old Farm Road
Red Hook, NY 12571
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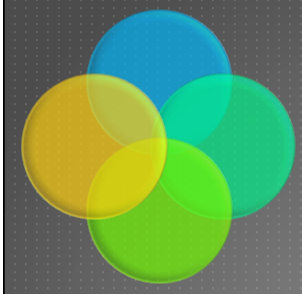
www.bockintegrative.com

The Epidemic of Autism:



- 1 in 50 children is now diagnosed with autism !!!!!!!!!!!!!!
- 1 in 10 children is diagnosed with ADHD
- 1 out of every 10 children is diagnosed with asthma
- 1 out of every 4 children suffers from respiratory or other allergies *

APPROXIMATELY 30 MILLION CHILDREN – MORE THAN 1/3 OF ALL OUR KIDS – ARE AFFECTED BY ONE OF THE FOUR NEW CHILDHOOD EPIDEMICS

Vital & Health Statistics
http://www.cdc.gov/nchs/data/series/sr_10/sr10_234.pdf

Autism Spectrum Disorders

ADHD | Asperger's Syndrome | PDD | Autism

Social Communication Disorder

AUTISM: *A MEDICAL DISORDER*

Autism is classically defined by a cluster of behaviors, but autism is a medical disorder

These behaviors include:

1. Impaired social interaction
2. Impaired social communication
3. Repetitive behaviors
4. Markedly restricted repertoire of activities and interests

TREATMENT CONSIDERATIONS

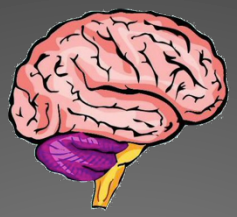
- Thinking of Autism as a strictly behavioral disorder
  - Leads to behavioral (and clinical) treatment approaches
- Expanding one's view of Autism as a medical disorder leads to integrative medicine treatment approaches
  - Medications
  - Dietary modifications
  - Nutritional supplementation
  - Detoxification

  PLUS
  - Behavioral and clinical approaches

DYSFUNCTION IN ASD

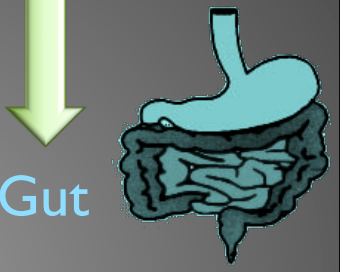
- Neurologic/Neurotoxic
- Gastrointestinal
- Metabolic
- Immunologic

CONCEPT OF BRAIN EFFECTS SECONDARY TO PATHOLOGY ELSEWHERE IN BODY



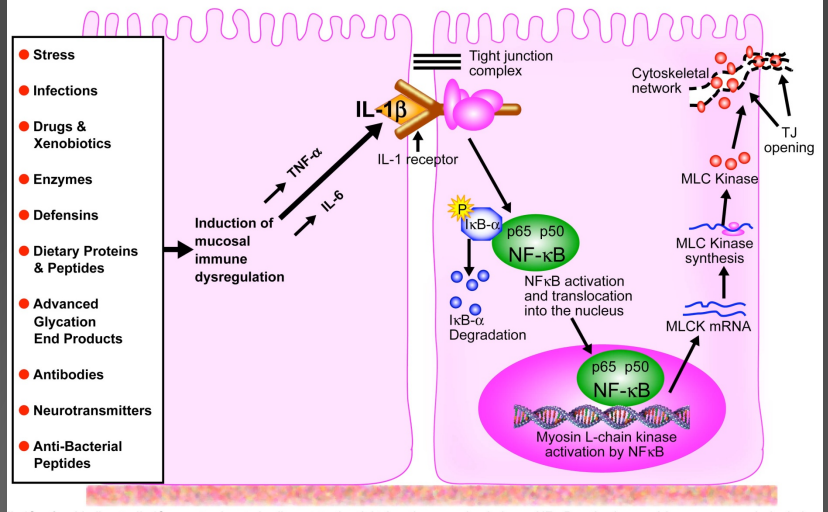
Brain

Gut-Brain Axis



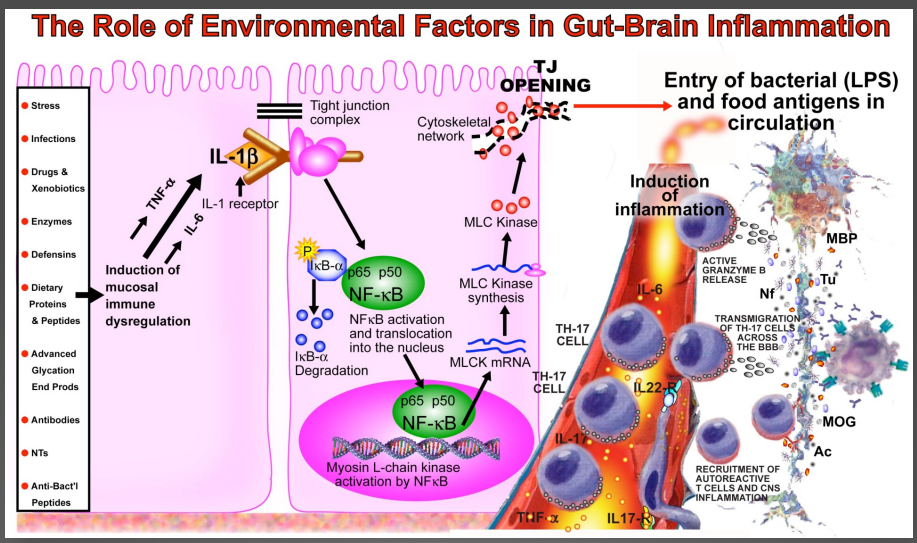
Gut

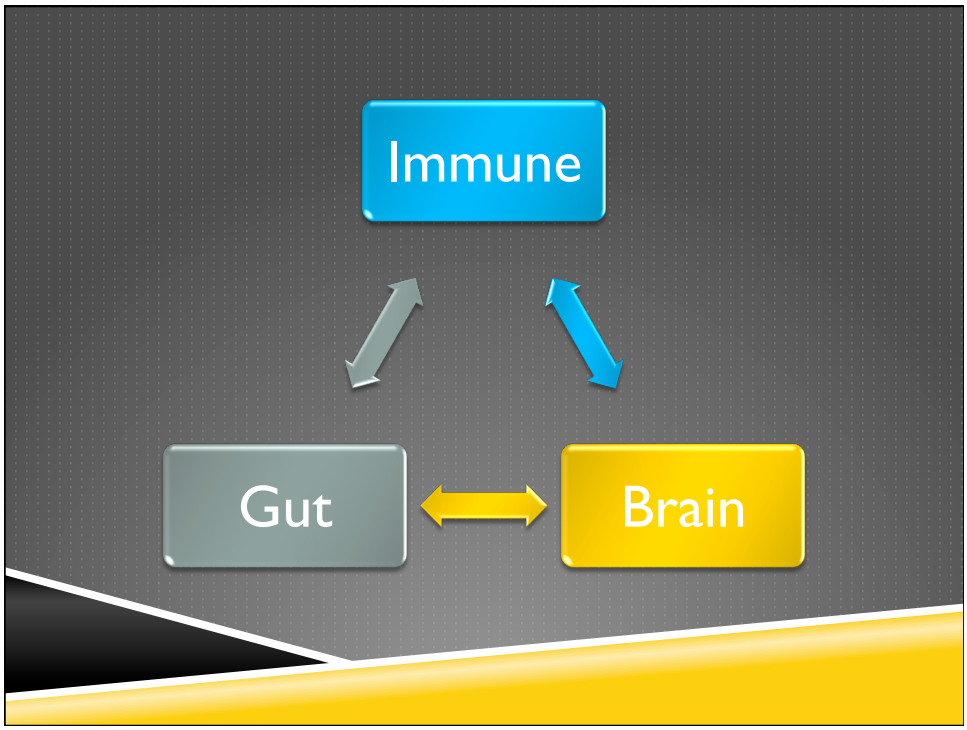
Modulation of tight junction barrier by environmental factors, induction of mucosal immune dysregulation and production of proinflammatory cytokines, in particular IL-1β.



