

Written Public Comments

**IACC Full Committee
Meeting**

April 26, 2017

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Note: Personally Identifiable Information (PII) has been redacted in this document

Linda Varsou

April 26, 2017

Dear Advocates and Researchers for the cause of Autism:

I know that most of you were aware about these excellent and scientifically documented free presentations on “**Vaccines Revealed**” that started on 1/10/17. In case you missed them, today, is the last day to register and even to buy the entire 9 episodes. It is not only about the relationship between vaccines and autism, but about vaccinations in general (HPV, Flu, Anthrax, etc.).

Back in 1972, I was a fellow and researcher in immunology at the Institute Pasteur of Paris, where Louis Pasteur in 1885 developed his first rabies vaccine. I was forced to get the BCG vaccine otherwise I had to leave the Institute! After this fist vaccination in my life, I ended up to a hospital and since some lesions are still in my skin. Later on, with a group of researchers on white lab coats, we marched in the streets of Paris to protest against the fact that the Institute Pasteur sent expired vaccines to Africa.

Since then, I have followed closely all the vaccines’ evolution and more intensively for the last 31 years, after the birth of my autistic son. Genes alone cannot explain the epidemic increase of autism, but epigenetics can at any level, prenatal and/or postnatal as well (toxic substances in vaccines, environment, etc.).

I am not against vaccinations. For example, for someone who plans to travel to a country where some bacteria or viruses are endemic while the local population is healthy and “immune” towards them, vaccination is an important safety measure to be taken. But based on the new scientific facts, we know that the immune and nervous system of newborns and babies is totally immature. Any vaccination at this early age, even with “safe vaccines” can cause immediate adverse reactions, even autism, or lifelong chronic autoimmune diseases, etc., due to the “immune-deviation” mechanism produced at a still immature human substrate.

I wish that the NIH/NIMH/IACC Board, together with the many autism organizations and based on the clear cut information gathered in this amazing documentary “**VaccinesRevealed**”, will support the initiative to solve the public health problem related to vaccinations, here and right now.

Thanking you for your attention. Sincerely yours,

*Linda-Anglique Varsou-Papadimitriou, PhD, MPH, DABCC, Immunologist, Hygienist, Clinical Biochemist, Associate Professor in Medicine, advocate for the Rights and Strengths of People with Autism; supporter of the concept: “**An Autism Friendly Society will benefit us all**”. Member of different autism related organizations in the US and Europe. Long-standing member of “Autism-Europe” and elected member to its Council of Administration. MSc and postgraduate studies in Biochemistry and Specialization in Immunology, Paris, France; MSc in Public Health; Diplomate of the American Board of Clinical Chemistry; Two PhDs in Diagnostic Laboratory Medicine; Research Associate at the University of Maryland School of Medicine, Faculty member at Johns Hopkins University School of Medicine where I studied neurosciences and initiated the “fever study in autism”. Contact: [PII redacted]*

P.S.: I have to disclose that I have not any financial or non-financial relationships related to the “Vaccines Revealed” documentary.

Autism: A special case of vaccine-induced cow's milk allergy?

https://www.researchgate.net/publication/313248416_Autism_A_special_case_of_vaccine-induced_cow%27s_milk_allergy

https://www.researchgate.net/publication/301602551_Pandemrix_induces_narcolepsy_and_cow%27s_milk_contaminated_vaccines_induce_Autism_Spectrum_Disorders

https://www.researchgate.net/publication/312125211_Professional_Misconduct_by_NAM_Committee_on_Food_Allergy?ev=prf_pub

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Carlos Garay

April 26, 2017

Hullo. I'm an Argentine University Therapist specialized in Autism. I'd like to make some special kind of researches at NIH's.

I think that ABA, SPEC, Tteach, and other excellent methods and techniques are excellent but they can get stuck.

I'm working in a PhysioMath Project.

Autistic patients manage/drive themselves in another kind of planes that have a lot to do with Physics and Math's concepts, even with Quantum fields.

We can verify by direct clinical observation that autistic children at least by perceiving it during the dialogue between he/she with the therapist. How they try to find a point of contact with our planes that belong to our dimension.

Below, in the ABA's therapy video I see that the autistic child willingly tries to find and follow a point of contact with his therapist but he repeats nouns or actions that is not able to manage himself completely. Following this explanation we could find the causes of his difficulty of his speech difficulties.

I've enclosed some videos to illustrate my idea and a copy of my CV ([PII redacted]).

Autism Therapy – ABA:

<https://www.youtube.com/watch?v=NbVG8IYE5Ns>

4th Dimension Tesseract, 4th Dimension Made Easy Carl Sagan YouTube:

<https://www.youtube.com/watch?v=N7K5KjOdLD8>

It's a special opportunity to express to IACC that I'm making researches on Autism and Therapies.

I've been following and studying famous Dr Albert Einstein's personal biography (it says that he suffered from Asperger Disorders).

Taking into account his theories I'm nearly sure that autistic patients watch reality not in a 3D like common people's natural synapse patterns but in an awesome 3D-4D.

That fact explains many important psych and physics facts specially why autistic children gets unquiet and/or produces "emotional shoots".

ABA, SPEC, Tteach and other therapies and methods are fantastic ones but would fail in an unforeseen time. Methods have to be continuously upgraded by following dimensional patterns.

IACC,

Please include the following attachments in your materials for distribution.

Tylenol is the trigger for Autism when given as a fever reducer. Tylenol depletes glutathione needed to metabolize the harmful metals in vaccines.

Substitution of Acetaminophen for aspirin began the Autism epidemic in the early 1980s. Stop the use of Acetaminophen and the Autism epidemic will end...

KerryScott Lane MD

Palm Beach Autism Institute

Attachments:

https://www.researchgate.net/publication/315324979_The_role_of_oxidative_stress_inflammation_and_acetaminophen_exposure_from_birth_to_early_childhood_in_the_induction_of_autism

<http://www.mdpi.com/1099-4300/14/11/2227/htm>

<https://www.google.ch/patents/US9442092>

Glutathione Loss by Gliotoxin, then Acetaminophen, Results in Metal Intoxication and Oxidative Stress-Causing Autism

Kerry Scott Lane M.D.

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Toxicity of Acetaminophen

- Background-APAP is the most widely used analgesic worldwide. Most common cause of accidental poisoning.
- Became commonly used in early 1980s due to concern aspirin was causing Reye's Syndrome
- Overdose occurs when conjugative pathways become saturated. APAP is then oxidatively metabolized by liver p450 to NAPBQI which normally is conjugated with glutathione and renally excreted.

