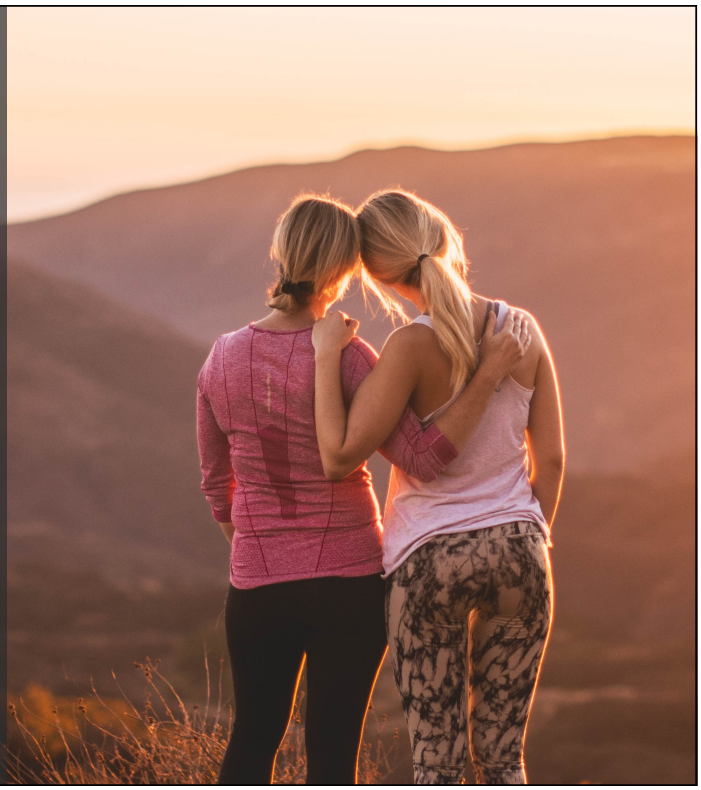
designs for health® Australia

Inflammation in Anxiety, ADHD and Autism

Dr Oscar Coetzee

Lea McIntyre ND



designs for health® Australia

Webinar Logistics

Participate | Download | Ask

DOWNLOAD WEBINAR PRESENTATION

Download Slides & Case Studies:

designsforhealth.com.au/references

ASK QUESTIONS

Use **Zoom Question Box** to ask questions

SPECIAL OFFER FOR ALL PARTICIPANTS

Check the Special Offer at the end of the webinar

DR OSCAR COETZEE

- **Associate Professor**
  Maryland University of Integrative Health
- **Associate Director D.Sc.**
  University of Bridgeport
- **Clinical Nutritionist**
  20 years clinical experience
- **Psychologist**
  Addiction/Sports
- **Advisory Board**
  Designs for Health

LEA McINTYRE ND

- **Customer Relations & Tech Manager**
  Designs for Health Australia
- **Masters Degree**
  Human Nutrition
- **Bachelors degree**
  Health Science
- **Qualified Naturopath, Herbalist & Nutritionist**
  19 years clinical experience
- **Special Interests**
  Paediatric Health & Gut Health

designs for health® Australia

Webinar Structure
IN THREE PARTS...

PART ONE
Overview of Factors + Relevant Tests + Rx + Case Study

PART TWO
Practitioner Questions

PART THREE
Resources | Ingredients | Protocols

Webinar Special Offer!

Download Slides & Case Studies:
designsforhealth.com.au/references

INFLAMMATION

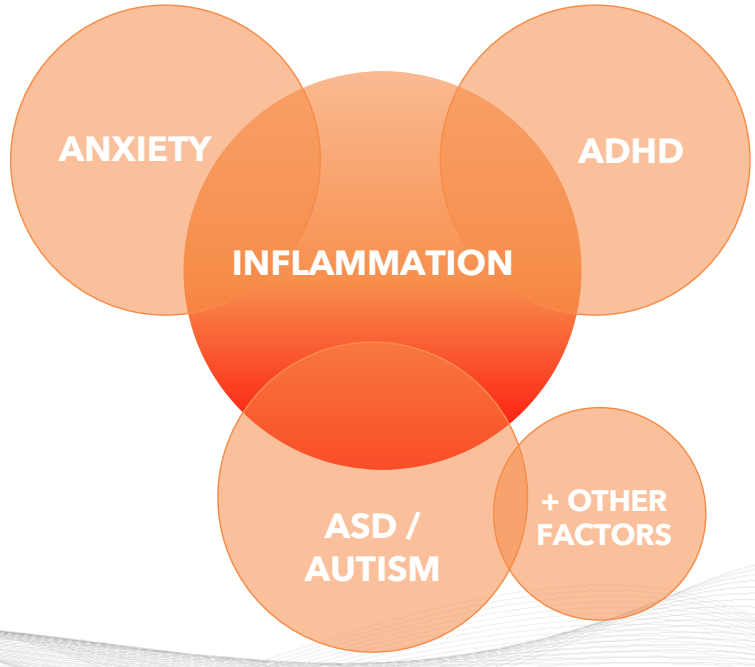
In Anxiety, ADHD and Autism

Root cause?

Side-effect?

Other factors?

Inflammation Correlations

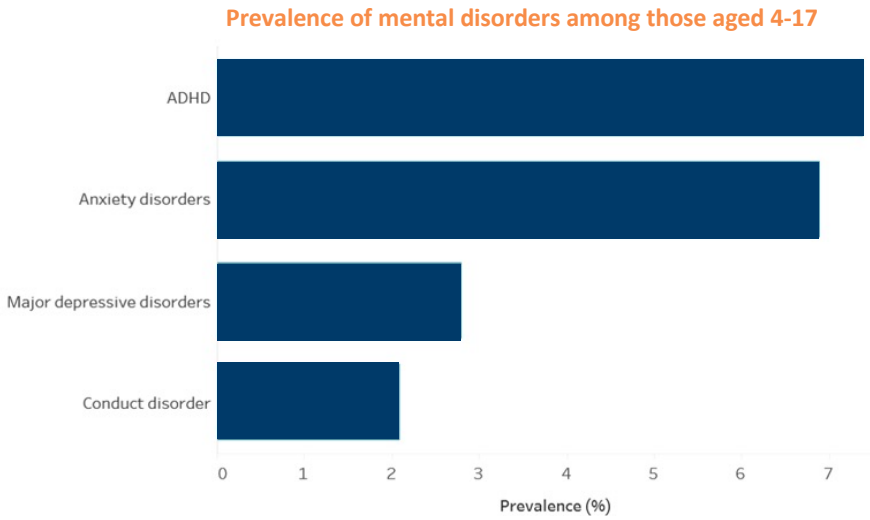


ASD - Epidemiology

- **1 in 160 people in developed countries estimated to have autism**
- **CDC estimates for 2018 are higher at 1 in 59 people** – 15% increase since 2002
- Both WHO and the CDC state that autism is on the rise
- **Boys four times more likely to be diagnosed with ASD than girls**; the gap may be narrowing, **11 boys to 1 girl with Asperger's**.
- **Autism is the fastest-growing serious developmental disability in the U.S.**