IL-1β, after binding to IL-1β receptor located adjacent to the tight junction complex induces NF-κB activation, and increases myosin L-chain kinase expression and activation. Activation and synthesis of MLC kinase result in tight junction destruction and intestinal permeability.

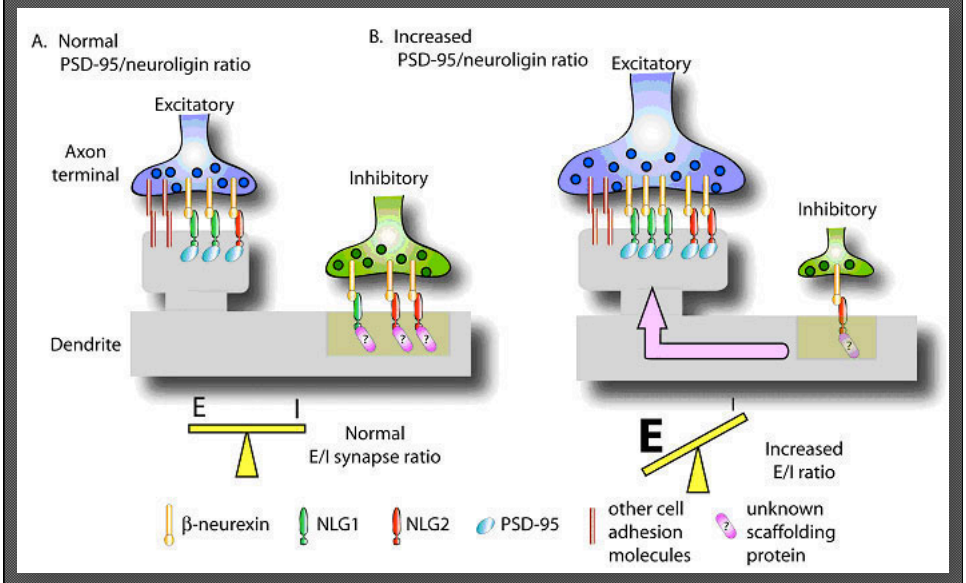
modified from Al-Sadi et al., The Journal of Immunology, 180: 5653-5661, April, 2008


The Role of Environmental Factors in Gut-Brain Inflammation
Stress
Infections
Drugs & Xenobiotics
Enzymes
Defensins
Dietary Proteins & Peptides
Advanced Glycation End Prods
Antibodies
NTs
Anti-Bact'l Peptides
Induction of mucosal immune dysregulation
TNF-α
IL-6
IL-1β
IL-1 receptor
Tight junction complex
TJ OPENING
Cytoskeletal network
MLC Kinase
MLC Kinase synthesis
MLCK mRNA
P
IκB-α
p65 p50
NF-κB
NFκB activation and translocation into the nucleus
IκB-α Degradation
p65 p50
NF-κB
Myosin L-chain kinase activation by NFκB
Entry of bacterial (LPS) and food antigens in circulation
Induction of inflammation
IL-6
TH-17 CELL
TH-17 CELL
IL22-R
IL-17
TGF-α
IL17-R
ACTIVE GRANZYME B RELEASE
TRANSMIGRATION OF TH-17 CELLS ACROSS THE BBB
RECRUITMENT OF AUTOREACTIVE T CELLS AND CNS INFLAMMATION
MBP
Tu
Nf
MOG
Ac


Immune
Gut
Brain

DISORDERED PROCESSES IN AUTISM

- Neurologic
  - Neurological Autoimmunity
  - Brain Inflammation
  - Dysregulated Neurotransmitters
  - Imbalanced Excitatory/Inhibitory Transmission



http://www.neuroscience.ubc.ca/husseini_projects1.htm

ARBACLOFEN

- The 12-week randomized controlled trial of STX209 (arbaclofen), an experimental agent that acts upstream from mGluR5 receptor signaling and is thought to augment inhibitory neurotransmission, showed potential for "clinically meaningful improvements in social function," according to investigators led by Jeremy Veenstra-VanderWeele, MD, associate professor of psychiatry, pediatrics and pharmacology, Vanderbilt University Medical Center, Nashville, Tennessee.

ARBACLOFEN

- "There is a long-standing hypothesis that there is an imbalance between excitatory and inhibitory neurotransmission in autism," Dr. Veenstra-VanderWeele said in a press conference.
- "And if arbaclofen acts as an agonist in the GABA [γ-aminobutyric acid] system like we think it does, it may rectify that imbalance in some patients."

DISORDERED PROCESSES IN AUTISM

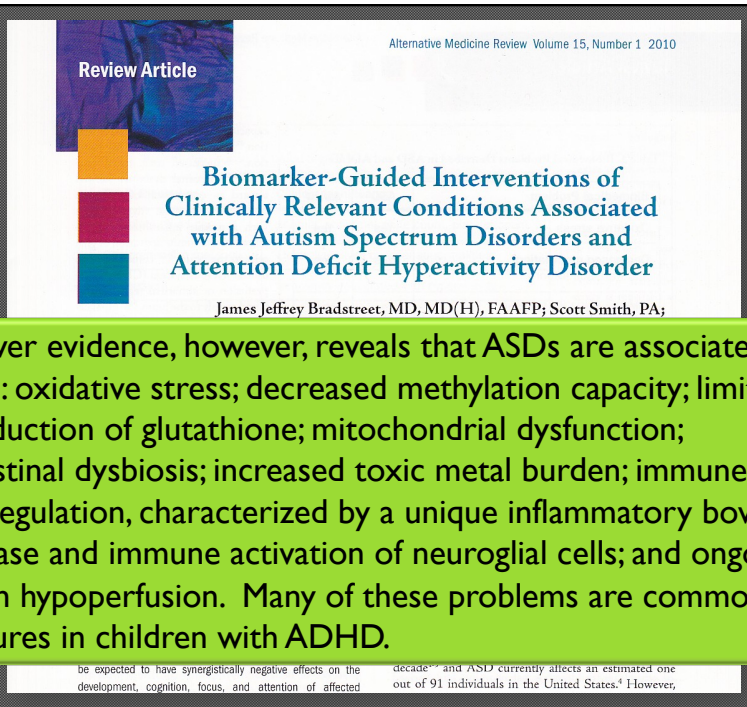
- GI
  - Maldigestion/Malabsorption
  - Intestinal Dysbiosis
  - Intestinal Hyperpermeability
  - Food Allergies/Sensitivities
  - Gut Inflammation
  - Nutritional Deficiencies/Imbalances