Acetaminophen Pathophysiology

- When glutathione levels are reduced, NAPQI binds to lipid hepatocytes and vital proteins, causing Oxidative Stress, inflammation and hepatocellular Zone III necrosis.
- Rx for APAP is N-Acetylcysteine-NAC, a glutathione precursor, and may enhance sulfate conjugation of free APAP.
- NAC is also anti-inflammatory, an antioxidant, and increases Nitric Oxide levels and local pO₂.
- NAC best given within 8hrs, decreasing mortality. New IV version called Acetadote.

Oxidant Stress, Reactive Nitrogen Species and APAP Toxicity

- APAP depletes Glutathione and binds to cellular and mitochondrial proteins. This inhibits and stresses mitochondrial respiration, causing ATP depletion.
- When Vitamin E is present, ROS do not induce lipid peroxidation, but superoxide anion reacts with Nitric Oxide-NO-to form peroxynitrite, thus modifying cell molecules and worsening mitochondrial dysfunction and ATP depletion.

- This liver model is likely worse in the brain due to the dependence of the brain on blood glutathione.
- Neuronal Cells in the brain are wholly dependent on circulating blood borne Glutathione.

Glutathione and Analgesics

- GSH is a vital antioxidant, it detoxifies drugs and ROS, regulates gene expression and effects membrane transport and apoptosis.
- Homeostasis is critical as turnover of GSH is dynamic, the precursors are the amino acids cysteine, glutamic acid and glycine.
- Toxic APAP levels deplete GSH, which is seen in Alcoholics, AIDS, malnutrition, cirrhosis, PIH, and likely others

APAP, MMR Vaccine & Autism

- Schultz (UCSD) et. al. (Autism May 2008) using an on-line case control study surveyed 83 Autism families and 80 controls.
- APAP after MMR was significantly associated with Autism at O.R. 6.1(less than 5y.o.). With Regression only (O.R. 3.97) and w/ post-vaccination sequelae only (O.R.8.23).
- Ibuprofen showed no association.
- Therefore, APAP after MMR is associated with Autism. How to we explain this pathobiology?

APAP Induced Oxidative Stress-Protection by N-Acetylcysteine

- APAP increased cell and mitochondrial glutathione disulfide (GSSG) and altered GSSG:GSH ratio suggesting an oxidative stress role.
- Autistic children have elevated oxidized/reduced GSH ratio.
- Rx with NAC enhanced cell glutathione and reduced cellular loss.
- APAP-Oxidative Stress precedes cellular injury and is involved in spread of cell injury.

Oxidative Stress, Mitochondria and Glutathione Protection

- Mitochondria provide energy and are involved in cell death. Oxidative phosphorylation generates ROS from the electron transport chain.
- Superoxide anion and H₂O₂ are produced by transition metals in aerobic respiration. GSH in mitochondria is the only defense to H₂O₂.
- Glutathione is required to move GSH from the cytosol to the mitochondria.
- Glutathione transport is stabilized by Selenium-Adenosyl Methionine (SAM).

SAM and NAC are Protective Against APAP Hepatotoxicity

SAM and NAC were compared in the treatment of APAP toxicity. In one study SAM was more protective. Cysteine/Glutamate exchange modulates Glutathione supply for neuroprotection against Oxidative Stress.

Glutathione synthesis is limited by Cysteine.

NAC, SAM and GSH treatment raise Erythrocyte GSH. E-GSH may be cheapest- quick screening test for Autism susceptibility.

Sulfate Metabolism is Abnormal in Autistic Children

- Sulfation is important in metabolism of neurotransmitters and digestive hormones.
- Children w/ Autism had low APAP/sulfate glucuronide level- suggesting decreased sulfation capacity in the liver. This is associated with increased urinary sulfite and sulfate excretion.

- Are Sulfate metabolism defects in Autistic children innate or acquired?

Glutathione Metabolism in CSF and the Blood Brain Barrier

- Glutathione in the Choroid Plexus is metabolized like urine in the tubule in kidneys. CSF GSH can be modified by drugs that do not affect brain GSH. GSH monoethyl ester can be transported into CSF (potential treatment?).
- Gamma-Glutamyl transpeptidase, which is high in the choroid plexus, is the only enzyme able to cleave the gamma-glutamyl bond of glutathione.
- Brain mitochondria is more susceptible to GSH loss and Oxidative Stress than liver. H₂O₂, if not reduced, can lead to lipid hydroperoxides, damaging the mitochondrial membranes and their function.
- Autistic children show loss of Purkinje Cells and Increased Brain Volume...What is the etiology of this?

Glutathione Redox State Regulates Mitochondrial Reactive Oxygen

- Impaired Glutathione stores in Brain further increase mitochondrial ROS causing a cascade of further damage.
- To function as an antioxidant, Glutathione must be in the reduced form- to act as an electron donor. Normally there are 100 reduced GSH molecules vs. oxidized. But Autistic patients have mostly oxidized GSH!!

Glutathione Loss, Oxidative Stress and Metabolic Errors in Autism

- Autistic children have Immune, Digestive, and Metabolic Abnormalities, i.e. low plasma inorganic sulfate and sulfur oxidation deficiencies.
- Irritable Bowel Disease (IBD) patients have low GSH- this deficiency may be target for intervention.
- Many Autistic children have GI abnormalities-why?
- Intrinsic defects in Immunity are seen in GI positive Autistic Syndrome Disorder (ASD) Children.
- CD4 cell studies should be done to evaluate this. How does this relate to the suspected pathology?

Oxidative Stress In Autism

A. & V. Chauhan

- Lipid Peroxidation markers are elevated in Autism.
- Transferrin (Iron binding) and Ceruloplasmin (Copper binding) levels are decreased, leading to altered Fe and Cu metabolism with sequelae.
- Antioxidant Serum proteins are decreased, i.e. albumin, total protein and others.
- Other enzymes altered include superoxide dismutase (SOD), glutathione peroxidase, catalase, homocysteine, and methionine, causing inflammation, excitotoxicity, mitochondrial and generalized immune dysfunction.

GSH Determines Sensitivity toward Hg-Induced Oxidative Stress

- Cortical and Cerebellar Areas exhibit differential sensitivity to Mercury-induced ROS. Where are the Purkinje cells located?
- Methylmercury (MeHg) is highly neurotoxic.
- GSH protects against MeHg neurotoxicity in neurons and astrocytes.

- Depletion of GSH increases MeHg toxicity and Stress. GSH precursors-NAC & SAM-are neuroprotective.
- Are APAP, Gliotoxin & Hg Synergistic ?