ADHD - Epidemiology

30.0% of 4–17 year old's in Australia had 2 or more mental disorders at some time in the previous 12 months.



https://www.aihw.gov.au/reports/mental-health-services/mental-health-services-in-australia/report-contents/summary/prevalence-and-policies

Anxiety - Epidemiology

"Anxiety: Australia's most common and most misunderstood illness"
One in seven Australians have an anxiety disorder

https://www.nps.org.au/professionals/anxiety

Common factors in Anxiety, ADHD & ASD

- Inflammation
- Gut health
- Metabolic function
- Psycho-Social
- Genetics
- Epigenetics
- Diet

BRAIN INFLAMMATION

Brain & Glucose (Dysglycemia)

REACTIVE HYPOGLYCEMIA

- Improper intake
- Less Substrate to process
- Reduces intra-neuronal pool to generate ATP

→ **LOW INTRANEURONAL GLUCOSE & ATP** ←

INSULIN RESISTANCE

- Proper/Excessive intake
- Abundant substrate availability
- Faulty insulin mechanism prevents access to neuron

GLYCEMIC CONTROL IS CRITICAL!

Brain Inflammation: Anaemia

IRON DEFICIENCY ANAEMIA

- Microcytic Hypochromic with low Ferritin
- Give 75 mg iron daily
- Identify and change mechanism

ANAEMIA OF INFLAMMATION

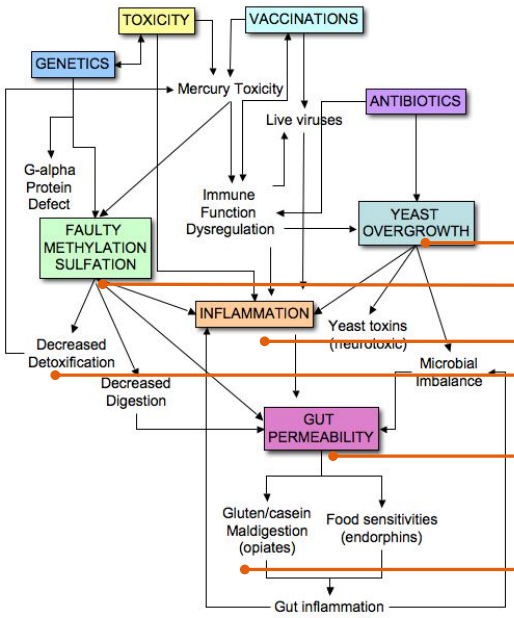
- Microcytic Hypochromic with elevated Ferritin
- Do NOT give iron!
- Use anti-inflammatory and anti-oxidative supplements
- Find and fix cause of inflammation

B12 ANAEMIA

- Megaloblastic Pattern
- Check Unsaturated B12 Capacity. Serum Methylmalonic Acid, Homocysteine
- Give B12 sublingual and/or injections
- Find and fix mechanism

Complex and Interrelated Whole Body Disorder

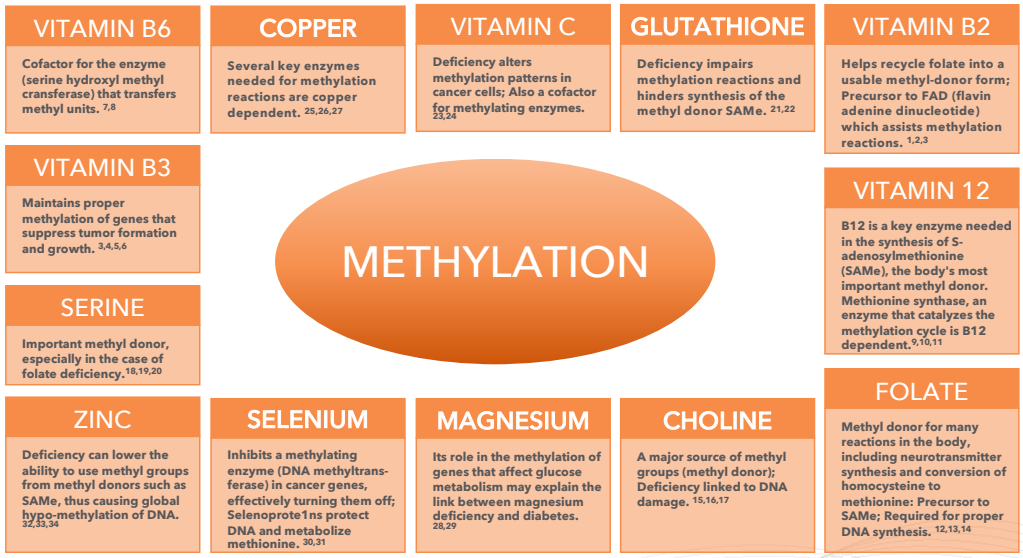
Inflammation cascade and its causes



Brain is Downstream

- **Yeast toxins**
- **Undermethylated neurotransmitters**
- **Brain inflammation**
- **Increased toxicity**
- **Nutrient deficiencies**
- **Opiates**