DISORDERED PROCESSES IN AUTISM

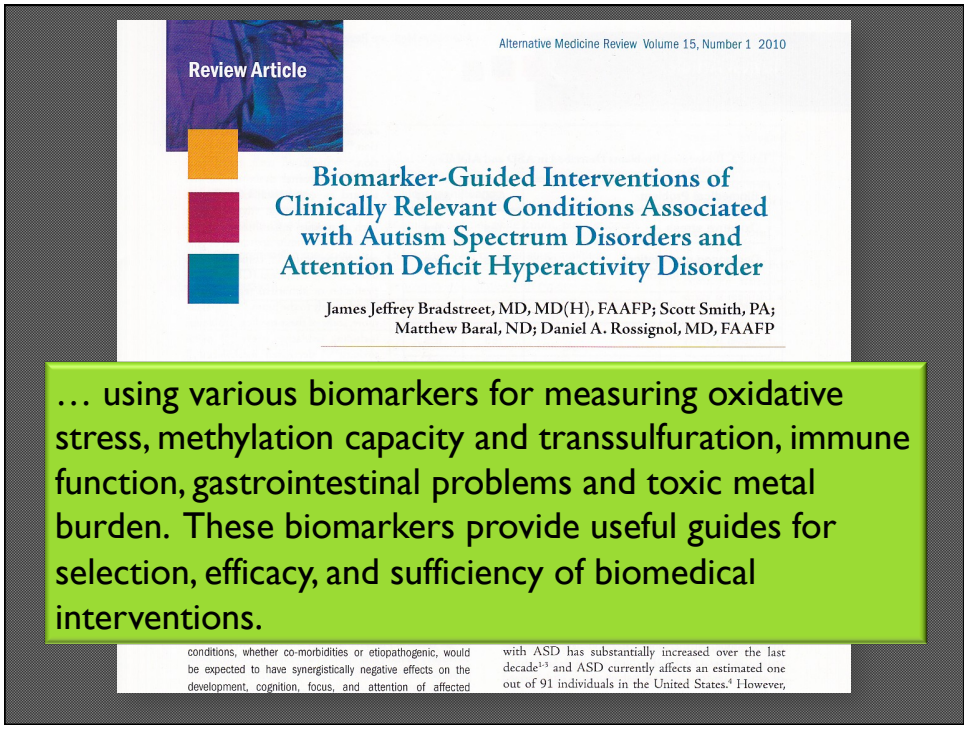
- Immune
  - Immune Deficiency
  - Recurrent Infections
  - Immune Dysregulation
  - Allergies
  - Autoimmunity
  - Chronic Inflammation
    - Upregulated microglia
    - Increased pro-inflammatory signals

DISORDERED PROCESSES IN AUTISM

- Metabolic/Biochemical
  - Oxidative Stress
  - Mitochondrial Dysfunction
  - Glutathione Depletion



Newer evidence, however, reveals that ASDs are associated with: oxidative stress; decreased methylation capacity; limited production of glutathione; mitochondrial dysfunction; intestinal dysbiosis; increased toxic metal burden; immune dysregulation, characterized by a unique inflammatory bowel disease and immune activation of neuroglial cells; and ongoing brain hypoperfusion. Many of these problems are common features in children with ADHD.



SUBGROUPS OF ASD/"AUTISMS"

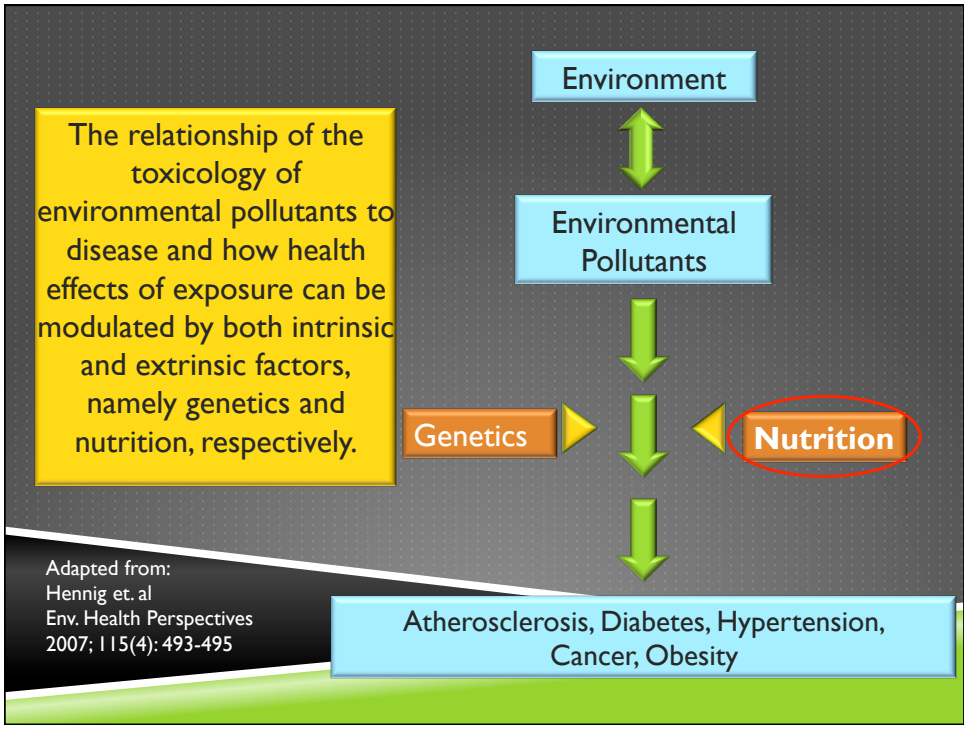
- Subgroups
  - GI/Gut-Brain
  - Immune Dysfunction
    - Immune Deficiency
    - Immune Dysregulation
      - Autoimmunity
      - Allergy/Sensitivities
      - Chronic Inflammation
  - Infections
    - Viral/Bacterial (Strep, Anaerobic)/Fungal/Parasitic

