





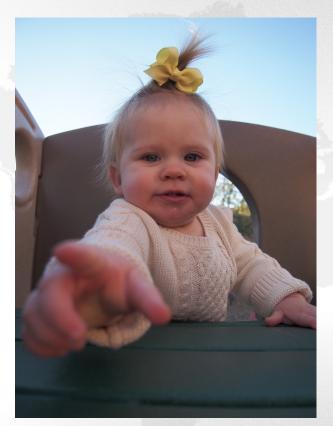
www.TheNationalWellnessFoundation.org

Dr. Thomas Tuzzolino, D.C

- Husband
- Father
- B.Sc AZ State University
- DC Southern California University of Health Sciences
- Pediatric & Family Chiropractor
- The National Wellness Foundation Member
 The National Wellness Foundation is a non-profit organization who's mission is to provide education, information and resources to raise Healthy Kids and Healthy Families



My why...







www.TheNationalWellnessFoundation.org

"It's not about what you take away from a child and a family, but what you give TO them!"

BUILDING HEALTH FROM WITHIN

The Plan

- Awareness
- Answers
- Action Steps



Awareness Test



Childhood Memories...

Don't Call Kids Names

Autism, ADHD, Sensory Processing Disorder, Asperger's, Oppositional Defiance, OCD, Anxiety, Bipolar, Allergies, Asthma

Don't Do Drugs

Ritalin, Strattera, Prozac, Concerta, Adderal, Focalin, Zoloft, Clonipine, Abilify, Phenobarbital, Keppra



Proper care requires proper understanding of the CAUSE

While modern medicine has gotten very good at "calling children names" (diagnosing), they have talked miserably at addressing, AND REMOVING, the true cause of childhood neurological disorders...In my opinion, no matter what "name" you call it, the cause is multi-factorial and often related...



What are we talking about?

ADHD/ADD

LEARNING & READING CHALLENGES

AUTISM

OCD, ODD, ETC...

SENSORY PROCESSING DISORDER



Physical Stress (Trauma)

- In-utero constraint
- Birth trauma
 - Forceps
 - Vacuum
 - Cord-wrapped
 - C-section (esp. emergency)
- Childhood falls
- Car seats, Baby Bjorn, Bouncy Seats..



"Funniest" Home Videos





Chemical Stress (Toxins)

- Preservatives, Pesticides, Processed Foods, Food Coloring, GMO
- Sugar, Grains, Dairy based diets (formula)
- Environmental chemicals EVERYWHERE
 - Household cleaners, detergents, plastic offgasing, etc.
- Vaccine ingredients, adjuvants, fillers
 - Mercury, aluminum, sorbitol, propylene glycol, egg & animal proteins, and much more...



Mental/Emotional Stress (Thoughts)

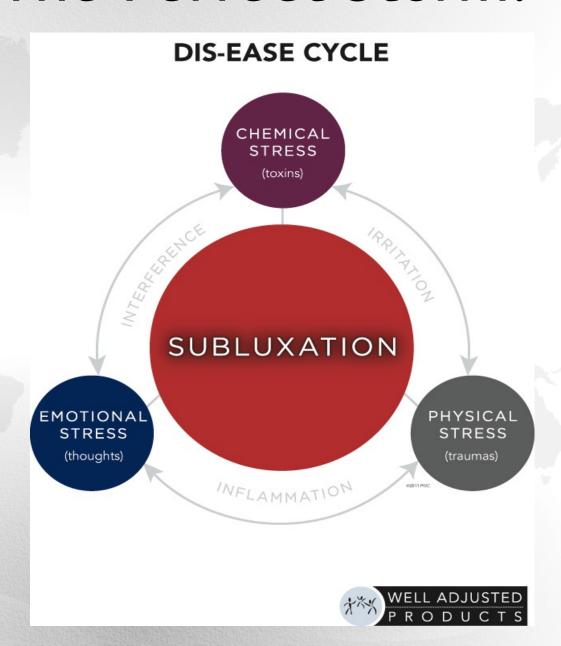
- Stressful, fear-based pregnancies
- Stressful, forced, emergency deliveries
- NICU stays
- Fast-paced, high stress life
- Getting labeled ("diagnosed")
- Bullying



What is The Perfect Storm?