Methylation



AVAILABLE AS A DOWNLOADABLE RESOURCE

Vaccine Doses Australian Children

1960	1975	1996
Diphtheria	Diphtheria	Diphtheria
Tetanus	Tetanus	Tetanus
Pertussis	Pertussis	Pertussis
Polio (Salk IPV)	Diphtheria	Hib
Diphtheria	Tetanus	Polio (Oral Sabin)
Tetanus	Pertussis	Diphtheria
Pertussis	Diphtheria	Tetanus
Polio (Salk IPV)	Tetanus	Pertussis
Diphtheria	Pertussis	Hib
Tetanus	Polio (Oral Sabin)	Polio (Oral Sabin)
Pertussis	Polio (Oral Sabin)	Diphtheria
Polio (Salk IPV)	Polio (Oral Sabin)	Tetanus
Polio (Salk IPV)	Measles	Pertussis
13 Doses	Diphtheria	Hib
	Tetanus	Polio (Oral Sabin)
	Pertussis	Measles
	Diphtheria	Mumps
	Tetanus	Rubella
	Rubella (Girls Only)	Hib
	18 / 19 Doses	Diphtheria
		Tetanus
		Pertussis
		Diphtheria
		Tetanus
		Pertussis
		Polio (Oral Sabin)
		26 Doses

2018			
Antenatal	Diphtheria		
	Tetanus		
	Pertussis		
	Influenza		
Birth	Hepatitis B		
2 mths	Diphtheria	12 mths	Measles
	Tetanus		Mumps
	Pertussis		Rubella
	Hepatitis B		Pneumococcal 13
	Polio (IPV)		Meningococcal ACWY
	Hib	18 mths	Diphtheria
	Pneumococcal 13		Tetanus
	Rotavirus (Oral)		Pertussis
4 mths	Diphtheria		Hib
	Tetanus		Measles
	Pertussis		Mumps
	Hepatitis B		Rubella
	Polio (IPV)		Varicella
	Hib	4 yrs	Diphtheria
	Pneumococcal 13		Tetanus
	Rotavirus (Oral)		Pertussis
6 mths	Diphtheria		Polio
	Tetanus	10–15 yrs	Diphtheria
	Pertussis		Tetanus
	Hepatitis B		Pertussis
	Polio (IPV)		HPV
	Hib		HPV
49 Doses			

avn.org.au

Consider:
- Vaccines and inflammation
- Impact of toxic load

ASD – GI Associations:

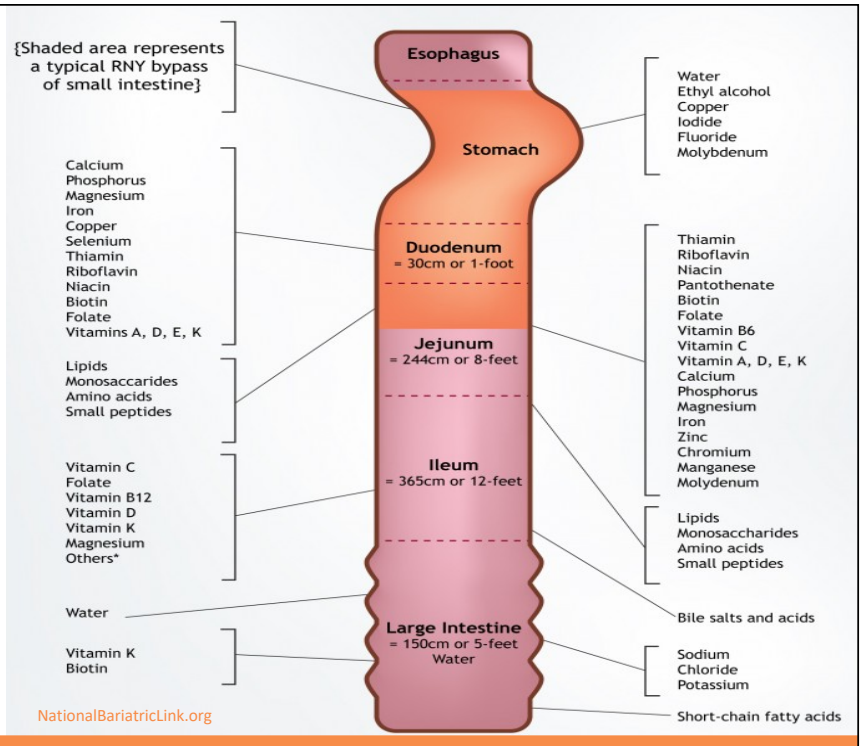
Endoscopic exam of 36 Autistics:

- Reflux 69.4%
- Gastritis 41.6%
- H. Pylori 0%
- **Duodenal Inflammation 66.6%**
  > Increased number of Paneth Cells 80.5%

(Anti-microbial compounds that are known to be important in immunity and host-defense, Paneth cells secrete some of these compounds into the lumen of the intestinal gland, thereby contributing to maintenance of the gastrointestinal barrier)

Nutrient Absorption

Consider:
- Key absorption areas
- Duodenal inflammation



GI-MAP™ Test

Quantitative PCR DNA stool test

- Function of the GI tract
- Level of beneficial bacteria
- Level of pathogenic bacteria
- Parasites, Worms, Viruses and Fungi

Reports include:

- GIT functional markers (e.g. calprotectin, Zonulin, Pancreatic elastase)
- How to know what's happening in the Gut
- Pointers for targeted treatment protocols

Gi-Map™ includes these Analytes:

- Digestion – Elastase 1, Steatocrit
- Immune Response - SIgA
- Inflammation – Calprotectin
- Zonulin-Leaky Gut marker
- Bacterial Pathogens
- Viral Pathogens

- Digestion – Elastase 1, Steatocrit
- Immune Response - SIgA
- Inflammation – Calprotectin
- Zonulin-Leaky Gut marker
- Bacterial Pathogens
- Viral Pathogens

ASD – GI Associations

SYMPTOMS OF VITAMIN B DEFICIENCY

- Fears
- Fatigue
- Depression
- Paranoia
- Confusion
- Hostility
- Rage
- Anxiety

SYMPTOMS OF NEUROPSYCHIATRIC DISORDERS

- Morbid fears
- Severe fatigue
- Depression
- Paranoia
- Confusion
- Anger
- Suicidal tendencies
- Anxiety

Fatty Acid Abnormalities in Children with Autism Parallel a Rodent Model of Autism

	Studies	Total N	Overall Prevalence
General ASD Population			
Mitochondrial disease in ASD	3	536	5.0% (3.2%, 6.9%)
Elevated lactate	6	479	31.1% (27.0%, 35.3%)
Elevated pyruvate	2	110	13.6% (7.2%, 20.1%)
Elevated lactate / pyruvate ratio	1	192	27.6% (21.2%, 33.9%)
Elevated alanine	1	36	8.3% (0.0%, 20.1%)
Low total carnitine	1	30	90.0% (81.0%, 99.0%)
Elevated creatine kinase	1	47	46.8% (32.4%, 61.2%)
Elevated ammonia	1	80	35.0% (24.5%, 45.5%)
Elevated AST	1	147	•45.6% (37.5%, 53.7%)[a]
Elevated ALT	1	87	7.0% (0.5%, 13.5%)

ORIGINAL ARTICLE:
Mitochondrial dysfunction in autism spectrum disorders: a systematic review and meta-analysis.