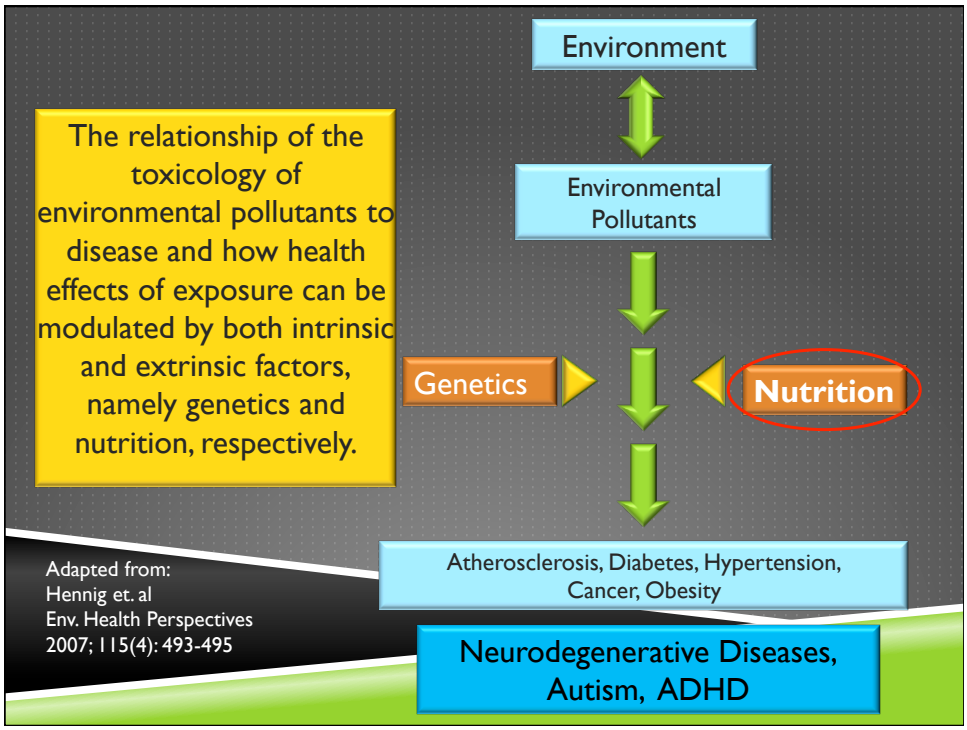
SUBGROUPS OF ASD/"AUTISMS"

- Subgroups (cont.)
  - Metabolic
    - Enzyme Dysfunction
    - Mitochondrial Dysfunction
  - Nutritional imbalances
  - Hormonal imbalances
  - Toxins
    - Heavy Metals/Chemicals

SUSPECTED COMMONALITIES OF ASD / "AUTISMS"

- Toxins
  - Heavy Metals/Chemicals
- Oxidative Stress
- Inflammation


The relationship of the toxicology of environmental pollutants to disease and how health effects of exposure can be modulated by both intrinsic and extrinsic factors, namely genetics and nutrition, respectively.
Environment
Environmental Pollutants
Genetics
Nutrition
Adapted from:
Hennig et. al
Env. Health Perspectives
2007; 115(4): 493-495
Atherosclerosis, Diabetes, Hypertension, Cancer, Obesity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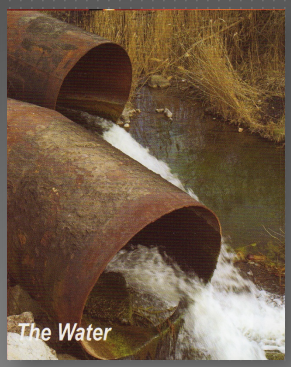
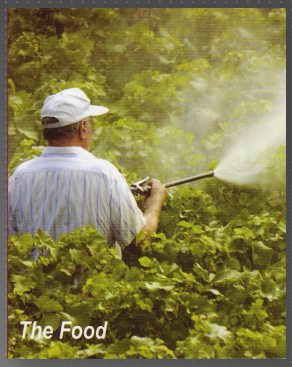
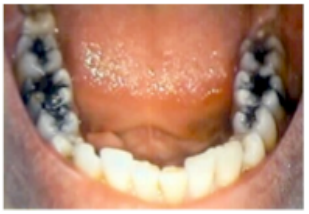
Adapted from:
Hennig et. al
Env. Health Perspectives
2007; 115(4): 493-495
Atherosclerosis, Diabetes, Hypertension, Cancer, Obesity
Neurodegenerative Diseases, Autism, ADHD

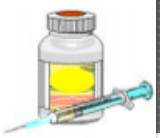
SOURCES OF TOXINS





Environmental Toxicity

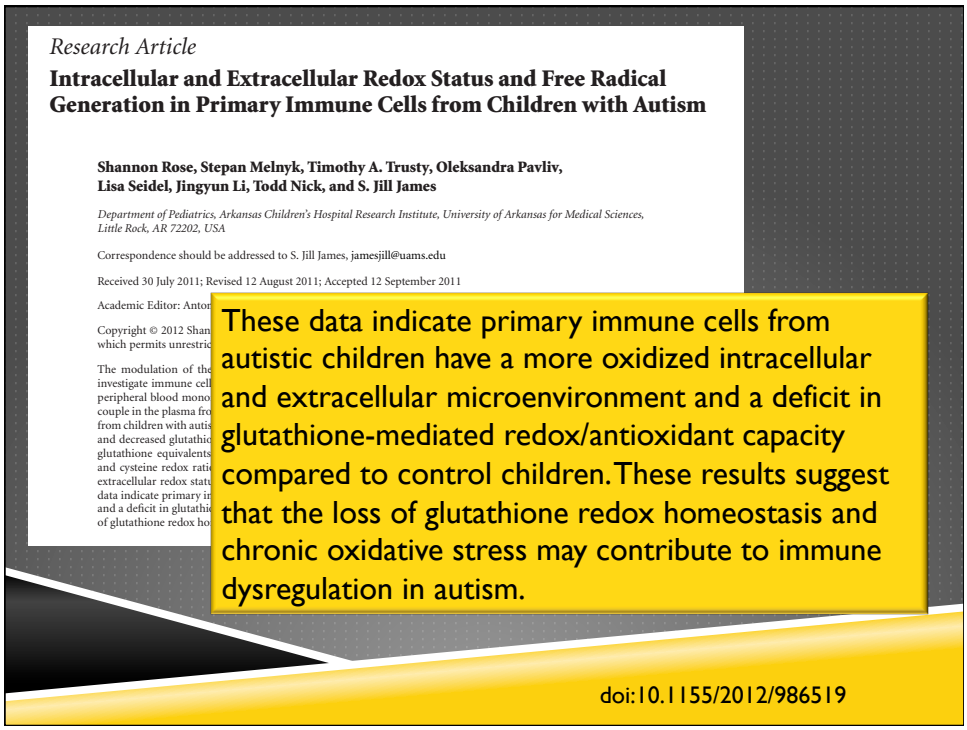




OXIDATIVE STRESS IN AUTISM

- Increased levels of prooxidants
  - Organic toxins
    - Pesticides
    - PCBs
  - Heavy metals
    - Mercury
    - Lead
  - Inflammatory cytokines

Sadjel-Sulkowski et al
Am J Biochem and Biotech. 4(2):73-84, 2008



Research Article

Intracellular and Extracellular Redox Status and Free Radical Generation in Primary Immune Cells from Children with Autism

Shannon Rose, Stepan Melnyk, Timothy A. Trusty, Oleksandra Pavliv, Lisa Seidel, Jingyun Li, Todd Nick, and S. Jill James

Department of Pediatrics, Arkansas Children's Hospital Research Institute, University of Arkansas for Medical Sciences, Little Rock, AR 72202, USA

Correspondence should be addressed to S. Jill James, jamesjill@uams.edu

Received 30 July 2011; Revised 12 August 2011; Accepted 12 September 2011

Academic Editor: Anton

The modulation of the
investigate immune cell
peripheral blood mono
couple in the plasma fro
from children with autis
and decreased glutathio
glutathione equivalents
and cysteine redox ratio
extracellular redox statu
data indicate primary i
and a deficit in glutathi
of glutathione redox ho

These data indicate primary immune cells from autistic children have a more oxidized intracellular and extracellular microenvironment and a deficit in glutathione-mediated redox/antioxidant capacity compared to control children. These results suggest that the loss of glutathione redox homeostasis and chronic oxidative stress may contribute to immune dysregulation in autism.