Thimerosal & Impaired Mitochondrial Oxidation/Reduction Activity

- Ethylmercurithiosalicylic acid, an Ethyl-Hg releasing compound, has been in use 70+ years. Thimerosal is 50% Mercury.
- Thimerosal neurological damage is similar to that seen in Autism.
- TM is a preservative used in multi-dose vaccines. TM was removed from most vaccines in 2002 but Autism epidemic continues unabated....Why?
- Salicylates linked to Mitochondrial Re-Dox impairment in Reye's Syndrome...a role here?

Candida and *Aspergillus* Species Yeasts Produce Gliotoxin

- Gliotoxin is immunosuppressive by killing CD4 cells (AIDS?), and disrupts **many** enzyme systems.
- 32 of 50 of *Candida* tested produced a ETP-epipolythiodioxopiperazine-like compound.
- Gliotoxin, a hydrophobic molecule, was found in many cancer patients at MD Anderson in amounts far in excess of that required to cause pathology.
- 93% of *Aspergillus fumigatus*, 75% *A. niger* and 25% *A. terreus* produced Gliotoxin.
- *A.fumigatus* had the highest concentrations of GT.

Gliotoxin Toxicity and Mode of Action

- Toxicity of ETPs is due to presence of the Disulphide bridge, which inactivates proteins via reaction with thiol groups, and generation of ROS by Redox cycling.
- Gliotoxin possibly reacts with disulphide cysteine bonds in insulin causing loss of function and Diabetes.
- Gliotoxin suppresses T-Cell response, inhibits the antigen presenting response and led to a preferential death of monocytes and derived Dendritic cells (AIDS?).
- Physiological effects of GT are way below levels of GT found in cancer patients. This is a dangerous molecule...
- Gliotoxin production by *A. Fumigatus* may be an immunoevasive mechanism, acting on T cells.

Gliotoxin Pathobiology

- Gliotoxin inactivates muscle Creatine Kinase by a reversible formation of a Disulfide Bond. CK is coupled to the regeneration of ATP to ADP and the Mitochondrial Permeability Pore (MPP).
- The Gliotoxin Disulphide bond reacts with accessible cysteine residues on proteins, enzymes, hormones and synaptic receptors. Is there a synaptic role in Autism?
- GT inhibits alcohol dehydrogenase, reverse transcriptase, farnesyltransferase, and transcription factor NFkappa B.
- GT may similarly inactivate insulin in Gestational Diabetes and alter Nitric Oxide signaling, causing PIH.
- Bioactivity may be restored when treated with reducing agents as the oxidized forms showed loss of function.

Gliotoxin-Glutathione Adducts Deplete Physiological GSH Stores

- Mechanism of Action of GT is disulfide linkage w/ Sulfur nucleophiles in a thiol-disulfide exchange mechanism.
- GT/GSH equilibrium depends on pH and GSH level.

- Depletion of GSH by GT has numerous pathophysiological consequences.
- ReDox mechanism for GSH dependent reversible uptake of GT in cells.
- GT uptake to cells is dependent on GSH, and a 1500 fold increase of reduced GT is found intracellularly, as opposed to outside the cell.
- Oxidation of intracellular GT effluxes GT, and the efflux of GT is also caused by low GSH from cellular apoptosis.
- The above allows for a minor Gliotoxin production, while causing a maximum intracellular concentration, yielding maximum killer efficacy against a competitive organism.
- Decreased intracellular Glutathione after apoptosis by GT releases more GT for further cell killing.

Metallothionein is Regulated by Oxidative Stress and Metal Ions

- MT are small cysteine-rich (30%) heavy metal binding proteins which protect against stress and regulate Zinc and metal levels.
- Cysteine residues bind and store metal ions, such as Zn, Cu, Cd and others (mercury?).
- MT has antioxidant capabilities.
- MT deficient knockout mice are sensitive to APAP hepatotoxicity.
- **APAP and Gliotoxin deplete Glutathione, essentially creating Metallothionein deficient Autistic children.**

Metallothionein and Zinc

- MT involved in Zinc metabolism. This control is linked to cellular Glutathione Status.
- Glutathione disulfide S-thiolation causes MT clusters to collapse and release Zinc.
- Zinc is released by oxidants, and antioxidants preserve this binding. A GSH/GSH disulfide redox coupling mechanism and Selenium effect Zinc delivery.
- Oxidation-Reduction state is critical to the direction of Zinc transfer and distribution.
- MTs regulate Mercury Neurotoxicity, esp. MT III.

Is Zinc Induced Neuronal Death a Pathology In Autism?

- Intracellular free Zinc contributes to Energy Failure by Loss of NAD⁺ and Inhibition of Glycolysis, prior to Mitochondrial Permeability Transition (MPT), inducing neuronal cell death.
- Zinc neuronal death is attenuated by oxaloacetate, dichloroacetate and niacinamide(possible treatment?)
- Zinc inhibits Mitochondrial respiration at bc1 and alpha-ketoglutarate dehydrogenase (see earlier slide).
- Zinc is a novel intracellular second messenger with likely implications when disrupted.

Synaptic Cysteine Sulfhydryls are targets of Neurotoxicants

- Sulfhydryl reactive metals (Hg, Cd, As, Pb) in the hair of Autistic children was lower than controls, leading to the conclusion these children had trouble excreting these metals.
- Impaired MT processing may be the etiology.
- Are these metals bound to neurotransmission sites or other critical areas in Autistic children-causing the pathobiology?

Brain Neurotoxicity

- Astrocytes are essential to provide GSH precursors to neurons.

- Thimerosal Neurotoxicity is associated with GSH depletion. GSH provides defense against Mercury toxicity.
- NAC & glutathione ethyl ester prevented cytotoxicity. GSH is poorly absorbed via GI tract so other routes must be chosen.
- Role for other neuroprotectants? (SAM, B6, B12 etc.)

The Etiology of Autism

Summary and Conclusions

- Antibiotic Therapy changes GI flora favoring toxic yeasts. Antibiotics can also decrease the ability to detoxify metals by 90%, esp. Mercury.
- Gliotoxin is produced by the new GI flora by *Aspergillus* and *Candida*. Gliotoxin adducts & depletes Glutathione to marginal levels. When GSH is less than 30% of normal or oxidized APAP becomes toxic.
- Acetaminophen-at perivaccination period-is then associated with Critical Glutathione Loss, especially in the brain by crossing the BBB. Gliotoxin and APAP both cause Oxidative Stress. GSH needed for metal metabolism
- Thus, Metallothionein-MT- and most metal metabolism and multiple enzyme pathways are affected, compounding the toxicology.
- This causes a “Perfect Storm” of Oxidative Stress, GSH Depletion, and Zinc and metal toxicity (Hg-Thimerosal?) creating a condition of mitochondria dysfunction in the brain-leading to hypoxia and Autism-CO?
- Multiple pathways of pathobiology possibly indicate multiple therapeutic modalities and preventive approaches, e.g. precursors, vitamins, etc.