Molecular Psychiatry (2011), 1-25
© 2011 Macmillan Publishers Limited All rights reserved
1359-4184/11
www.nature.com/mp

ASD Related Research & References

Brain Sci 2019 Jun 13;9(6). Pii: E137. doi: 10.3390/brainsci9060137
First Case Report of Primary Carnitine Deficiency Manifested as Intellectual Disability and Autism Spectrum Disorder.
Guevara-Campos J[1]. Gonzalez-Guevara L[2]. Guevara-Gonzalez J[3]. Cauli O[4]

World J Biol Chem 2019 Jan 7;10(1):7-16 doi: 10.4331/wjbc.v10.i1.7.
Autism and carnitine: A possible link.
Demarquoy C[1] , Demarquoy J[2]

Bioessays 2017 Aug;39(8) doi: 10.102/bies.201700012.Epub 2017 Jul 13
Brain carnitine deficiency causes nonsyndromic autism with an extreme male bias: A hypothesis.
Beaudet AL[1]

Behav Brain Funct 2015 Feb 7;11:4 doi: 10.1186/s12993-014-0048-2.
Reduced levels of plasma polyunsaturated fatty acids and serum carnitine in autistic children: relation to gastrointestinal manifestations.
Mostafa GA, Al-Ayadhi LY.

Mol Psychiatry 2012 Mar. 17(3):290-314. dai: 10.1038/mp.2010.136. Epub 2011 Jan 25
Mitochondrial dysfunction in autism spectrum disorders: a systematic review and meta-analysis.
Rossignol DA[1] , Frye RE.

Altern Med Rev 2010 Sep; 15(3):187.
Testing the blood of autistic children for ammonia.
Good P.

Metab Brain Dis 2016 Aug;31(4):727-35 dai: 10.1007/s11011-016-9815-0. Epub 2016 mar 8.
Association of methylenetetrahydrofolate reductase (MTHFR) gene C677T polymorphism with autism: evidence of genetic susceptibility
Rossignol DA[1] , Frye RE.

Med Sci (Basel), 2018 Apr 4;6(2). Pii: E29. doi: 10.3390/medsci6020029
Clostridium Bacteria and Autism Spectrum Conditions: A Systematic Review and Hypothetical Contribution of Environmental Glyphosate Levels.
Argou-Cardozo I[1] . Zeidan-Chulia F[2,3]

Clin Nutr ESPEN 2018 Feb.23:171-183. doi: 10.106/j.clnesp.2017.10.005 Epub 2017 Dec 1.
Evidence the U.S. autism epidemic initiated by acetaminophen (Tylenol) is aggravated by oral antibiotic amoxicillin/clavulanate (Augmentin) and now exponentially by herbicide glyphosate (Roundup).

Neurotoxicology 2019 Aug 20;75:1-8. doi: 10.106/j.neuro.2019.08.006. [Epub ahead of print]
Gut microbiota and neurological effects of glyphosate.
RuedaRuzafa L[1] . Cruz F[2] . Roman P[3] . Cardona D[4] .

Integr Med (Encinitas), 2017 Feb 16; (1) 50 57.
Elevated Urinary Glyphosate and Clostridia Metabolites With Altered Dopamine Metabolism in Triplets With Autistic Spectrum Disorder or Suspected Seizure Disorder: A Case Study.

Surg Neurol Int 2015 Mar 24; 6.45 doi: 10.4103/2152-7806.153876. ecollection 2015
Glyphosate, pathways to modern diseases III: Manganese, neurological diseases, and associated pathologies.
Samsel A[1] . Seneff S[2] .

Organic Acids

FATTY ACID METABOLISM

- Adipate
- Suberate
- Ethylmalonate
- Consider value of Carnitine

BBB INTEGRITY

- CYREX Labs Array 20
- GABA Challenge

ENERGY PRODUCTION CAC

- α-Ketoglutarate
- Requires Vitamins B1, B2, B3, B5 & lipoic acid to function
- **Mineral factors:** magnesium, iron, and manganese
- Hydroxymethylglutarate (HMG) - precursor to CoQ10

B VITAMINS METHYLATION COFACTORS

- Vitamin B12
- Folic Acid
- Methylmalonate
- Formiminoglutamate

OXIDATIVE DAMAGE (ANTI-OXIDANT) STATUS

- P-Hydroxyphenyllactate
- Tyrosine degradation
- Vitamin C
- 8-OHdG
- Increased oxidative stress

TOXICANST AND DETOXIFICATION

- Xylene exposure
  - Glycine
- Orotate marker
  - Magnesium
- Glucarate
  - As biomarker
  - General detox

AVAILABLE AS A DOWNLOADABLE RESOURCE

FOOD TOXICITY & INFLAMMATION

Food is medicine

Chemical Contaminants in Milk

- Mycotoxins 1.2% (fungal colonisation of cow's foods)
- B1/M1aflotoxin resistant to pasteurization, discovered in 95.2% samples
- Pesticides, organochlorides, and PCBs 23.8%
- Antiparasitics 8.3% (macrolytic lactones)
- Antimicrobials 35.7%
- Carbamates 8.3%
- Organophosphates 14.3%
- Pyrethroids 8.3%

Bulletin of the International Dairy Federation. 465/**2013.**
Identification and Assessment of Emerging Issues Associated with Chemical Contaminants in Dairy Products
Maximum Residues Limits of some veterinary drugs in milk according to EU regulations.
European Commission, 1997

Pesticides in Food Supply

Canadian Food Inspection Agency. (2010-2011)
Pesticides in Coffee, Fruit Juice and Tea.