doi:10.1155/2012/986519

Citation: Transl Psychiatry (2012) 2, e134; doi:10.1038/tp.2012.61

www.nature.com/tp

Evidence of oxidative damage and inflammation associated with low glutathione redox status in the autism brain

S Rose, S Melnyk, O Pavliv, S Bai, TG Nick, RE Frye and SJ James

Despite increasing evidence of oxidative stress in the pathophysiology of autism, most studies have not evaluated biomarkers within specific brain regions, and the functional consequences of oxidative stress remain relatively understudied. We examined frozen samples from the cerebellum and temporal cortex (Brodmann area 22 (BA22)) from individuals with autism and unaffected controls ($n=15$ and $n=12$ per group, respectively). Biomarkers of oxidative stress, including reduced glutathione (GSH), oxidized glutathione (GSSG) and glutathione redox/antioxidant capacity (GSH/GSSG), were measured. Biomarkers of oxidative protein damage (3-nitrotyrosine; 3-NT) and oxidative DNA damage (8-oxo-deoxyguanosine; 8-oxo-dG) were also assessed. Functional indicators of oxidative stress included relative levels of 3-chlorotyrosine (3-CT), an established biomarker of a chronic inflammatory response, and aconitase activity, a biomarker of mitochondrial superoxide production. Consistent with previous studies on plasma and immune cells, GSH and GSH/GSSG were significantly decreased in both autism cerebellum ($P<0.01$) and BA22 ($P<0.01$). There was a significant increase in 3-NT in the autism cerebellum and BA22 ($P<0.01$). Similarly, 8-oxo-dG was significantly increased in autism cerebellum and BA22 ($P<0.01$ and $P=0.01$, respectively), and was inversely correlated with GSH/GSSG in the cerebellum ($P<0.01$). There was a significant increase in 3-CT levels in both brain regions ($P<0.01$), whereas aconitase activity was significantly decreased in autism cerebellum ($P<0.01$), and was negatively correlated with GSH/GSSG ($P=0.01$). Together, these results indicate that decreased GSH/GSSG redox/antioxidant capacity and increased oxidative stress in the autism brain may have functional consequence in terms of a chronic inflammatory response, increased mitochondrial superoxide production, and oxidative protein and DNA damage.

Translational Psychiatry (2012) **2**, e134; doi:10.1038/tp.2012.61; published online 10 July 2012

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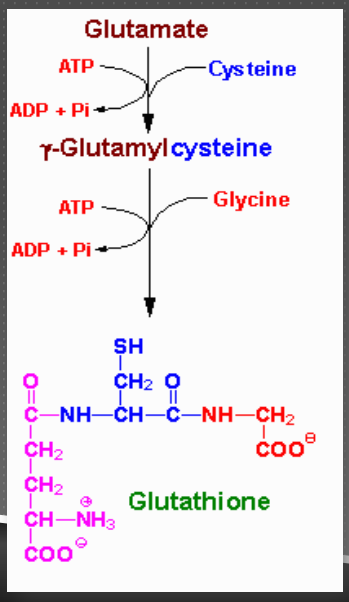
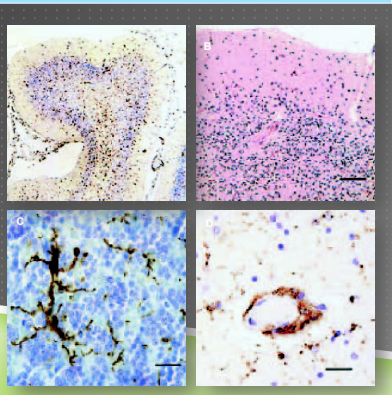


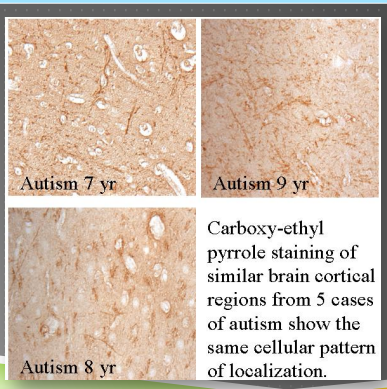
GLUTATHIONE

NEUROINFLAMMATION AND OXIDATIVE STRESS IN AUTISM

Neuroglial activation and neuroinflammation in the brain of patients with autism
Vargas et al, 2005, Annals of Neurology

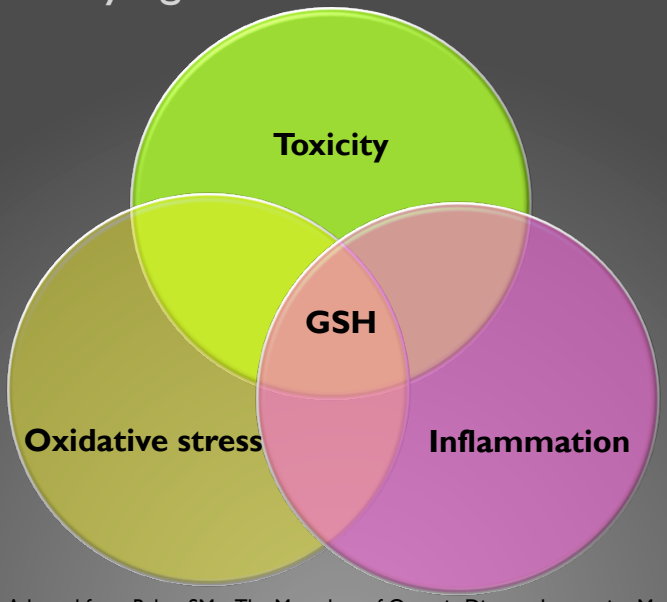


Carboxy-ethyl pyrrole staining in autism brains - dendrites: evidence of lipid peroxidation (from oxidative stress) that could impact synaptic transmission – Perry, Salomon 2005 abstract



Carboxy-ethyl pyrrole staining of similar brain cortical regions from 5 cases of autism show the same cellular pattern of localization.

Underlying Features of Chronic Illness



Adapted from Baker, SM – The Metaphor of Oceanic Disease, Integrative Medicine, February-March, 2008

OPINION

An Immune Disorder at the Root of Autism

By MOISES VELASQUEZ-MANOFF
Published: August 25, 2012

IN recent years, scientists have made extraordinary advances in understanding the causes of autism, now estimated to afflict 1 in 88 children. But remarkably little of this understanding has percolated into popular awareness, which often remains fixated on vaccines.

Eleanor Davis

So here's the short of it: At least a subset of autism — perhaps one-third, and very likely more — looks like a type of inflammatory disease. And it begins in the womb.

It starts with what scientists call immune dysregulation. Ideally, your immune system should operate like an enlightened action hero, meting out inflammation precisely, accurately and with deadly force when necessary, but then quickly returning to a Zen-like calm. Doing so requires an optimal balance of pro- and anti-inflammatory muscle.

In autistic individuals, the immune system fails at this balancing act. Inflammatory signals dominate. Anti-inflammatory ones are inadequate. A state of chronic activation prevails. And the more skewed toward inflammation, the more acute the autistic symptoms.