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The New Normal... The Perfect Storm

FEAR & INTERVENTION BASED PREGNANCY

FEAR & INTERVENTION BASED DELIVERY

DIFFICULTY NURSING-FORMULA FED

COLIC (DIFFICULTY SLEEPING)

CHRONIC EAR INFECTIONS

ANTIBIOTICS

TOXIC FOOD & ENVIRONMENT

3-4X'S THE # OF VACCINES



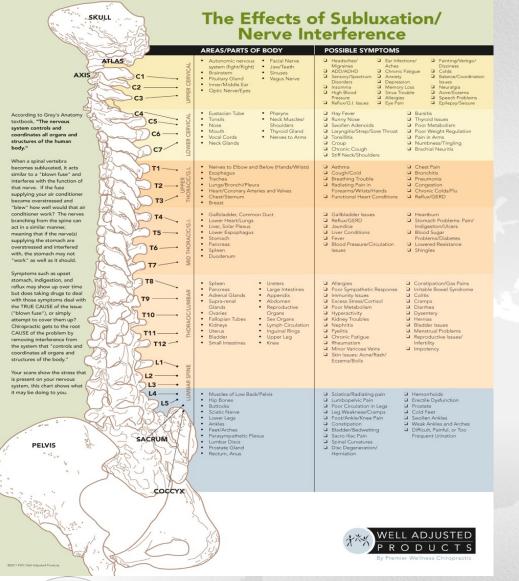
Understanding Chiropractic

Chiropractors are nerve doctors, not just back doctors...

"The function of the nervous system is to perceive the environment and coordinate the behavior of all other cells."

- Dr. Bruce Liption, PhD Biologist





balance is the key CENTRAL NERVOUS SYSTEM



SYMPATHETIC (GAS PEDAL)

- · Fight or flight response
- · Protection and survival
- · Stress response
- Adrenal (stress) glands activated

PARASYMPATHETIC (BRAKE PEDAL)

- Rest
- Digest Relax
- · Growth & development





"You can't be in growth and protection at the same time."

Dr. Bruce Lifton



SYMPATHETIC OVERDRIVE LEADS TO CHRONIC STRESS RESPONSE





WIDE WELLNESS

Subluxation: Cause and Effect (Nerd Time!)

THE CAUSE:

- 1. Physical Stress (Trauma)
- 2. Chemical Stress (Toxins)
- 3. Emotional Stress (Thoughts)



THE EFFECT:

- 1. Dys-kinesia
- 2. Dys-afferentation
- 3. Dys-autonomia
- 4. Dys-ponesis



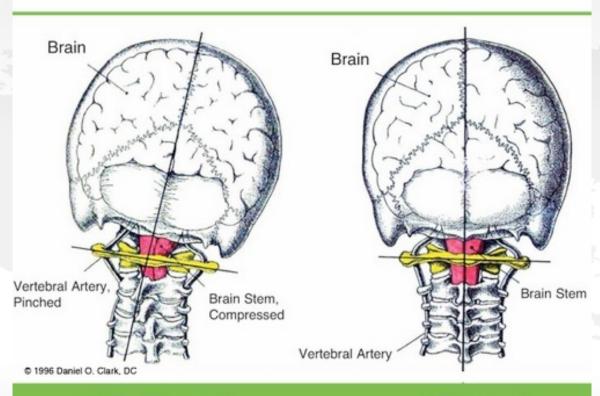
DYS-KINESIA

Subluxation occurs when a joint becomes MISALIGNED and FIXATED losing its ability to go through proper range of motion.





The "Main Breaker"



Misaligned

Aligned

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The Real Pandemic...

- In his research, Gottfried Guttman MD, found that spinal injury was present in 80% of the infants he examined shortly after birth, causing interference to neurologic and immune function.
- Concludes that "observations of motor development and manual control of the occipito-atlanto-axial joint complex should be obligatory after every difficult birth."
- Furthermore, this occipito-atlanto-axial joint complex "should be examined and, if required, specifically adjusted... (as) the success of adjustment overshadows every other type of treatment.