References

1. Toxicity of Acetaminophen, S.E. Farrell, MD, Assistant Professor of Medicine, Harvard Medical School; Dept. of Emergency Medicine, Brigham and Women's Hospital.
2. Analgesics and Glutathione, Am. J. Ther. 2002 May-Jun;9(3):225-33, Lauterburg BH. Department of Clinical Pharmacology, University of Bern, Bern, Switzerland.
3. Acetaminophen use, measles-mumps-rubella vaccination, and autistic disorder, Autism 2008, The National Autistic Society Vol. 12(3) 293-307.
4. Acetaminophen-Induced Oxidant Stress and Cell Injury in Cultured Mouse Hepatocytes: Protection by N-Acetyl Cysteine, M. L. Bajt, T. Knight, J. Lemasters, H. Jaeschke, Toxicological Sciences, 80, 343–349 (2004).
5. Oxidative stress: Role of mitochondria and protection by glutathione, BioFactors, 1872-8081, Issue Volume 8, Numbers 1-2/1998 Pages 7-11, J.C. Fernandez-Checa¹, C. Garcaia-Ruiz¹, A. Colell, A. Morales, M. Marai, M. Miranda¹, E. Ardite, Instituto Investigaciones Biomedicas, Consejo Superior Investigaciones Cientificas (CSIC), and Liver Unit, Department of Medicine, Hospital Clinic I Provincial, Barcelona, Spain.
6. An update of N-acetylcysteine treatment for acute acetaminophen toxicity in children. Curr. Opin. Pediatr. 2005 Apr; 17(2):239-45, L. Marzullo.
7. Comparison of S-Adenosyl-L-methionine an N-Acetylcysteine, Protective Effects on Acetaminophen Hepatic Toxicity, M. Terneus, K. Kinningham, A. Carpenter, S. Sullivan, M. Valentovic, The Journal of Pharmacology and Experimental Therapeutics.
8. Abnormal Sulfate Metabolism in Autism, Horvath, K., Waring, R.H., Rabszlyn A., Blagg, S., Campbell, Z., Klovra, L.V., Journal of Pediatric Gastroenterology and Nutrition Volume 39, June 2004

9. Cystine/Glutamate Exchange Modulates Glutathione Supply for Neuroprotection from Oxidative Stress and Cell Proliferation, *The Journal of Neuroscience*, October 11, 2006 26(41):10514-10523, A. Shih, H. Erb, X. Sun, S. Toda, P. W. Kalivas, T. Murphy.
10. Cysteine supplementation improves the erythrocyte glutathione synthesis rate in children with severe edematous malnutrition, A. Badaloo, M. Reid, T. Forrester, W. Heird and F. Jahoor, *The American Journal of Clinical Nutrition*.
11. Glutathione metabolism at the blood-cerebrospinal fluid barrier, M. Anderson, M. Underwood, R. Bridges, A. Meister, Departments of Biochemistry and Neurobiology, Cornell University Medical College, NY, NY 10021.
12. Evidence of Toxicity and Oxidative Stress and Neuronal Insult in Autism, J. Kern, A. Jones, Department of Psychiatry, University of Texas, Southwestern Medical Center at Dallas, Texas, USA, *Journal of Toxicology and Environmental Health, Part B*, 9:485–499, 2006.
13. Impairment of intestinal glutathione synthesis in patients with inflammatory bowel disease, B. Sido, V. Hack, A. Hochlehnert, H. Lipps, C. Herfarth, W. Droge, *Gut* 1998; 42:485-492.
14. Glutathion Depletion and Formation of Glutathione-Protein Mixed Disulfide Following Exposure of Brain Mitochondria to Oxidative Stress, V. Ravindranath and D. Reed, *Biochemical and Biophysical Research Communications*, Vol. 169, No.3, June 29, 1990, Pages 1075-1079.
15. Diagram of GSH/GSSG Redox Pair Anti-Oxidant Defense.
16. Oxidative stress in autism, A. Chauhan, V. Chauhan, *Pathophysiology*, 13 (2006) 171–181.
17. The role of oxidant stress and reactive nitrogen species in acetaminophen hepatotoxicity. *Toxicology Letters*, 2003, Oct 15; 144(3):279-88 Jaeschke H, Knight TR, Bajt ML.
18. Glutathione Redox State Regulates Mitochondrial Reactive Oxygen Production, *The Journal of Biological Chemistry*, Vol. 280, No. 27, Issue of July 8, pp. 25305-25312, 2005, D. Sen, T. Dalton, D. Nebert, H. Shertzer.
19. Role of glutathione in determining the differential sensitivity between the cortical and cerebellar regions towards mercury-induced oxidative stress, *Toxicology*, Volume 230, Issues 2-3, 12 February 2007, Pages 164-177, P. Kaur, M. Aschner, T. Syversen.
20. The protective effects of glutathione against methyl mercury cytotoxicity, L. Kromidas, L. Trombetta, I. Siraj Jamall, *Toxicology Letters*, Volume 51, Issue 1, March 1990, Pages 67-80.
21. Glutathione modulation influences methyl mercury induced neurotoxicity in primary cell cultures of neurons and astrocytes, P. Kaur, M. Aschner, T. Syversen, *NeuroToxicology* 27 (2006) 492-500.
22. Mitochondrial dysfunction, impaired oxidative-reduction activity, degeneration, and death in human neuronal and fetal cells induced by low-level exposure to thimerosal and other metal compounds, D. A. Geier, P. G. King, M. R. Geier, *Toxicological & Environmental Chemistry*, Volume 91, Issue 4, June 2009, pages 735 – 749.
23. Dysregulated Innate Immune Responses in Young Children with Autism Spectrum Disorders: Their Relationship to Gastrointestinal Symptoms and Dietary Intervention, H. Jyonouchi, L. Geng, A. Ruby, B. Zimmerman-Bier, *Neuropsychobiology* 2005; 51:77-85.
24. Clinical isolates of yeast produce a gliotoxin-like substance, D. Shah and B. Larsen, *Mycopathologia*, Vol 116, Number 3, December 1991.
25. Frequency and Species Distribution of Gliotoxin-Producing *Aspergillus* Isolates Recovered from Patients at a Tertiary-Care Cancer Center, R. Lewis, N. Wiederhold, M. Lionakis, R. Prince, D. Kontoyiannis, *Journal of Clinical Microbiology*, Dec. 2005, p. 6120-6126.
26. The epipolythiodioxopiperazine (ETP) class of fungal toxins: distribution, mode of action, functions and biosynthesis, D. Gardiner, P. Waring, B. Howlett, *Microbiology* (2005), 151, 1021-1032.
27. *Aspergillus fumigatus* suppresses the human cellular immune response via gliotoxin-mediated apoptosis of monocytes, M. Stanzani, E. Orciuolo, R. Lewis, D. Kontoyiannis, S. Martins, L. St. John, K. V. Komanduri, *Blood*, 2005; 105:2258-2265.