Nowhere are the consequences of this dysregulation more evident than in the autistic brain. Spidery cells that help maintain neurons — called astroglia and microglia — are enlarged from chronic activation. Pro-inflammatory signaling molecules abound. Genes involved in inflammation are switched on.

These findings are important for many reasons, but perhaps the most noteworthy is that they provide evidence of an abnormal, continuing biological process. That means that there is finally a therapeutic target for a disorder defined by behavioral criteria like social impairments, difficulty communicating and repetitive behaviors.

The New York Times
August 2012

NIH Public Access
Author Manuscript
Neurotoxicology. Author manuscript; available in PMC 2009 March 1.

NIH-PA Author Manuscript

Published in final edited form as:
Neurotoxicology. 2008 March ; 29(2): 226–231. doi:10.1016/j.neuro.2007.10.010.

AUTISM: MATERNALLY DERIVED ANTIBODIES SPECIFIC FOR FETAL BRAIN PROTEINS

Daniel Braunschweig[1,7,8], Paul Ashwood[2,7,8], Paula Krakowiak[3,7,8], Irva Hertz-Picciotto[3,7,8], Robin Hansen[4,7,8], Lisa Croen[5], Isaac N. Pessah[6,7,8], and Judy Van de Water[1,7,8,*]

1 *Division of Rheumatology, Allergy and Clinical Immunology, University of California at Davis. Davis, CA 95616 USA*

The presence of these antibodies in the plasma of some mothers of children with autism, as well as the differential findings between mothers of children with early onset and regressive autism may suggest an association between the transfer of IgG autoantibodies during early neurodevelopment and the risk of developing autism in some children.

and adult brain proteins were analyzed by western blot in 61 mothers of children with autistic disorder and 102 controls matched for maternal age and birth year (62 mothers of typically developing children (TD) and 40 mothers of children with non-ASD developmental delays (DD)). We observed reactivity

Brain-Specific Autoantibodies in the Plasma of Subjects with Autistic Spectrum Disorder

MARICEL CABANLIT [a], SHARIFIA WILLS [a], PAULA GOINES [a], PAUL ASHWOOD [b], AND JUDY VAN DE WATER [a]
[a] Division of Rheumatology, Allergy and Clinical Immunology, University of California, Davis, California, USA [b] Department of Microbiology, and the MIND Institute, University of California, Davis, California, USA

Address for correspondence: Judy Van de Water, Division of Rheumatology, Allergy and Clinical Immunology, 451 E. Health Sci. Dr., Suite 6510 GBSF, UC Davis, Davis, CA 95616, USA. Voice: 530-752-2154; fax: 530-752-4669.
javandewater@ucdavis.edu

KEYWORDS
autism • autoantibodies • human brain • hypothalamus • thalamus

ABSTRACT

Multiple brain-specific autoantibodies are present at significantly higher frequency in children with AU. While the potential role of these autoantibodies in AU is currently unknown, their presence suggests a loss of self-tolerance to one or more neural antigens during early childhood.

Pediatric

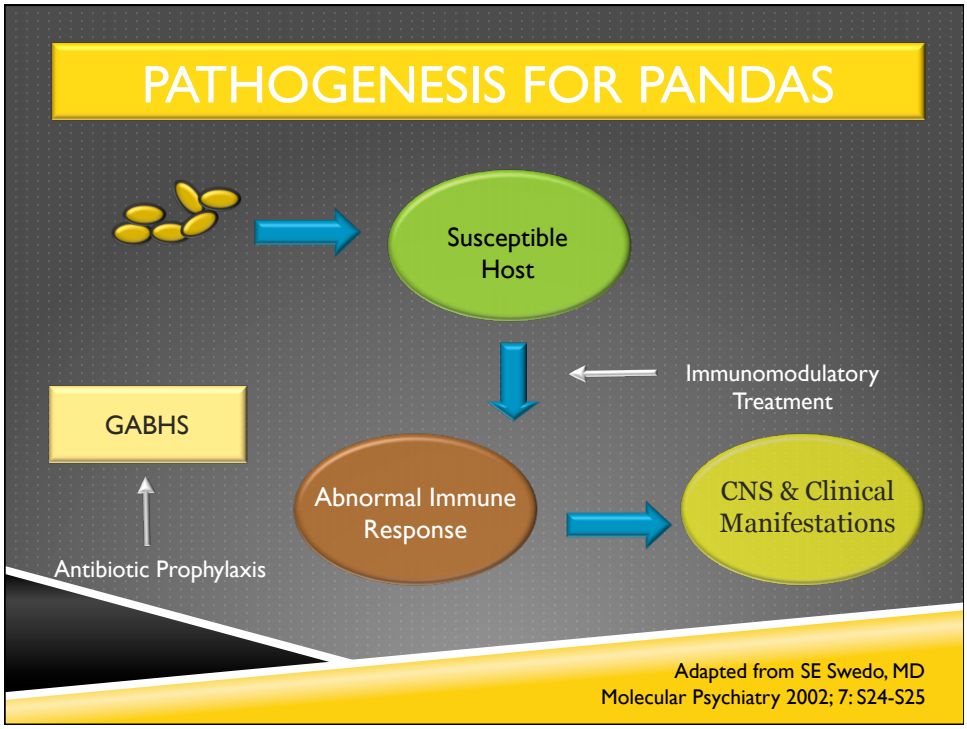
Autoimmune

Neuropsychiatric

Disorders

Associated with

Streptococcal infections



CLASSIC ILLUSTRATIVE CASE

- 12 year-old child suddenly awakens one morning "a different child"
- Fears of being contaminated with germs
  - Doesn't want to be touched
  - Excessive hand washing
- Abrupt onset of twitches and tics, motoric hyperactivity, anxiety, separation anxiety and handwriting deterioration

EVIDENCE SUPPORTING PANDAS DIAGNOSIS

- Elevated Antistreptolysin O (ASO) titer
- Elevated Anti-DNase B titer
- Elevated antineuronal antibodies
- Increased basal ganglia volume measured using volumetric MRI
- Elevated levels of cells expressing the D8/17 marker
- Positive family history of OCD and tic disorders
- Evidence of response to immunomodulatory therapies

IV IG IN CHILDREN WITH AUTISM

- IV IG is used in the treatment of immunological diseases that affect the entire neuroaxis, including the brain, spinal cord, peripheral nerves, muscles and neuromuscular junction
- Minimal risks
- Certain subset of autistic children might benefit
  - Immune deficiency
    - Low immunoglobulin levels
  - Increased autoantibodies
    - Anti-MBP
    - Anti-thyroid
    - Anti-DNase B and anti-streptolysin O