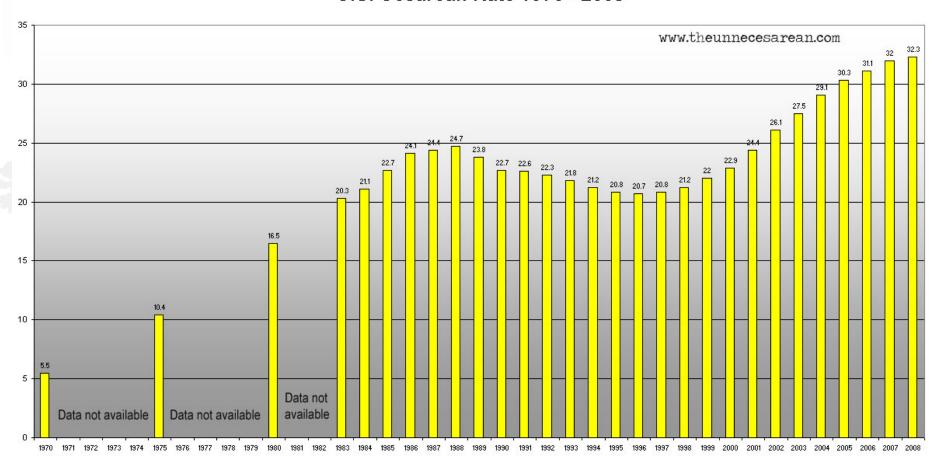


Where does it start?





U.S. Cesarean Rate 1970 - 2008



SOURCES: http://www.cdc.gov/nchs/data/nvsr/nvsr58/nvsr58_16.pdf http://www.cdc.gov/nchs/data/nvsr/nvsr54/nvsr54_04.pdf http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1647267/?page=2 http://www.cdc.gov/mmwr/preview/mmwrhtml/00036845.htm



The Cerebellum, Sensitive Periods, and Autism

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Cerebellar research has focused principally on adult motor function. However, the cerebellum also maintains abundant connections with nonmotor brain regions throughout postnatal life. Here we review evidence that the cerebellum may guide the maturation of remote nonmotor neural circuitry and influence cognitive development, with a focus on its relationship with autism. Specific cerebellar zones influence neocortical substrates for social interaction, and we propose that sensitive-period disruption of such internal brain communication can account for autism's key features.

In recent decades, much neuroscience research has focused narrowly on the cerebellum's role in balance, posture, and motor control. This framework has been explored in the greatest detail in cases where input pathways convey sensory information to the cerebellum, and outputs influence motor effectors. Emerging from this program is the view that the cerebellum acts as a processor that uses a variety of inputs to quide movement.

Receiving much less emphasis has been the role of the cerebellum in higher function. This idea is not new: cognitive to be cerebellum have been discussed since the total converse of the cerebellum have been discussed since the total cerebellum have been discussed since the cerebellum cerebellum and Wingeles 11. White a regreence interest in recent years a regree of the cerebellum cerebellum cerebellum discussed in the cognitive of the cerebellum congenitation of the cerebellum defects, where deficits are much more pronounced (Basson and Wingate, 2013; Steinling).

2013). Two facts have stood in the way of wider re nonmotor aspects of cerebellar function. First, nent deficits in acute cerebellar injury in adults a ture. Monitoring the short-term results of injury of long-term consequences that can accumulate consequences of cerebellar deficit are highly when the outcome is assessed. Second, cereb is highly differentiated, and focal injury typical deficits (Romaniella and Borgatti, 2012). While regions project predominantly to sensorimotor gous connections project to cognitive and a and comprise a large fraction of cerebellar cor et al., 2009). Recently, the extension of this parc to nonmotor brain structures has become clear methods (Buckner et al., 2011; Strick et al., 2009) cortex and nuclei have a distinctive circuit repeated in a modular fashion throughout the is highly conserved among vertebrates (App 2009). This has led to the proposal that the cere a common algorithm upon a variety of inputs, motor, cognitive, or affective.