28. Effect of Gliotoxin on Human Polymorphonuclear Neutrophils, D.T. Shah, S. Jackman, J. Engle, and B. Larsen, *Infectious Diseases in Obstetrics and Gynecology*, 6:168-175 (1998).
29. Exacerbation of invasive aspergillosis by the immunosuppressive fungal metabolite, gliotoxin, P. Sutton, P. Waring, A. Mullbacher, *Immunology and Cell Biology* (1996) 74, 318-322.
30. The Immunosuppressive Fungal Metabolite Gliotoxin Specifically Inhibits Transcription Factor NF- κ B, H. Pahl, B. Kraub, K. Schulze-Osthoff, T. Decker, E. Britta, Mareen Traenckner, M. Vogt, C. Myers, T.Parks, P. Waring, A.Mullbacher, A. Peter Czernilofsky, P. A. Baeuerle, *J. Exp. Med.*, Volume 183, April 1996, 1829-1840.
31. The Isolation of Gliotoxin and Fumigacin From Culture Filtrates of *Aspergillus Fumigatus*, A. Menzel, O. Wintersteiner, J. C. Hoogerheide, *The Journal of Biological Chemistry*, p. 419.
32. Gliotoxin Causes Oxidative Damage to Plasmid and Cellular DNA, R. Eichner, P. Waring, A. Geue, A. Braithwaite, A. Mullbacher, *The Journal of Biological Chemistry*, Vol 263, No. 8, March 15, pp. 3772-3777, 1988.
33. Gliotoxins disrupt alanine metabolism and glutathione production in C6 glioma cells: a ^{13}C NMR spectroscopic study, L. Brennan, C. Hewage, J. Malthouse, J. McBean, *Neurochemistry International*, 45 (2004) 1155-1165.
34. Immunosuppression *in vitro* by a metabolite of a human pathogenic fungus. A. Mullbacher, R. D. Eichner, *Proc. National Academy Science, USA*, Vol. 81, pp. 3835-3837, June 1984.
35. Gliotoxin production by clinical and environmental *Aspergillus fumigatus* strains, C. Kupfahl, *International Journal of Medical Microbiology*, Volume 298, Issues 3-4, 1 April 2008, Pages 319-327.
36. Detection of Gliotoxin in Experimental and Human Aspergillosis, R. Lewis, N. Wiederhold, J. Han, K. Komanduri, D. Kontoyiannis, and R. Prince, *Infection and Immunity*, Jan. 2005, p. 635-637, Vol.73, No. 1.
37. Gliotoxin inactivates alcohol dehydrogenase by either covalent...*Biochemical Pharmacology*, Vol 49, Issue 9, 11 May 1995, pages 1195-1201.
38. Fungal Metabolite Gliotoxin Inhibits Assembly of the Human Respiratory Burst NADPH Oxidase, S. Tsunawaki, L. Yoshida, S. Nishida, T. Kobayashi, T. Shimoyama, *Infection and Immunity*, June 2004, p. 3373-3382.
39. Inactivation of Rabbit Muscle Creatine Kinase by Reversible Formation of an Internal Disulfide Bond Induced by the Fungal Toxin Gliotoxin, A. Hurne, C. Chai, P. Waring, *The Journal of Biological Chemistry*, Vol. 275, No. 33, Issue of August 18, pp. 25202-25206, 2000.
40. Evidence for Gliotoxin-Glutathione Conjugate Adducts, P. Bernardo, C. Chai, G. Deeble, X.Ming Liu, P. Waring, *Bioorganic & Medicinal Chemistry Letters*, 11 (2001) 483-485.
41. A Novel Redox Mechanism for the Glutathione-dependent Reversible Uptake of a Fungal Toxin in Cells, P. Bernardo, N. Brasch, C. Cha, P. Waring, *The Journal of Biological Chemistry*, Vol 278, No. 47, Issue of November 21, pp. 44549-46555, 2003.
42. Glutathione intensifies gliotoxin-induced cytotoxicity in human neuroblastoma SH-SY5Y cells, V. Axelsson, K. Pikkarainen and A. Forsby, *Cell Biology and Toxicology*, 2006; 22; 127-136.
43. Mechanism of Action of Gliotoxin: Elimination of Activity by Sulfhydryl Compounds, P.W. Trown, J. A. Bilello, *Antimicrobial Agents and Chemotherapy*, Oct. 1972, p. 261-266. Vol. 2, No. 4.
44. Anti-metallothionein IgG and levels of metallothionein in autistic children with GI disease, A. J. Russo...
45. Metallothionein-I/II Knockout Mice are Sensitive to Acetaminophen-Induced Hepatotoxicity, J. Liu, Y. Liu, D. Hartley, C. Klaassen, S. Shehin-Johnson, A. Lucas, S. Cohen, *The Journal of Pharmacology and Experimental Therapeutics*, Vol. 289, No. 4, 289: 580-586, 1999.
46. Regulation of Metallothionein Gene Expression by Oxidative Stress and Metal Ions, G. Andrews, *Biochemical Pharmacology*, Vol. 59, pp. 95-104, 2000.