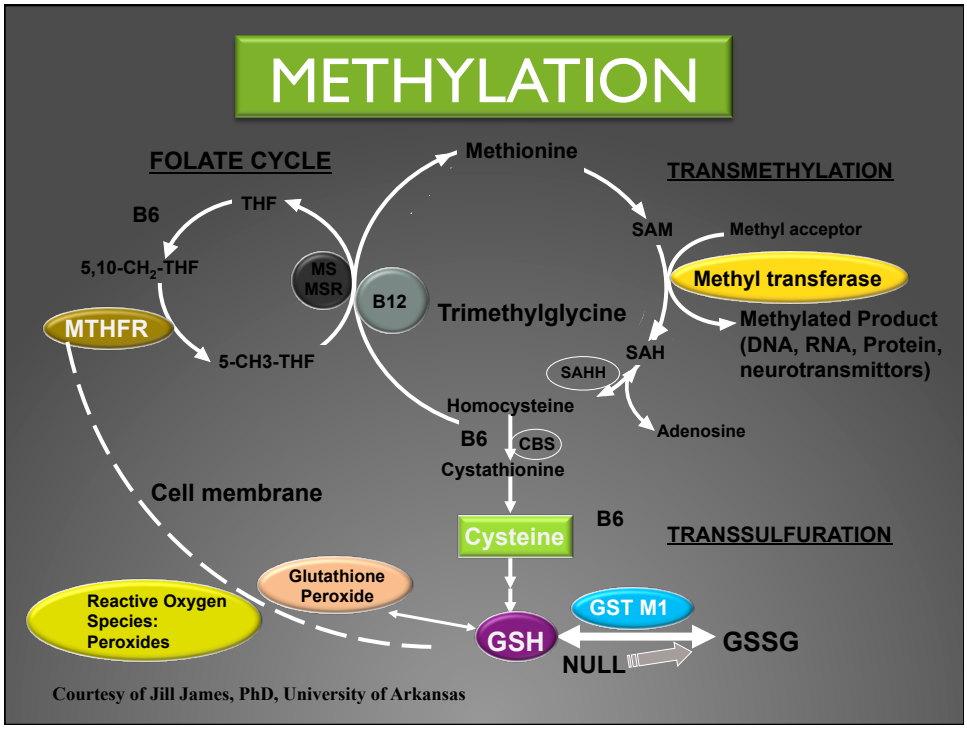
Boris et al
Nutr and Environ Med 2006; 15(4):1-8

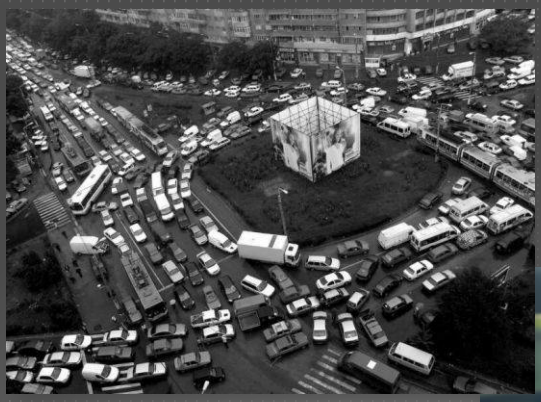
J Clin Immunol (2010) 30 (Suppl 1):S90–S96
DOI 10.1007/s10875-010-9402-9

Adaptive and Innate Immune Responses in Autism: Rationale for Therapeutic Use of Intravenous Immunoglobulin

Furthermore, the presence of autoantibodies against neuronal antigens in mothers of autistic children and in children with autism and the induction of stereotypical changes in mice and rhesus monkeys by autistic maternal immunoglobulin G (IgG) [5–8] argues in favor of the role of the immune system in the pathogenesis of a subset of patients with autism. Therefore, it is not surprising that some studies have reported the beneficial effect of intravenous immunoglobulin (IVIG). Here we have reviewed immunological abnormalities in autism spectrum disorders (ASD) and their response to biological therapies, with a special emphasis on IVIG.

ronal antigens in mothers of autistic children and in children





FIND A PHYSICIAN YOU CAN TRUST AND WHO MAKES YOU FEEL COMFORTABLE

- STRONG KNOWLEDGE BASE
- OPEN
- WILLING TO LOOK AND TO LISTEN
- ABILITY TO SORT THROUGH, ANALYZE AND SYNTHESIZE COMPLEX DATA

THE MEDICAL DETECTIVE

- MULTISYSTEM/ MULTISYMPTOM DISORDERS
- "IF YOU DON'T LOOK, YOU WON'T SEE"
- "IF YOU DON'T LISTEN, YOU WON'T HEAR"

INTEGRATIVE/FUNCTIONAL MEDICINE APPROACH TO CHRONIC INFLAMMATION AND OXIDATIVE STRESS

- Deal with potential underlying contributing factors
  - Infections, Toxins (heavy metals/chemicals), Allergens
  - GI issues
    - Dysbiosis
    - Intestinal hyperpermeability
    - Food allergies/sensitivities
  - Environmental allergies/sensitivities
  - Nutritional deficiencies/imbalances
  - Hormonal imbalances
  - Immunological imbalances

IMPORTANT LAB PRINCIPLES

- Labs should be ordered and interpreted by a doctor/ trained practitioner and then reviewed with him/her
- What is currently in vogue may not hold up upon further research scrutiny
  - i.e., urinary porphyrins

THERAPEUTIC IMPLICATIONS

DIETARY MODIFICATIONS
ANTIOXIDANTS
ANTI-INFLAMMATORY
DETOXIFICATION
IMMUNOMODULATION

RECOVERY CONSIDERATIONS IN ASD

- To be considered "recovered," the child must now be learning and applying a core set of skills at a level and with a quality that reaches the trajectory of typical development in most or all areas
- Furthermore, the recovered individual no longer meets criteria for any ASD

Helt, Kelley et al (2008) Neuropsychol Rev. 18(4): 339-66

POST-RECOVERY RESIDUALS

- Residual co-morbidities after recovery
  - Tics
  - Depression
  - Anxiety
  - Phobias
  - OCD

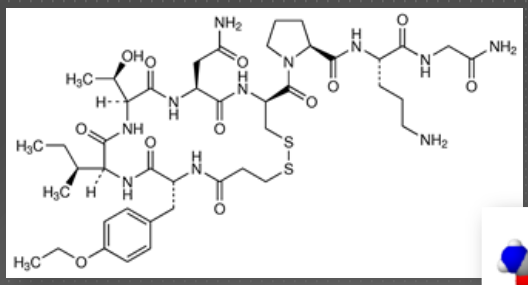
Helt, Kelley et al (2008) Neuropsychol Rev. 18(4): 339-66
Sutera et al (2007) J Autism Dev Disord 37(1): 98-107

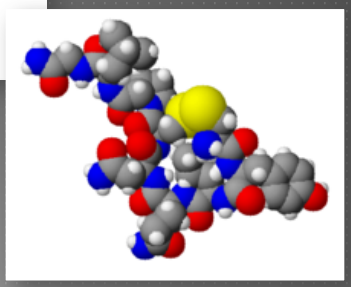
POST-RECOVERY RESIDUALS

- Residual vulnerabilities
  - Higher-order communication
  - Subtle pragmatic deficits
  - Social awkwardness
  - Attention

Helt, Kelley et al (2008) Neuropsychol Rev. 18(4): 339-66
Sutera et al (2007) J Autism Dev Disord 37(1): 98-107

OXYTOCIN





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Review

Intranasal administration of oxytocin: Behavioral and clinical effects, a review

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http://dx.doi.org/10.1016/j.neubiorev.2013.04.012, How to Cite or Link Using DOI

The intranasal (IN-) administration of substances is attracting attention from scientists as well as pharmaceutical companies. The effects are surprisingly fast and specific. …over the last decade most studies focused on their behavioral 'prosocial' effects: from social relations and 'trust' to treatment of 'autism'. …We conclude that IN-OT administration may be a promising approach to influence human communication…

J Child Psychol Psychiatry. 2013 Mar 2. doi: 10.1111/jcpp.12061. [Epub ahead of print]

Research Review: Social motivation and oxytocin in autism - implications for joint attention development and intervention.