In this Perspective, we outline a developme work for understanding the nonmotor roles of c riety of observations can be explained by the hypothesis: in addition to its role in the mature brain, the cerebellum acts in early life to shape the function of other brain regions, especially those relating to cognition and affect. We propose that the cerebellum takes an early role to processing external sensory and internally cone cast in a county in figurence neocortical circuit in the let during developmental sensitive per us. We in by each bing now new methods for large of manning at the processing neural circuits can be used except the complex role of the cerebellum in guiding nonmonance.

As part of this framework, we propose that cerebellar dysfunction may disrupt the maturation of distant neocortical circuits. To summarize the concept of developmental influence between brain regions, we use the term developmental diaschisis. Diaschisis (\di-as'-k-ses\; Gr. dia: across, schisis: break) is an existing neurological term indicating a sharp in-



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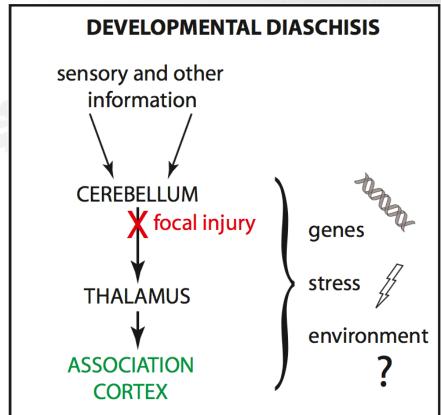
DIASCHISIS

(From Greek meaning "Shocked Throughout") is a sudden loss (or change) of function in a portion of the brain connected to a distant, but damaged, brain area. The site of the originally DAMAGED area and of the Diaschisis are connected to each other by neurons.

"DISTORTION & DISRUPTION" of "Brain and Body" communication and cross-talk



End Result: Brain (stem) Injury







DYS-AFFERENTATION

"NOISY BRAIN"

INPUT CONTROLS OUTPUT

- There are actually 7 senses!
- Taste, Touch, Smell, Hear, See....AND....
 - Movement
 - Proprioception (good)
 - Nociception (bad)
 - Balance/Coordination
 - Vestibular

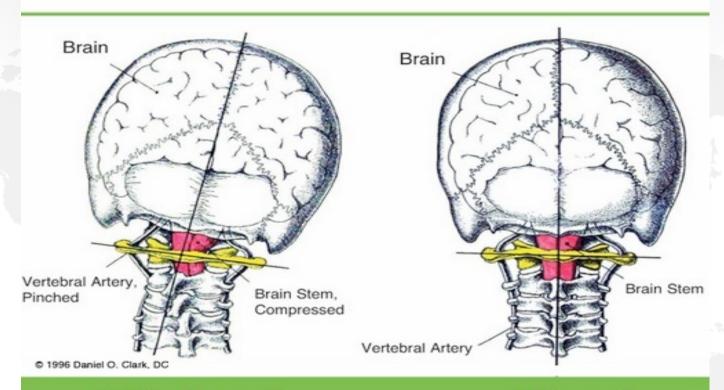


Proprioception-The Role Of The Spine...

- Neurological stimulation from MOVEMENT "charges the brain's batteries"
- Over half of the neurons in the spinal cord are there for this specific function
- 60% of proprioceptive input comes from spine
- 33% of it just from the upper neck / brainstem



Road Block? Communication is "jammed"



Misaligned

Aligned

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The Gate Theory

SUBLUXATION



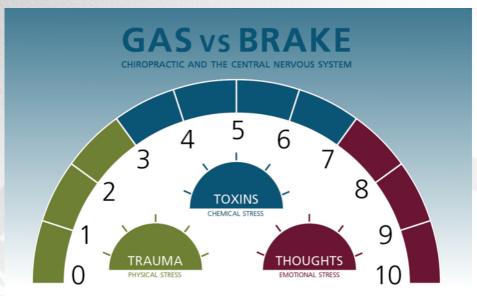
DECREASED PROPRIOCEPTION





"You can't be in growth (development) and protection at the same time."

-Dr. Bruce Lipton, PhD



Subluxation occurs when a misalignment and fixation within the spine creates stress and tension on the nerves. This stress causes the Central Nervous System to go into a protective state of fight or flight.