Samples: 297 coffee items, 510 juice items, and 267 tea items
Analyzed for over 430 pesticide residues

- 67% of juice not from concentrate had pesticide residues
- 48% of juice from concentrate had detected pesticides

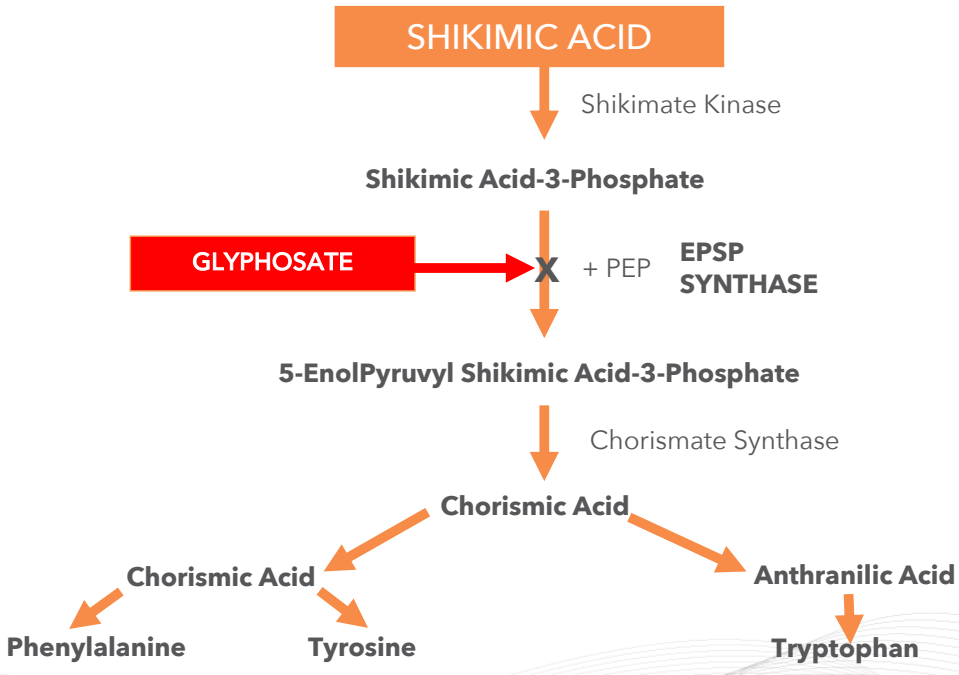
All types of tea samples had pesticide residues

- 66 tea samples containing at least one pesticide violation
- 75% of Oolong tea contained pesticide residue violations
- 50% of white tea
- 32% of green tea
- 20% of herbal and black tea

Glyphosate

- Glyphosate is the active ingredient in the herbicide Roundup.
- Broad-spectrum herbicide, considered to be nearly nontoxic to humans (Williams *et al.*, 2000).
- Glyphosate suppresses 5-enolpyruvylshikimic acid-3-phosphate synthase (EPSP synthase), the rate-limiting step in the synthesis of the aromatic amino acids, tryptophan, tyrosine, and phenylalanine, in the shikimate pathway of bacteria and plants (de Maria *et al.*, 1996).
- In plants, aromatic amino acids collectively represent up to 35% of the plant dry mass (Franz, 1997).

Glyphosate



Glyphosate

"Disruption of mucosal folds and disarray of microvilli structure" in the intestinal wall, along with an exaggerated secretion of mucin throughout the alimentary tract.

These features are highly reminiscent of celiac disease.

(Senapati *et al.*, 2009)

Glyphosate

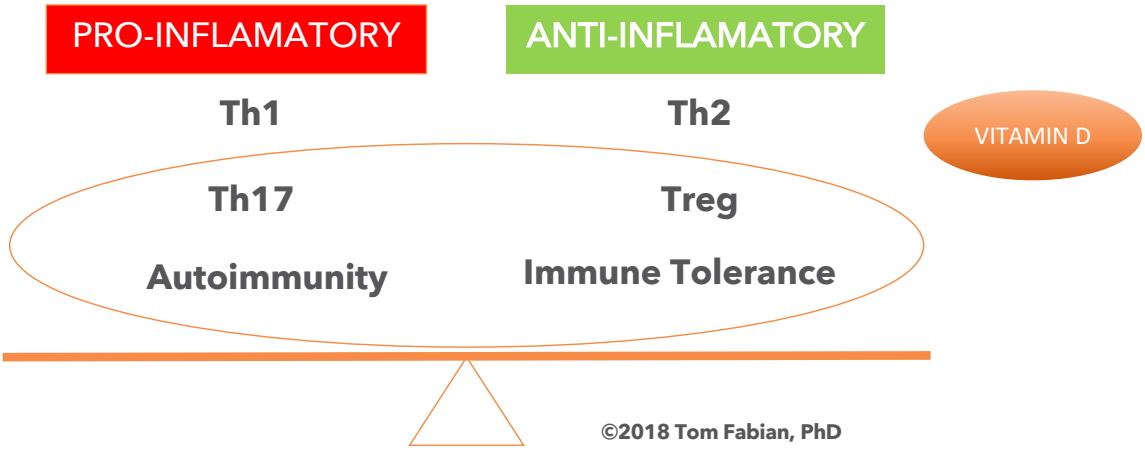
Glyphosate's Suppression of Cytochrome Enzymes and Amino Acid Biosynthesis by the Gut Microbiome: Pathway to Modern Diseases.
By Anthony Samsel and Stephanie Seneff
Entropy 2013, *15*(4), 1416-1463

Interference with CYP enzymes
- depletion of vitamin D3, due to impairments in the CYP enzyme responsible for its activation

Disruption of the biosynthesis of aromatic amino acids in gut bacteria
- depletion of serotonin through dysbiosis of its substrate, tryptophan
- depletion of dopamine through impaired synthesis of its substrate, tyrosine

Impairment in serum sulfate transport
- depletion of sulfate through interference with its synthesis
  - **induction of excess synthesis of ammonia in the gut**
  - **depletion of zinc through impaired absorption**

www.mdpi.com/1099-4300/15/4/1416/pdf



©2018 Tom Fabian, PhD

Other considerations for ASD

Autism, Gut Bugs and Fecal Transplant

Microbiome

RESEARCH Open Access

Microbiota Transfer Therapy alters gut ecosystem and improves gastrointestinal and autism symptoms: an open-label study

Conclusions

Together, these findings suggest that MTT is safe and well-tolerated in children with ASD ages 7–16 years. MTT led to significant improvements in both GI- and ASD-related symptoms, and the improvements were sustained at least 8 weeks after treatment. Coincident with these clinical improvements, both microbiota and phage from the donors appear to have engrafted, at least partially, in the recipients. This shifted gut microbiota of children with ASD toward that of neurotypical children is consistent with the hypothesis that gut microbiota may be at least partially responsible for GI and ASD symptoms. While this study was an open-label trial that is subject to placebo effects, these results are promising and provide a crucial step for understanding the connection between the microbiome and ASD. A randomized, double-blind, placebo-controlled study is the next step to investigate the value of MTT in treating children with ASD and GI problems.