Stavropoulos KK, Carver LJ.

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Abstract

BACKGROUND AND SCOPE: The social motivation hypothesis (SMH) suggests that individuals with autism spectrum disorders (ASD) are less intrinsically rewarded by social stimuli than their neurotypical peers. This difference in social motivation has been posited as a factor contributing to social deficits in ASD. Social motivation is thought to involve the neuropeptide oxytocin. Here, we review the evidence for oxytocin effects in ASD, and discuss its potential role in one important social cognitive behavior.

METHODS: Systematic searches were conducted using the PsychINFO and MEDLINE databases and the search terms 'oxytocin' and 'autism'; the same databases were used for separate searches for 'joint attention', 'intervention', and 'autism', using the same inclusion criteria as an earlier 2011 review but updating it for the period 2010 to October 2012.

FINDINGS: Several studies suggest that giving oxytocin to both individuals with ASD and neurotypical individuals can enhance performance on social cognitive tasks. Studies that have attempted to intervene in joint attention in ASD suggest that social motivation may be a particular obstacle to lasting effects.

CONCLUSIONS: The review of the evidence for the SMH suggests a potential role for oxytocin in social motivation deficits in ASD. Because of its importance for later communicative and social development, the focus here is on implications of oxytocin and social motivation in the development of and interventions in joint attention. Joint attention is a central impairment in ASD, and as a result is the focus of several behavioral interventions. In describing this previous research on joint attention interventions in ASD, we pay particular attention to problems encountered in such studies, and propose ways that oxytocin may facilitate behavioral intervention in this area. For future research, integrating behavioral and pharmacological interventions (oxytocin administration) would be a worthwhile experimental direction to improve understanding of the role of oxytocin in ASD and help optimize outcomes for children with ASD.

PMID: 23451765 [PubMed - as supplied by publisher]

CONCLUSIONS: The review of the evidence for the SMH suggests a potential role for oxytocin in social motivation deficits in ASD. Because of its importance for later communicative and social development, the focus here is on implications of oxytocin and social motivation in the development of and interventions in joint attention.

J Child Adolesc Psychopharmacol. 2013 Mar;23(2):123-7. doi: 10.1089/cap.2012.0048. Epub 2013 Mar 12.

Long-term administration of intranasal oxytocin is a safe and promising therapy for early adolescent boys with autism spectrum disorders.

Tachibana M, Kagitani-Shimono K, Mohri I, Yamamoto T, Sanefuji W, Nakamura A, Oishi M, Kimura T, Onaka T, Ozono K, Taniike M.

Molecular Research Center for Children's Mental Development, Osaka University, Suita, Japan.

Abstract

OBJECTIVE: Oxytocin (OT) has been a candidate for the treatment of autism spectrum disorders (ASD), and the impact of intranasally delivered OT on ASD has been investigated. However, most previous studies were conducted by single-dose administration to adults; and, therefore, the long-term effect of nasal OT on ASD patients and its effect on children remain to be clarified.

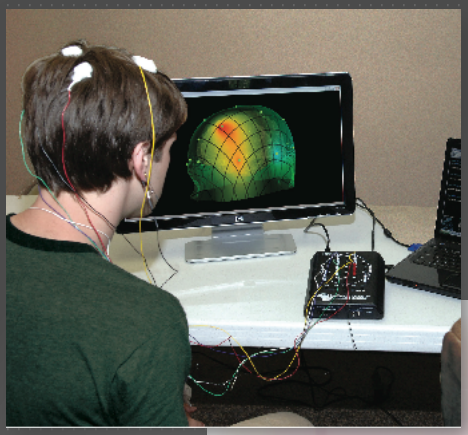
METHODS: We conducted a singled-armed, open-label study in which OT was administered intranasally over the long term to eight male youth with ASD (10-14 years of age; intelligence quotient [IQ] 20-101). The OT administration was performed in a stepwise increased dosage manner every 2 months (8, 16, 24 IU/dose). A placebo period (1-2 weeks) was inserted before each step. The outcome measures were autism diagnostic observation schedule–generic (ADOS-G), child behavior checklist (CBCL), and the aberrant behavior checklist (ABC). In addition, side effects were monitored by measuring blood pressure and examining urine and blood samples.

Six of the eight participants showed improved scores on the communication and social interaction domains of the ADOS-G. … Caregivers of five of the eight participants reported certain positive effects of the OT therapy, especially on the quality of reciprocal communication.

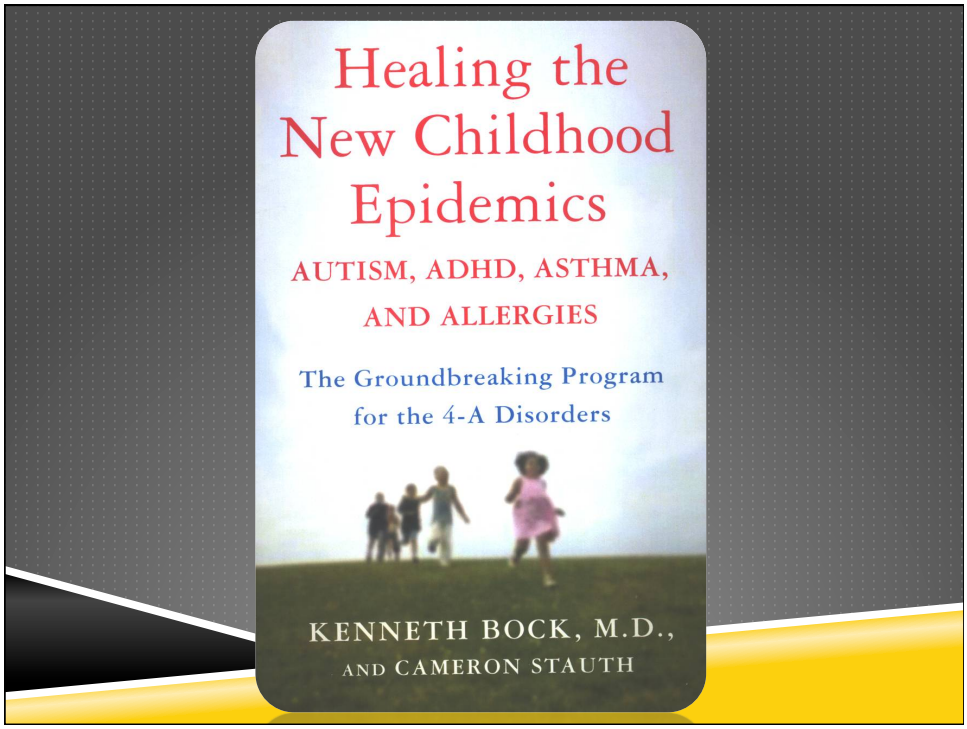
Although our results on the efficacy of long-term nasal OT therapy still remain controversial, to the best of our knowledge, this is the first report documenting the safety of long-term nasal OT therapy for children with ASD

RECOVERY PERSPECTIVES

ASD/PDD → ADHD



NEUROFEEDBACK



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