BRAKE PEDAL

PARASYMPATHETIC

- Chiropractic adjustment removes subluxation (stress)
- 2. Rest, relaxation, digestion, and immune function resume
- 3. Growth, healing, and development are restored

"Oh my gosh, he's been on the gas pedal his whole life."

"My daughter is so much more calm, connected, and happy than she's ever been."

"I've been stressed for so long, it's almost hard to describe how great I feel now!"

GAS PEDAL

SYMPATHETIC

- Misalignment, tension, and fixation occur within the spine (subluxation)
- 2. Stress response (adrenal glands) activated
- 3. Central Nervous System 'gets stuck' in fight or flight



"You can't be in growth and protection at the same time." - Dr. Bruce Lipton



DYS-AUTONOMIA

Leads to Dis-Organization & Dis-Regulation

Autonomic Nervous System



Ear Infections
Strep Throat
Eczema
Asthma
Allergies
Chronic Colds

Digestive System

Constipation
Colic
Acid Reflux
Cramping/Pain
Inflammation
IBS/Chrohn's

Majority of Serotonin

& Dopamine is made
in the gut

Endocrine System

CHEMICAL IMBALANCES???

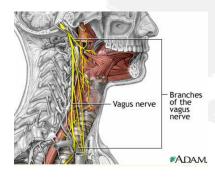
- I. Stimulus detected by N.S.
- -Sympathetic system engages
- -Directly influences visceral function
- -Adrenals produce stress hormones
- 2. Dysafferntation reaches CNS
- -Amygdala...stress/anxiety center
- -Hippocampus...learning center
- 3. Continues the cycle
- -HPA Axis

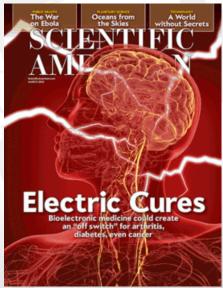
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The Vagus Nerve

Connects Gut & Brain Parasympathetic (Break Pedal) Modulates Inflammation







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The vagal innervation of the gut and immune homeostasis

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The central nervous system interacts dynamically with the immune system to modulate inflammation through humoral and neural pathways. Recently, in animal models of sepsis, the vagus nerve (VN) has been proposed to play a crucial role in the regulation of the immune response, also referred to as the cholinergic anti-inflammatory pathway. The VN, through release of acetylcholine, dampens immune cell activation by interacting with α -7 nicotinic acetylcholine receptors. Recent evidence suggests that the vagal innervation of the gastrointestinal tract also plays a major role controlling intestinal immune activation. Indeed, VN electrical stimulation potently reduces intestinal inflammation restoring intestinal homeostasis, whereas vagotomy has the reverse effect. In this review, we will discuss the current understanding concerning the mechanisms and effects involved in the cholinergic anti-inflammatory pathway in the gastrointestinal tract. Deeper investigation on this counter-regulatory neuroimmune mechanism will provide new insights in the cross-talk between the nervous and immune system leading to the identification of new therapeutic targets

INTRODUCTION

to treat intestinal immune disease.

Accumulating evidence supports the idea that an intricate communication network exists between the nervous and immune systems, and that this crosstalk could play a crucial role in the regulation of the immune response.1 The interplay between those diverse systems occurs through a complex set of neurotransmitters, cytokines and hormones that act as counter-regulatory mechanisms able to dampen inflammation and restore homeostasis.1 2 On a systemic level, neuroendocrine mechanisms reduce inflammation by the hypothalamic-pituitary-adrenal (HPA) axis through the anti-inflammatory effect of glucocorticoids, by the hypothalamic-pituitarygonadal axis through sex hormones, and by the hypothalamic-pituitary-thyroid hormone axis through thyroid hormones.3 4 Although modulation of the immune system by the nervous system, in particular the adrenergic nervous system, has been introduced decades ago,5 interest in the role of the autonomic nervous system as a key player in immune homeostasis has recently increased exponentially. In 2000, Tracey and coworkers demonstrated that vagus nerve (VN) stimulation potently suppresses cytokine production in a rodent model of sepsis.6 This discovery has led to the introduction of the concept of the cholinergic anti-inflammatory pathway,7 a hard-wired connection between the immune and nervous systems closely interacting to regulate inflammation. It is currently supposed that

inflammatory mediators activate sensory nerves and send signals concerning the state of the inflammation to the central nervous system. The latter, through efferent nerves, releases neuromediators that influence immune cells and modulates local inflammation.8 Consequently, it is now clear that the nervous system is able to regulate inflammation in peripheral tissues and to restore local immune