Kang *et al. Microbiome* (2017) 5:10

Other considerations for ASD

Novel role of the vitamin D receptor in maintaining the integrity of the intestinal mucosal barrier

Juan Kong,[1] Zhongxi Zhang,[1] Mark W. Musch,[1] Gang Ning,[2] Jun Sun,[1] John Hart,[3] Marc Bissonnette,[1] and Yan Chun Li[1]

[1]Department of "Medicine and "Pathology, The University of Chicago, Chicago, Illinois; [2]The Huck Institutes for Life Sciences, The Pennsylvania State University, University Park, Pennsylvania; and [3]Gastroenterology and Hepatology Division, Department of Medicine, University of Rochester Medical Center, Rochester, New York

Submitted 31 August 2007; accepted in final form 23 October 2007

Kong J, Zhang Z, Musch MW, Ning G, Sun J, Hart J, Bissonnette M, Li YC. Novel role of the vitamin D receptor in maintaining the integrity of the intestinal mucosal barrier. *Am J Physiol Gastrointest Liver Physiol* 294: G208–G216, 2008. First published October 25, 2007; doi:10.1152/ajpgi.00394.2007.—Emerging evidence supports a pathological link between vitamin D deficiency and the risk of inflammatory bowel disease (IBD). To explore the mechanism we used the dextran sulfate sodium (DSS)-induced colitis model to investigate the role of the vitamin D receptor (VDR) in ensuring barrier homeostasis. While $VDR^{+/+}$ mice were mainly resistant to 2.8% DSS, $VDR^{-/-}$ mice developed severe diarrhea, rectal bleeding, and marked body weight loss, leading to death in 2 wk. Histological examination revealed extensive ulceration and impaired wound healing in the colonic epithelium of DSS-treated $VDR^{-/-}$ mice. Severe ulceration in $VDR^{-/-}$ mice was preceded by a greater loss of intestinal transepithelial electric resistance (TER) compared with $VDR^{+/+}$ mice. Confocal and electron microscopy (EM) revealed severe disruption in epithelial junctions in $VDR^{-/-}$ mice after 5-day DSS treatment. Therefore, $VDR^{-/-}$ mice were much more susceptible to DSS-induced mucosal injury than $VDR^{+/+}$ mice. In cell cultures, 1,25-dihydroxy-vitamin D_3 [1,25(OH)$_2$D$_3$] markedly enhanced tight junctions formed by Caco-2 monolayers by increasing junction protein expression and TER and preserved the structural integrity of tight junctions in the presence of DSS. VDR knockdown with small interfering (si)RNA reduced the junction proteins and TER in Caco-2 monolayers. 1,25(OH)$_2$D$_3$ can also stimulate epithelial cell migration in vitro. These observations suggest that VDR plays a critical role in mucosal barrier homeostasis by preserving the integrity of junction complexes and the healing capacity of the colonic epithelium. Therefore, vitamin D deficiency may compromise the mucosal barrier, leading to increased susceptibility to mucosal damage and increased risk of IBD.

tight junction; inflammatory bowel disease; dextran sulfate sodium

THE INTESTINAL EPITHELIAL barrier consists of epithelial cells and the intercellular junctions. The barrier regulates macromolecule trafficking between the lumen and the internal milieu and protects the host by preventing harmful solutes, microorganisms, toxins, and luminal antigens from entering the body (30). Compromise or disruption of the intestinal barrier function causes deleterious effects and results in exposure of the host to luminal antigens and bacteria, leading to inflammation. Impaired barrier functions have been described in a number of common gastrointestinal disorders, including inflammatory bowel disease (IBD) (7).

The integrity of the intestinal mucosal barrier is preserved by the enormous regenerating capacity of the mucosal epithelium. The intestinal stem cells, located at the base of the crypt, are responsible for replenishing the epithelium through cell division and differentiation. After extensive destruction, rapid resealing of the surface epithelium is accomplished by epithelial cell restitution, proliferation, and differentiation (6). Another important component of the mucosal barrier is the apical and subapical intercellular junctions between the epithelial cells, namely tight junctions and adherens junctions (18). These junction structures seal the paracellular space and regulate the permeability of the mucosal barrier.

IBD, including Crohn's disease and ulcerative colitis, is a major chronic disorder affecting the gastrointestinal tract in humans. Although the etiopathogenesis of IBD has not been clearly elucidated, it is thought to involve a complex interplay among genetic, environmental, microbial, and immune factors (33). One potential pathogenic factor is impaired mucosal barrier function, and intestinal hyperpermeability is common in IBD patients (11). A relatively high number of first degree relatives of patients with Crohn's disease have increased intestinal permeability in the absence of clinical symptoms (32, 42), suggesting barrier dysfunction precedes, or is at least a very early defect, in the disease process that might require genetic predisposition and environmental triggers. Indeed, previous studies have demonstrated decreased expression and differential localization of junction complex proteins in the mucosa of patients with IBD (10, 18, 29). Therefore, dysregulation of junction proteins is an important pathogenic mechanism underlying the increased permeability seen in the intestinal epithelium of IBD patients.