47. Oxidative metal release from metallothionein via zinc-thiol/disulfide interchange, W. Maret, Proc. Natl. Acad. Sci. USA, Vol. 91, pp. 237-241, January 1994.
48. The Function of Zinc Metallothionein: A Link between Cellular Zinc and Redox State, W. Maret, The Journal of Nutrition, p. 1455.
49. Control of zinc transfer between thionein, metallothionein, and zinc proteins, C. Jacob, W. Maret, B. Vallee, PNAS.....
50. Metallothioneins: Mercury Species-Specific Induction and Their Potential Role in Attenuating Neurotoxicity, M. Aschner, T. Syversen, D. O. Souza, J. Rocha.....
51. Phenotypic variation in xenophobic metabolism and adverse environmental response: focus on sulfur-dependent detoxification pathways, S. McFadden, Toxicology 111 (1996) 43-65.
52. Zinc-Induced Cortical Neuronal Death: Contribution of Energy Failure Attributable to Loss of NAD and Inhibition of Glycolysis, C. Sheline, M. Behrens, D. Choi, The Journal of Neuroscience, May 1, 2000, 20 (9): 3139-3146.
53. Zinc Irreversibly Damages Major Enzymes of Energy Production and Antioxidant Defense Prior to Mitochondrial Permeability Transition, I. Gazaryan, I. Krasinskaya, B. Kristal, A. Brown, Journal of Biological Chemistry, Vol. 282, Issue 33, 24373-24380, August 17, 2007.
54. Zinc is a novel intracellular second messenger, S. Yamasaki, K. Sakata-Sogawa, A. Hasengawa, T. Suzuki, K. Kabu, E. Sato, T. Kurosaki, S. Yamashita, M. Tokunaga, K. Nishida, and T. Hirano, The Journal of Cell Biology, Vol. 177, No. 4, 637-645.
55. Sulfhydryl-Reactive Metals in Autism, J. Kern, B. Grannemann, M. Trivedi, Journal of Toxicology and Environmental Health, Part A, 70: 715-721, 2007.
56. Synaptic Cysteine Sulfhydryl Groups as Targets of Electrophilic Neurotoxicants, R. LoPachin, D. Barber, Toxicological Sciences, 94 (20), 240-255 (2006).
57. The Consequences of Methylmercury Exposure on Interactive Functions between Astrocytes and Neurons, J. Allen, G. Shanker, K. Tan, M. Aschner, Neurotoxicology 23 (2002) 755-759.
58. Thimerosal Neurotoxicity is Associated with Glutathione Depletion: Protection with Glutathione Precursors, S. J. James, W. Slikker III, S. Melnyk, E. New, M. Pogribna, S. Jernigan, NeuroToxicology 26 (2005) 1-8.
59. Sequential Opening of Mitochondrial Ion Channels as a Function of Glutathione Redox Thiol Status, M. Aon, S. Cortassa, C. Mack, B. O'Rourke, The Journal of Biological Chemistry, Vol. 282, No. 30, pp. 21889-21900, July 27, 2007.

Note: Profanity has been redacted in this document

John Best

April 26, 2017

You [profanity redacted] have been lying about autism for a long time. You are hereby ordered to tell the public that autism is caused by mercury in vaccines.

For the people,
John Best,
Londonderry, NH

I have a 5-½ year old moderate / severe ASD boy with tics and PANs. My statement and public request of the IACC board is again, being repeated to address my previous letters from the last two meetings (that have gone unaddressed).

1.) Speak with parents more and focus research on their feedback. There is truth in the herd.

I request that the IACC facilitates a survey the parents of ASD children in the United States. I request that this survey is over 50 but under 100 questions pertaining an ASD child and overseen and co-managed by a third party foundation, or organization for Autism that is recommended and voted on by the public. I request that the IACC proposes and allocates funding for this study in the fiscal year of 2016 to be published no later than the spring of 2017.

Over the past 28 months since my son's official diagnosis we have invested all free time, over \$89,000 out of pocket on ABA, OT, Speech, accessories, learning aids, medical tests and vitamins. In addition to 28-32 hours a week of ABA in-home he is also attending a special education class 5 days a week, 4 hours a day. We've done the EEG's, 4 rounds now of different gene testing as well.

16 months ago we finally gave in and had allergies, hair, stool and urine tested? My son is allergic to many items. He's off the charts in aluminum, copper, lithium, rubidium and cesium. Then he was diagnosed with PANs and had a scare of Lyme as well.

We immediately started natural chelation with nutrients. We went GF/CF/SF and eliminated all sugars. We went 100% organic and juice every day. All chicken is free-range, antibiotic free and expensive. All beef is grass fed, non-GMO and expensive. Every bit of food that enters his body is known to the source and purity.

Results::

- any gluten, any sugar causes extreme aggression and yeast flare
- any "normal" produce produces foul stool, changes behavior and increases stims

What I also learned:

My road has many miles to travel, but I've covered more ground with natural healing than I did with any Dr's 7 minute consultation or prescription recommendation (what the hell is Marinol anyways and why would my child be prescribed this and not natural cannabis oil?) I'm not the only one. My path was paved by many, walked by thousands and is continuously modified with new tests, strategies and nutrients.

Parents live autism. They see changes that are microscopic. They notice what causes changes. They talk to one-another and compare notes. Compare Dr's. Compare protocols. Compare results.

2.) Glyphosate. What are the affects on the human brain? What are the affects on the human ASD brain? Are there correlations, that have been studied between Glyphosate exposure and Autism Spectrum Disorders?

Why would a 4-½ year old child on the spectrum who was breast fed for two years and ate a natural, healthy diet have over 3x the normal levels of Glyphosate in his blood? We do not live near a farm, he does not work in produce, nor a processing plant.

Can the IACC to investigate how Glyphosate is affecting children with ASD vs. Non-ASD in the fiscal year of 2016?

3.) The IACC makes a formal request to Congress to subpoena Dr. William Thompson at the CDC.

Since his admission of falsifying tests, at the request of his superiors on how children receiving the MMR vaccine before 36 months were 340% more likely to receive an autism diagnosis or develop tics. Dr. Thompson made admissions to Biochemical Engineer Brian Hooker in a series of phone calls and not only gave specifics on how to obtain the correct data but also expressed remorse in his cover-up.

I ask: why hasn't the IACC been concerned with this information? Why hasn't the IACC even asked for clarification from the CDC and response been made public?

I request that the IACC makes a public, formal request to Congress to subpoena Dr. William Thompson of the CDC.

I request that the IACC makes a public, formal request to Nancy Messonnier, MD at the CDC for a full debriefing of the study to be included in the next IACC Summary of Advances in Autism Spectrum Disorder Research: Calendar Year 2016 that Dr. Thompson authored and the allegations of the link between autism and the MMR.

I request that the IACC demand retraction of published study (PubMed 2004 Feb;113(2):259-66.) at the AAP of the MMR/Autism paper co-authored by Dr. DeStefano and Dr. Thompson.

Note: Personally Identifiable Information (PII) has been redacted in this document

Jesse Ryan Bayer

April 26, 2017

Today I met a lady walking with her autistic son to the grocery store, while I was in baton rouge, Louisiana awaiting funds to travel to DC. I bless them with money for lunch. She asked if I was aware of autism, I said I heard of it, but didnt know much. So, as the uninformed owner of Bayer corporation, I would like to know what is available to help her and son.

If there is a treatment list I can put her kid on, let me know. I do know a healthcare company is hiding 40 billion dollars, which I intend to spend helping people access advanced cutting edge treatment or, cures available.

JESSE RYAN BAYER
[PII redacted]

Note: Personally Identifiable Information (PII) has been redacted in this document

Eileen Nicole Simon, PhD, RN

April 26, 2017

I hope my comments can be discussed, not just included in summaries of comments submitted. If brain damage is not important, please explain why.