In the present review, the current knowledge and the clinical implication of the intestinal cholinergic anti-inflammatory pathway will be discussed. Readers interested in the sympathetic modulation of the immune response are referred to excellent reviews on this topic.9-11

THE CHOLINERGIC ANTI-INFLAMMATORY

While studying the anti-inflammatory effect of the

inhibitor of p38 MAP kinase, CNI-1493, it became clear that this compound suppressed carrageenaninduced paw oedema at doses at least 6-logs lower when injected intracerebroventricular than required for a systemic effect. 12 This potent anti-inflammatory effect was abrogated after bilateral vagotomy. Conversely, recording of the efferent VN electrical activity revealed an increase in discharge rate after infusion of CNI-1493, suggesting anti-inflammatory properties of VN activation. Similarly, electrical stimulation of the transected peripheral VN for 20 min prevented the development of an acute inflammation in response to carrageenan injection in the paw and increased survival in a model of sepsis6 by reducing cytokine (tumor necrosis factor (TNF)) production of splenic macrophages. This anti-inflammatory effect could be reproduced in vitro using isolated human macrophage cultures; the release of TNF, interleukin (IL)-1β, IL-6 and IL-18 in response to endotoxin was significantly reduced by acetylcholine (ACh) and nicotine. In a search to pharmacologically mimic the effect of VN stimulation. Wang et al identified the o7 subtype of the nicotinic acetylcholine receptor (a7nAChR) as the main receptor by which splenic macrophages are modulated. 13 The anti-inflammatory effect of VN stimulation is lost in α7nAChR knockout mice, can be blocked by specific antagonists a7nAChR, and is mimicked both in vivo and in vitro by α7nAChR agonists.13 Based on these findings, the 'cholinergic anti-inflammatory pathway' was introduced, whereby the VN modulates the immune response in the spleen providing an additional protective mechanism to the host (figure 1). This mechanism protects against the lethal effects of cytokines by restraining the magnitude of a potentially fatal peripheral immune response.6-1



To cite: Matteoli 6. Boedoestaens GE, GL 2013:62:1214-1222

1214

Matteoli G. et al. Gut 2013:62:1214-1222; doi:10.1136/gutjnl-2012-302550

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Cell Host & Microbe Perspective

Antibiotics, Pediatric Dysbiosis, and Disease

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Antibiotics are by far the most common medications prescribed for children. Recent epidemiological data suggests an association between early antibiotic use and disease phenotypes in adulthood. Antibiotic use during infancy induces imbalances in gut microbiota, called dysbiosis. The gut microbiome's responses to antibiotics and its potential link to disease development are especially complex to study in the changing infant gut. Here, we synthesize current knowledge linking antibiotics, dysbiosis, and disease and propose a framework for studying antibiotic-related dysbiosis in children. We recommend future studies into the microbiome-mediated effects of antibiotics focused on four types of dysbiosis: loss of keystone taxa, loss of diversity, shifts in metabolic capacity, and blooms of pathogens. Establishment of a large and diverse baseline cohort to define healthy infant microbiome development is essential to advancing diagnosis, interpretation, and eventual treatment of pediatric dysbiosis. This approach will also help provide evidence-based recommendations for antibiotic usage in infancy.

Introduction

Antibiotics are by far the most common prescription drugs given to children (Chai et al., 2012). Epidemiological studies have identified associations between antibiotic usage in early infancy and occurrence of diseases such as obesity, diabetes, and asthma in later life. Longitudinal studies of antibiotic usage have demonstrated profound short- and long-term effects of antibiotics on the diversity and composition of the gut microbiota. Finally, a large and growing number of studies implicate a causal role for microbiome imbalance (dysbiosis) in numerous diseases (Biedermann and Rogler, 2015). Understanding the short- and long-term effects of early life antibiotic use on the diversity and composition of the gut microbiota is critical in identifying the risks associated with the emerging prescription trends. However, the existing literature is limited in directly implicating microbial dysbiosis as the link between childhood antibiotics and development of disease in later life.