Previous studies have suggested a link between vitamin D deficiency and IBD risk (25). The prevalence of IBD exhibits a north-south gradient (24), paralleling sunlight exposure, an important source of vitamin D. Populations near the equator are at relatively lower risk for developing IBD. Seasonal variations in the onset and exacerbation of IBD have also been reported (27, 36) with high incidence in the winter. Early studies have reported a high prevalence of vitamin D deficiency in patients with established Crohn's disease (12, 36). Decreased vitamin D levels have also been detected in patients with newly diagnosed IBD (17, 19, 35). In the IL-10 $^{-/-}$ mouse model of intestinal inflammation, vitamin D deficiency or vitamin D receptor (VDR) deficiency exacerbates the symp-

Vitamin D and the Gut

In vitro experiments demonstrate that VDR mediates the activity of 1,25(OH)2D3 that induces junction protein expression and strengthens the tight junction complex. These data are consistent with, and explain at least in part, the observation reported in the literature that vitamin D deficiency is linked to increased incidence of IBD in human population.

Upcoming DFH Australia & New Zealand Tour

ADDRESSING INFLAMMATORY FACTORS

Clinical Considerations and Treatments

Clinical Considerations for Anxiety, ADHD, ASD

- Check for BBB integrity
- Find and fix inflammatory mechanisms:

Gut:

- Permeability and/or infections and/or food reactivity

Inflammation associated with:

- Dysglycemias
- Poor detox and high environmental loads
- Hormones
- Chronic stress etc.

Treatment Considerations
Inflammation associated with Anxiety, ADHD, ASD

- Bioflavonoids and polyphenols (wide range)
- **Turmeric / Curcumin**
- **Resveratrol**
- Ginger
- Green and White Tea Extracts
- Baicalein
- And many more...

Diet options to choose from:

ASD Diet Options	***ARI Survey Results (ASD)*** *parents' reporting symptomatic improvement*
GFCF (Gluten-free and Casein-free) No gluten (wheat, rye, barley, spelt, kamut, and oats) or casein (dairy)	GFCF - 65% improved No Dairy - 50% improved No Wheat - 49% improved
Food Sensitivity Elimination Eliminating all other food sensitivities: Soy, corn, eggs, citrus, peanuts, chocolate, cane sugar	No Eggs – 49% improved No Chocolate – 49% improved No Sugar – 48% improved Rotation Diet – 49% improved
Feingold Diet/Low Phenols Restricts high phenolic foods, including all artificial ingredients and high salicylate fruits	54% - improved
SCD (Specific Carbohydrate Diet) Restricts carbohydrates to only fruits, non-starchy vegetables, and honey. No grains, starchy vegetables, or mucilaginous fibers	SCD - 66% improved Candida Diet – 54% improved
Body Ecology Diet Anti-yeast diet combining principles of anti-yeast diets including no sugar, acid/alkaline, fermented foods	
Nourishing Traditions/ Weston A. Price Good quality fats, soaking and fermenting for digestion	
Low Oxalate Diet Restricts high oxalate foods (nuts, beans, greens)	

Dietary Cautions and Swaps

THE DANGER LIST		
Additive	**Where Used**	**Potential Problems**
E102 – tartrazine	Sweets, biscuits, mushy peas	Hyperactivity, asthma, rashes
E124 – ponceau 4R	Sweets, biscuits, drinks	Allergy, intolerance
E110 – sunset yellow	Sweets, ice cream, drinks	Gastric upset, allergy
E122 – camoisine	Biscuits, jelly, sweets, ready meals	Allergy, intolerance
E104 – quinoline yellow	Sweets, smoked haddock, pickles	Hyperactivity, asthma, rashes.
E129 – allura red	Soft drinks, cocktail sausages	Some evidence of hypersensitivity
E211 – sodium benzoate	Soft drinks, baked goods, lollies	Hyperactivity; asthma

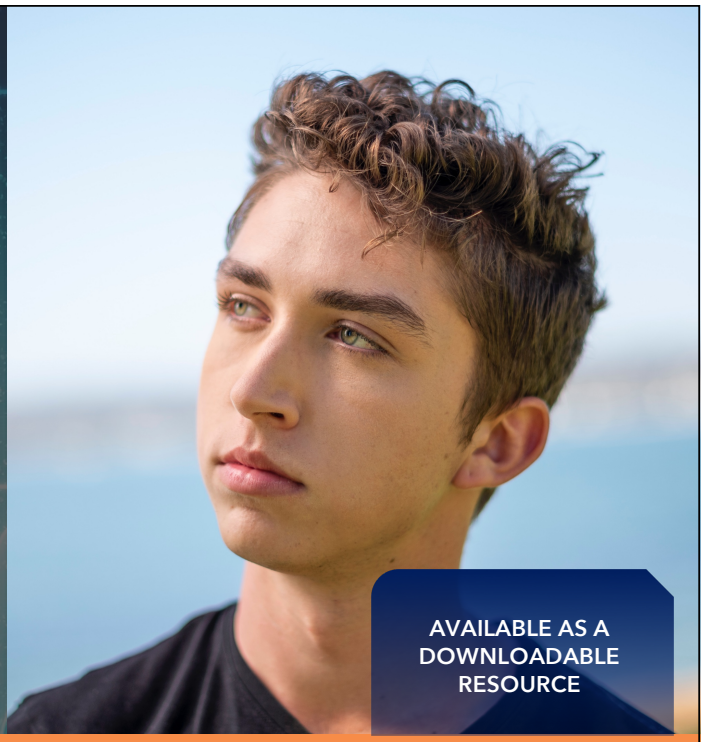
Toxins Around the Kitchen	Safer Cooking Alternatives
Avoid aluminum cans	Buy in glass
Avoid storing in plastic	Store in glass w/metal or plastic lid
Avoid Teflon, copper, and aluminum pans	Use stainless steel (attracts a magnet), cast iron or enameled cast iron
Avoid the microwave, do not reheat in plastic	Heat in oven or on stove
Avoid plastic wrap & aluminum foil	Use wax paper or glass with lid