Below are ideas I want discussed by members of the IACC, and to be included in the Public Record:

Eileen Nicole Simon, PhD, RN
[PII redacted] Cambridge MA [PII redacted]

1. Repetitious comments?

Dr. Daniels stated at the last meeting that too many public comments are duplications, that some of us come in person (or submit in writing) the same things multiple times. Then Dr. Battey stated that IACC committee members remember these comments; they don't need to hear the same thing more than once a year (transcript p185).

Yes! Since the IACC meeting in 2003, following the "Autism Summit Meeting," I have been trying to request discussion over and over and over again about the same few things:

- (1) Clamping the umbilical cord immediately after birth is a medical error.
- (2) Clamping the umbilical cord before the first breath can result in a lapse in respiration.
- (3) What is the danger when a baby doesn't breathe right away at birth?
- (4) Brain damage caused by asphyxiation of monkeys at birth was described in 1959.
- (5) Similar brain damage caused by asphyxiation of human infants has been described many times in the medical literature.

(6) Damage by asphyxia is most severe in acoustic relay nuclei of the brainstem auditory pathway.

(7) Damage by asphyxia is an ischemic variant of Wernicke's Encephalopathy, first described 136 years ago (Wernicke 1881).

(8) Traumatic injury of auditory nuclei in the midbrain results in loss of the ability to comprehend spoken language.

(9) Injury at birth of the same auditory nuclei in the midbrain should be investigated as the reason for impaired language development in autistic children.

I have said more about birth injury as a cause of autism, and I have spent money on bus or train transportation and hotel rooms to come to IACC meetings with the futile hope of engaging in discussion.

You may remember my comments, but your refusal to discuss them is pointedly unkind. I can only conclude there must be a need to avoid discussion of any possibility of medical error.

2. Discordant twins

The in-person comment by Patricia Swanson at the January 13 meeting was most interesting! She described her identical twin sons who are discordant for autism. She asked an important question about why discordant twins have not been sought out for research studies.

In my 50+ years of dealing with autism, I got to know one pair of identical twins who were both autistic. They were not discordant, but one twin was much more severely affected than the other. Both of these twins were also "hyperlexic;" they could read anything you gave them even though they did not seem to understand the content. These twins were on the children's unit with my son, at the Massachusetts Mental Health Center. One of them is a gifted artist at VinFen's Gateway Crafts workshop; I was told his brother has died.

Folstein and Rutter (*Journal of Child Psychology and Psychiatry* 1977; 30:405-416) reported on 21 twin pairs, of whom 11 pairs were identical. Concordance for autism was found in only 4 of the 11 pairs of identical twins. None of the fraternal twin pair were both autistic. Folstein and Rutter pointed out that in the discordant pairs, autism was associated with an event likely to cause brain damage.

In the concordant pairs, all were male, and complications in pregnancy were noted in each case. Three of the discordant pairs were female; one of these was a breech birth with delayed breathing, and her umbilical cord was described as very narrow and white (suggesting placental blood flow ceased before or during birth). I discussed Folstein and Rutter's cases further in chapter 7 of my ebook, *Autism and the Inferior Colliculus, Book 2: Language, Brain, and Iatrogenic Peril*, amazon.com.

I agree with Patricia Swanson that more research should be done on identical twins discordant for autism.

3. Autism Definition

Language disorder is no longer a criterion for diagnosis of autism, according to DSM-5. This seems clearly an effort to include other psychological problems in the autism diagnosis. Isn't this a coverup attempt to hide the horrific increase in severely neurologically impaired children over the past three decades?

Language development, repetitive movements, and diminished awareness are the three most serious problems of autistic children. Many are also afflicted with seizure disorders, a clear sign of disrupted brain activity.

"Autism" (used by Bleuler in 1911 to describe withdrawal in schizophrenia) was adopted by Kanner to describe children who displayed social detachment from early infancy. But Kanner's patients all suffered oddities in language development. Omission of developmental language disorder in DSM-5 discards the definition of Kanner-autism as an affliction evident from early childhood.

"Autism" that becomes evident later in childhood (or adolescence) is not part of a spectrum in which developmental language disorder is the most disabling feature. But wasn't the IACC intended to investigate reasons for the increase of disabling childhood autism that became evident in the early 1990s?

4. Autism Disabilities

Language is the distinguishing feature of the human species.

Language is the means by which inter-generational progress occurs.

Failure of language development is the most disabling handicap of autistic children.

Loss of language in adulthood (aphasia) is among the most serious of neurological disorders.

Aphasia is not secondary to loss of "shared attention" or social withdrawal.

Hand flapping, wide arm swinging, and jumping from foot to foot are signs of choreo-athetoid movement disorder. These repetitive movements should be compared with those observed in kernicterus (resulting from bilirubin damage of the basal ganglia).

Diminished awareness is a sign of diminished level of consciousness (LOC).

Explanations of how language problems are secondary to "social" disorder are not based on what is known about language circuits in the brain. Social circuits in the brain are glibly linked to face-recognition (fusiform gyrus), amygdala, oxytocin, and parental "autistic trait" genes.

Asphyxia at birth is a taboo subject, but the brainstem auditory pathway incurs damage from asphyxia. Hypersensitivity to ongoing noise from a vacuum cleaner or jack-hammer should be linked to impairment within the auditory system. Shouldn't impairment of auditory processing also merit consideration as an impediment for language development?

The auditory system is continuously active, even during sleep, and should be considered an important component of brain systems that underlie the conscious state. Delayed developmental

milestones and repetitive movements are evidence of impaired circuits for motor control (the basal ganglia).

5. Language Circuits

Failure of language development is the most serious handicap of autistic children. Circuits between the temporal and frontal lobe language areas may therefore be incomplete, or neurotransmitters in these circuits may be deficient. Maturation of the language areas is guided by trophic neurotransmitters produced in the brainstem auditory pathway [1].

Signal processing centers within the auditory pathway have been found to have greater metabolic activity than anywhere else in the brain [2]. These way-stations for detection and transmission of environmental sounds are thus more vulnerable to injury than other areas of the brain.

Asphyxia at birth damages tiny midbrain sites in the auditory pathway [3]. This damage was at first overlooked in monkeys subjected to asphyxia at birth, but maturation of the brain was clearly disrupted [4].

These midbrain acoustic signal processing centers are also prominently affected by exposure to toxic substances in the pattern of damage known as Wernicke's encephalopathy [5-7].