In this review, we synthesize numerous complementary sources, including microecological studies linking antibiotics and dysbiosis, mechanistic studies linking specific types of dysbiosis to specific disease outcomes, and reviews of epidemiological studies supporting antibiotics and increased disease risk. By this approach, we have identified four major types of antibiotics-related dysbiosis, and we have presented a framework for discussing and measuring pediatric dysbiosis in the context of several major diseases. Our analyses indicate substantial existing evidence for a number of causal mechanisms by which the microbiome mediates antibiotic-related disease risk.

Overuse of Antibiotics

The vast majority of antibiotic use occurs in the outpatient setting, where up to a third of prescriptions are unnecessary. In 2010, children received 74.5 million outpatient antibiotic pre-

scriptions—one for the US—accounting for or fourth of all medic s think the US—accounting for or

Numerous studies have demonstrated that antibiotics are often prescribed unnecessarily (Gonzales et al., 2001; McCaig et al., 2003; Nash et al., 2002), with estimates as high as 50% (Kronman et al., 2014). Nearly 30% of children receive an antibiotic prescription during an outpatient primary care visit (McCaig et al., 2003), most often inappropriately, for viral upper respiratory tract infections (Gonzales et al., 2001; Nash et al., 2002; Nyquist et al., 1998). Overuse of broad-spectrum antibiotics for conditions responsive to narrow-spectrum agents has been dramatically increasing (Hersh et al., 2013). Even after adjusting for differences in patient age, comorbidities, and sociodemographic factors, children with the same infections can receive vastly different rates of antibiotic prescriptions depending upon the practice or clinician visited (Fierro et al., 2014; Gerber et al., 2014), This phenomenon also seems to be universal; per capita antibiotic prescribing rates vary widely across US states (Hicks et al., 2013) and European countries (Goossens et al., 2005) without reasonable cause for geographic differences in bacterial infection rates.

In addition to the gut-microbiome-mediated effects as discussed in detail below, inappropriate prescribing of antibiotics can lead to both drug-related adverse effects and the promotion of antibiotic resistance. More than 140,000 emergency department (ED) visits occur annually in the US for antimicrobial-related adverse effects, comprising almost 20% of all ED visits for drugrelated adverse effects (Shehab et al., 2008). In addition to this direct patient harm, antibiotic use has been associated with the emergence of antimicrobial resistance, identified by the World Health Organization (WHO) as "one of the three greatest breats to human health." Importantly, a recent study found that the prevalence of antibiotic resistance genes in the infant gut microbiome increases with age, and infants born via C-section harbored a larger proportion of antibiotic resistance genes (blickhed et al., 2015). Infections with resistant bacteria increase morbidity and mortality, and grintly increase the cost of medical



WIDE WELLNESS www.TheNationalWellnessFoundation.org

A VISION OF WORLD

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DYS-PONESIS

Defined as a <u>reversible</u> physiological state consisting of unnoticed, misdirected neurophysical reactions to various agents (environmental events, bodily sensations, emotions, and thoughts) and the repercussions of these reactions throughout the organism.

ERRORS IN ENERGY OUTPUT

DISORGANIZED ENERGY

LOSS OF BALANCE





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I. Raging Bull Kid

Stuck on the "gas pedal" at all times (fight/flight) ADHD (hyper/behavior type), anxiety, etc.