CASE STUDY

Clinical Considerations and Treatments

OVERVIEW

- Andrew
- Male
- Age 12 Years



AVAILABLE AS A DOWNLOADABLE RESOURCE

designs for health® Australia

Case Study

Andrew | Male | Age 12 Years

- Andrew presented with ASD, Environmental allergies, asthma, insomnia, migraines, dysgraphia, muscle cramps, stomach pain, nausea and diarrhea. He was first diagnosed with ASD/ADHD at 5 years of age. He repeated kindergarten because of attention and behavioral difficulties. He began methylphenidate (Ritalin) in first grade and continued through fourth grade, although parents indicated that it didn't change his behavior significantly. In 4th grade the drug was discontinued due to growth retardation. Off the medication, he put on weight but also started to increase junk food and deli meats.
- Andrew has never received a positive teachers report. Off the Ritalin he continued to be disruptive and unfocussed.
- Andrew's allergy symptoms were sinus congestion, and post nasal drip, sore throats and hives for which he took Zyrtec. His mom removed the carpets from his room and installed hardwood floors, but this had minimal improvement. His mom noticed symptoms worsen with dairy. He would end up with sinusitis 3-4 times a year for which he was prescribed antibiotics.

designs for health® Australia

Case Study

Andrew | Male | Age 12 Years

- As a toddler, Andrew developed asthma, hyperkeratosis pilaris and eczema. For the asthma he was maintained on Albuterol, Levalbuteral (Xopenex) and Flunisolide (AeroBid). The asthma made breathing and sleeping at night difficult.
- He also suffered with cold induced asthma attacks. His skin and anus were frequently itchy, and he got frequent canker sores in his mouth.
- Andrew suffered from migraine headaches with hypersentivity to light and sound, and had muscle cramps and spasms for which he was given acetaminophen and ibuprofen.
- His stomach pain was treated with cimetidine. He also complained of nausea and had frequent bouts of diarrhea.

designs for health® Australia

Case Study

Andrew | Male | Age 12 Years

LABORATORY DATA
RBC minerals

Zinc: Low 2.5 ppm

Mg: Low 15 ppm

Cu: Low normal 260 ppb

Mn: low normal 22 ppb

Chromium: Low normal- 1.7 ppb

Vitamin E: Low normal 7.2 mg/L

Vitamin A: Low normal .55 mg/L

Beta-carotene: Low <0.2 mg/L

Serum D 25OH: Low 24 ng/m

Fatty Acids

Trans: High 215 µM

Arachadonic acid: High 785 µM

IgG antigliadin:
High normal 17 units

Urine toxic metals:
Lead- High 17 µg/g creatinine

Plasma Amino Acids:
Tryptophan- Low 30 µmol/L

Commentary

Therapeutic considerations

PRACTITIONER QUESTIONS

Live Webinar and Pre-submitted Questions

Practitioner Questions 1

- How much of ADHD is Genetic vs Environmental?
- How do you recommend getting supplements into ASD kids who have a limited diet and who are particularly sensory?
- Can you offer any guidance/precautions to be aware of when co-prescribing herbs and nutrients with clients' existing medications?

Practitioner Questions 2

- Recommendations on the preferred type of exercise to help with managing ADHD?
- Would you suggest other mental health conditions such as depression, PTSD, chronic headaches have a similar aetiology to those focused on today?

  Do you suggest a similar treatment plan for these conditions?

Practitioner Questions – Live Webinar

DR OSCAR COETZEE

LEA McINTYRE ND

B VITAMINS

- Specifically, B2, B3, Folate B12 for methylation
- B12 for B12 anaemia
- B1,B2,B3.B5 for energy support

VITAMIN D

- Depleted in Glyphosate exposure
- Anti-inflammatory action

CURCUMIN

- Anti-oxidant support
- Anti-inflammatory

GLUTATHIONE

- Involved in methylation
- Antioxidant support
- Detoxification support

DIGESTIVE ENZYMES

- Reduces exorphins and dietary peptides the interfere with cellular process
- Reduces complex carbs/sugars that lead to dysbiosis
- Improving nutrient supply
- Improving fat digestion - ASD kids often have poor fat digestion

UBIQUINOL

- Energy support
- Mitochondria Support

VITAMIN C

- Anti-oxidant support
- Required for methylation
- Required for Adrenal function

designs for health®

INGREDIENTS TO CONSIDER

MAGNESIUM

- Supporting healthy glucose metabolism
- Nervous system support
- Required for glutathione synthesis

BERBERINE

- Supporting healthy Glucose Metabolism
- Offering Antimicrobial support

PROBIOTICS

- Maintaining gut homeostasis
- Offering antimicrobial protection
- Maintaining mucosal integrity

SACCHAROMYCES

- Offering anti-microbial support
- Supporting intestinal immunity

OMEGA 3 FATTY ACIDS

- Commonly low levels in ASD kids
- Anti-inflammatory support
- Cell Membrane support
- Look for high DHA content

designs for health® Australia

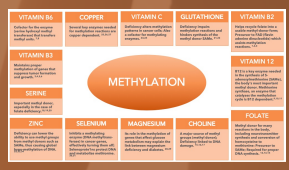
Resources & Downloads

PRESENTATION SLIDES

ORGANIC ACIDS REFERENCE

CASE STUDY

METHYLATION CHART



CERTIFICATE OF ATTENDANCE

AVAILABLE AS DOWNLOADABLE RESOURCES

designs for health® Australia

Download here: designsforhealth.com.au/references

Upcoming DFH Australia & New Zealand Tour

Register NOW
Places filling fast

designs for health®

Metabolic Syndrome Masterclass

with Dr Oscar Coetzee

OCTOBER 2019

Auckland - Brisbane
Melbourne - Perth
Sydney

Register at:
designsforhealth.com.au/masterclass

designs for health® Australia

WEBINAR: Inflammation in Anxiety, ADHD & Autism

MetS Masterclass

Register NOW
Limited places left

Learn Strategies to Prevent & Treat the Metabolic Syndrome Epidemic

Metabolic syndrome (MetS) is increasingly common in Australia and worldwide. This increase is associated with the global epidemic of obesity, diabetes and cardiovascular disease.

With the elevated risk there is an urgent need for clinicians to understand relevant strategies to prevent and treat this increasingly common cluster of conditions.

Metabolic syndrome can be a master of disguise and can occur with symptoms of:

- **Glucose intolerance**
- **Central obesity**
- **Lipid abnormalities**
- **Hypertension**
- **Insulin resistance associated with excess abdominal weight**
- **Leptin Resistance**
- **Lipoedema**
- **Uric Acid**
- **Anthropometric measurements**
- **Fatty Liver**
- **Conditions such as PCOS and sleep apnoea can also be associated**

designs for health®

designsforhealth.com.au