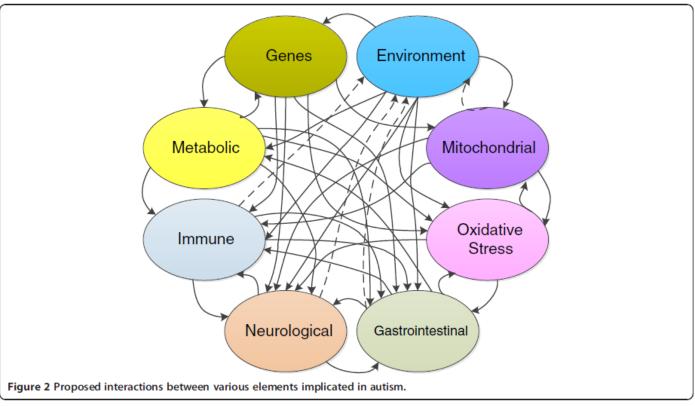
A Multiple System Approach to Autism Rethinking how we treat autism.

Autism is defined and diagnosed solely on the basis of symptoms. If a child's symptoms meet enough of these criteria (as defined by DSM-IV/ DSM-5), a diagnosis of autism is given. What has to be understood though is that these "autistic" behaviours are most often merely surface manifestations of underlying biomedical problems.

Studies and clinical experience have shown that many children on the autistic spectrum have a range of health problems, often concerning the gut and immune system, but involving other areas and body systems as well. Many children endure ongoing problems that include constipation or impaction, irritable or painful bowel and other problems related to poor gut function; impaired blood flow to the brain (confirmed in autism through SPECT scans); abnormal glucose metabolism; low cellular energy production, with mitochondrial abnormalities having been confirmed in a large proportion of children and adults with autism; high levels of bacterial toxins circulating the blood (which correlate to severity of autism); impairments/delays in sensory processing; and inability to properly digest food, which results in reduction of available nutrients. Each of these conditions can affect cognitive and emotional function—imagine the children who suffer multiple health problems.



Source: Randolph-Gips and Srinivasan. Modeling autism: a systems biology approach. Journal of Clinical Bioinformatics 2012, 2:17.

There is no ASD without a co-morbid condition!

Changing the way we think about treating autism.

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Helping Children Achieve Their Full Potential

Summary of abnormal biomedical findings in autism:

generalised immune dysfunction and inflammation including microgliosis and astrogliosis (inflammation of brain microglia and astrocytes) and raised inflammation in the CSF, vascular endothelial inflammation, abnormal vasoconstriction and permeability, reduced blood flow to brain, oxidative stress, systemic glutathione depletion, mitochondrial dysfunction, autoimmune reactivity, mast cell activation, cardiovascular abnormalities (raised median diastolic blood pressure, abnormal QRS complex), increased intestinal permeability, microbial translocation (presence of bacterial toxins in the blood), hyperplasia of intestinal epithelial cells, pancreatic enzyme deficiency, disaccharide intolerance and malabsorption, autonomic/vagal stem dysfunction, abnormal cytokine profiles, antibodies to folate receptors, increased presence of polyomaviruses in the brain, increased bacterial and viral infections, abnormal gene methylation, cerebral folate deficiency.

What is becoming apparent is that although children with autism share symptoms that define the diagnosis they may have arrived there by a great many different paths. Therefore this would suggest that autism needs to have a multidisciplinary approach to treatment. The best approach at the moment is to get a clearer idea of different 'subgroups' of autism, and to get clear biomarkers for those subgroups.

We need to identify subsets of children for which we can offer effective treatments.

With the current research studies available we have clues as to what testing we should be doing on autistic children to identify these "subgroups" of children with medical issues that can be treated successfully. Addressing identified health needs often has positive effects on what are perceived to be "autistic" behaviours and symptoms.

Autism is a complex medical disorder, likely of many etiologies. It's a simple concept to grasp, but one that medical professionals, researchers, and government officials have been **incomprehensibly and inexcusably slow to embrace**.

References

Neuroglial activation and neuroinflammation in the brain of patients with autism. Vargas DL et al. (2005). Ann Neurol. 57(1):67-81.

Elevated immune response in the brain of autistic patients. Li X et al. (Feb 2009) J Neuroimmunol. 207(1-2):111-6.

Aberrant NF-KappaB Expression in Autism Spectrum Condition: A Mechanism for Neuroinflammation. Young AM at al. (2011). Front. Psychiatry. 2: 27.

IL-6 is increased in the cerebellum of autistic brain and alters neural cell adhesion, migration and synaptic formation. Wei H et al (2011) J Neuro-inflammation.; 8: 52.

A study of nuclear transcription factor-kappa B in childhood autism. Naik US et al (2011) PLoS One. May 9;6(5):e19488.

Abnormal regional cerebral blood flow in childhood autism. Ohnishi T (Sep 2000) Brain 123(9):1838-44.

Impaired Carbohydrate Digestion and Transport and Mucosal Dysbiosis in the Intestines of Children with Autism and Gastrointestinal Disturbances. Williams BL et al. (2011) PLoS ONE 6(9): e24585.

Gastrointestinal flora and gastrointestinal status in children with autism -- comparisons to typical children and correlation with autism severity. Adams JB et al. (2011) BMC Gastroenterol.2011 Mar 16;11:22.

Intestinal disaccharidase activity in patients with autism: effect of age, gender, and intestinal inflammation. Autism. Kushak RI et al (2011) May; 15(3):285-94. BMC Gastroenterology, 11:22

Intestinal lymphocyte populations in children with regressive autism: evidence for extensive mucosal immunopathology. Ashwood P et al. (2003). J Clin Immunol. 23(6):504-17.

Abnormal intestinal permeability in children with autism. D'Eufemia P et al. (1996). Acta Paediatr. 85(9):1076-9.

Gastrointestinal abnormalities in children with autistic disorder. Horvath K et al (1999). J Pediatr. 1999 Nov;135(5):559-63.

Colonic CD8 and gamma delta T-cell infiltration with epithelial damage in children with autism. Furlano RI et al (2001). J Pediatr.138(3):366-72. Urinary Metabolic Phenotyping Differentiates Children with Autism from Their Unaffected Siblings and Age-Matched Controls. J Nicholson J et al. (2010) Journal of Proteome Research

Metabolic biomarkers of increased oxidative stress and impaired methylation capacity in children with autism. James SJ et al. (2004). Am J Clin Nutr. 80(6):1611-7.

Further references available on request.