2. Drunken Bull Kid

Brain is disorganized and disconnected Sensory Processing Disorder, ADD, balance/motor issues

3. Drunken Raging Bull Kid

Is both "stuck on" and disorganized Autism Spectrum Disorder, epilepsy



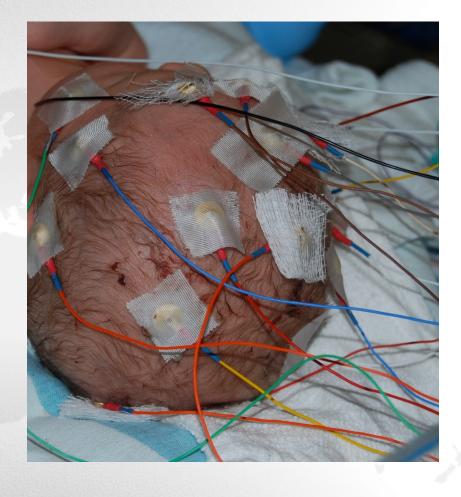
Autism: "Locked in" (Evan's Story)





Autism: Where It Startes...







Autism-The 4 D's

- Mostly non-verbal
- 3-5 words
- Unable to follow directions
- No eye contact, perception, etc.
- Very emotional, disruptive
- Horrible sleeper
- No interaction with parents and siblings
- Dx'd with "autism" in July
- Started care that day



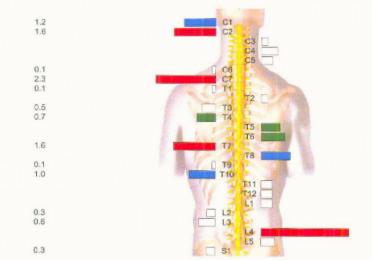
Patient Number: Patient Identifier:

62278968

Practice Address: 41

411 Congress Pkwy Suite C Crystal Lake, IL 600

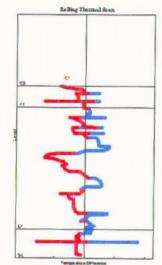
Rolling Thermal Scan NCM Bar Graph on (07/07/2011 11:16 AM) 6 degrees Farenheit



0.2 0.6 0.4 0.2 0.7 0.9 1.1 0.4 0.4 0.4

Rolling Thermal Scan NCM Line Graph on (07/07/2011 11:16 AM) 6 degrees Farenheit

6 degrees Farenheit



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Evan's Results:

After 2 Weeks:

- fell asleep in parking lot, 4PM-7AM
- began talking more within days
- imitating speech for the first time
- playing with his brother and sister and imitating their play
- doing funny things purposely to engage us
- pooping a ton detoxing!
- developed a fever -HEALING!

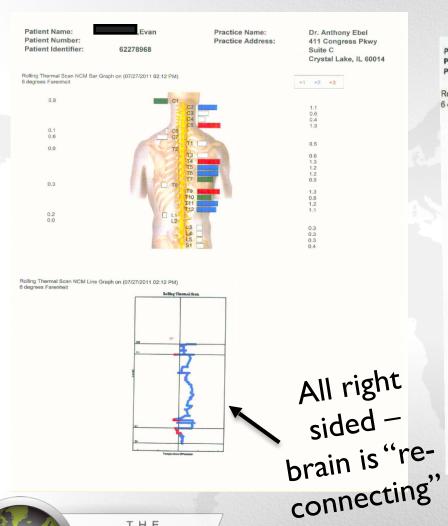
After 2 Months

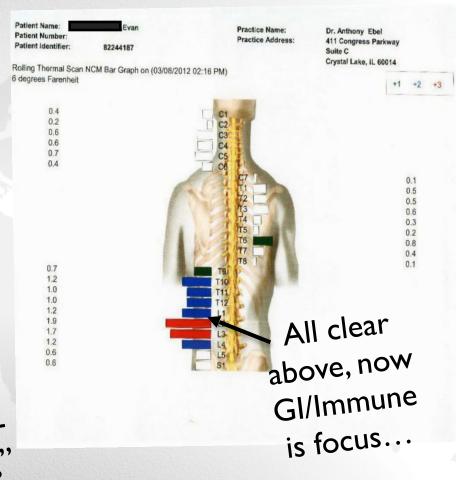
- speaking in full sentences
- exploding with development and speech
- sleeping better
- behaving better
- potty-trained
- never sick
- nearly back on track!!!

Autism Unlocked... (Evan After Just 2 months)



Autism Unlocked... 2nd and 3rd Scans







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After 10 Months...





There is Hope!



Tonights Opportunity

JUST GET CHECKED!