References:

- [1] Friauf E, Lohmann C. Development of auditory brainstem circuitry. Activity-dependent and activity-independent processes. *Cell Tissue Res.* 1999 Aug;297(2):187-95.
- [2] Sokoloff L et al. The [14C]deoxyglucose method for the measurement of local cerebral glucose utilization: theory, procedure, and normal values in the conscious and anesthetized albino rat. *J Neurochem.* 1977 May;28(5):897-916.
- [3] Ranck JB Jr, Windle WF. Brain damage in the monkey, macaca mulatta, by asphyxia neonatorum. *Exp Neurol.* 1959 Jun;1(2):130-54.
- [4] Faro MD, Windle WF. Transneuronal degeneration in brains of monkeys asphyxiated at birth. *Exp Neurol.* 1969 May;24(1):38-53.
- [5] Thomson AD et al. Wernicke's encephalopathy revisited. Translation of the case history section of the original manuscript by Carl Wernicke 'Lehrbuch der Gehirnkrankheiten für Aerzte and Studirende' (1881) with a commentary. *Alcohol Alcohol.* 2008 Mar-Apr;43(2):174-9.
- [6] Morgan DL et al. Neurotoxicity of carbonyl sulfide in F344 rats following inhalation exposure for up to 12 weeks. *Toxicol Appl Pharmacol.* 2004 Oct 15;200(2):131-45.
- [7] Cavanagh JB. Methyl bromide intoxication and acute **energy** deprivation syndromes. *Neuropathol Appl Neurobiol.* 1992 Dec;18(6):575-8.

6. Auditory Agnosia?

Sensory agnosia is described as reaching into your pocket but being unable to recognize which objects are your keys, coin purse, or dollar bills.

Isabelle Rapin pointed out that the language problems of some autistic children are the result of an "auditory agnosia" [1]. They are able to hear, but unable to identify distinctive components of language within streams of speech. Syllable boundaries appear to be among the components we become less able to hear as we outgrow the ability to learn a new language. Auditory agnosia creeps up on us as we age.

Auditory agnosia can strike unexpectedly following injury of the auditory pathway in the midbrain (tiny anatomical structures known as the inferior colliculi). Below are citations in the medical literature that describe this tragedy [2-15].

I will continue to point out that damage of these same tiny midbrain centers was found in monkeys subjected to asphyxia at birth. The protocol to clamp the umbilical cord immediately after birth can lead to asphyxia if the newborn infant does not breathe right away and needs resuscitation.

References:

- [1] Rapin I. Verbal auditory agnosia in children. *Dev Med Child Neurol*. 1988 Oct;30(5):685.
- [2] Joswig H, et al. Reversible pure word deafness due to inferior colliculi compression by a pineal germinoma in a young adult. *Clin Neurol Neurosurg*. 2015 Dec;139:62-5.
- [3] Poliva O, et al. Functional Mapping of the Human Auditory Cortex: fMRI Investigation of a Patient with Auditory Agnosia from Trauma to the Inferior Colliculus. *Cogn Behav Neurol*. 2015 Sep;28(3):160-80.
- [4] Kimiskidis VK, et al. Sensorineural hearing loss and word deafness caused by a mesencephalic lesion: clinicoelectrophysiologic correlations. *Otol Neurotol*. 2004 Mar;25(2):178-82.
- [5] Pan CL, et al. Auditory agnosia caused by a tectal germinoma. *Neurology*. 2004 Dec 28;63(12):2387-9.
- [6] Musiek FE, et al. Central deafness associated with a midbrain lesion. *J Am Acad Audiol*. 2004 Feb;15(2):133-51
- [7] Hoistad DL, Hain TC. Central hearing loss with a bilateral inferior colliculus lesion. *Audiol Neurootol*. 2003 Mar-Apr;8(2):111-3.
- [8] Vitte E, et al. Midbrain deafness with normal brainstem auditory evoked potentials. *Neurology*. 2002 Mar 26;58(6):970-3. (2 cases)
- [9] Masuda S, et al. Word deafness after resection of a pineal body tumor in the presence of normal wave latencies of the auditory brain stem response. *Ann Otol Rhinol Laryngol*. 2000 Dec;109(12 Pt 1):1107-12.
- [10] Johkura K. et al. Defective auditory recognition after small hemorrhage in the inferior colliculi. *J Neurol Sci*. 1998 Nov 26;161(1):91-6.
- [11] Hu CJ, Chan KY, Lin TJ, Hsiao SH, Chang YM, Sung SM. Traumatic brainstem deafness with normal brainstem auditory evoked potentials. *Neurology*. 1997 May;48(5):1448-51.
- [12] Meyer B, et al. Pure word deafness after resection of a tectal plate glioma with preservation of wave V of brain stem auditory evoked potentials. *J Neurol Neurosurg Psychiatry*. 1996 Oct;61(4):423-4. Free PMC Article
- [13] Nagao M. et al. Haemorrhage in the inferior colliculus. *Neuroradiology*. 1992;34(4):347.
- [14] Jani NN, et al. Deafness after bilateral midbrain contusion: a correlation of magnetic resonance imaging with auditory brain stem evoked responses. *Neurosurgery*. 1991 Jul;29(1):106-8; discussion 108-9.
- [15] Howe JR, Miller CA. Midbrain deafness following head injury. *Neurology*. 1975 Mar;25(3):286-9.

7. Speech Recognition?

"What exactly doesn't he hear?" someone in my writers' workshop asked about my autistic son.

I had just described the research by Roger Brown on child language development. He observed that children begin to speak using single words or word fragments. The word fragments are combinations of syllables. Young children seem to be able to easily distinguish syllable boundaries in the speech they hear around them.

The ability to hear syllable boundaries in rapid streams of speech appears to be lost during the first decade of life, which is why learning a foreign language is difficult already by adolescence.

"What is it we all fail to hear beyond early childhood?"

Learning a new language we often rely on phrase handbooks. When we use these phrases in a foreign country, we are doing what the echolalic autistic child does. Then we may have difficulty when a native speaker tries to clarify what we meant, and feel as socially inept as an autistic child.

Why are telephone conversations in a foreign language especially difficult, even terrifying? What are we not able to hear?

Speech recognition software is becoming more and more accurate. Can we learn what acoustic features must be detected? Can inability to detect these features be looked for in children and adults trying to learn a new language?

References:

- [1] Brown R. *A First Language: The Early Stages*. Cambridge, MA: Harvard University Press, 1973.
- [2] Kirchhoff K, Schimmel S. Statistical properties of infant-directed versus adult-directed speech: insights from speech recognition. *J Acoust Soc Am*. 2005 Apr;117(4 Pt 1):2238-46.

Lisa Bertone

April 26, 2017

What is being done about the crisis for adults with severe autism, who will outlive their parents, or their parents are too old to care for them & have little to no housing options or options in general. This will hit like a tsunami and resources, housing & higher pay for those who care for autistic adults are needed. Please see the attached link which writes about this crisis occurring right now in Staten Island NY. Thank you

http://www.silive.com/news/index.ssf/page/dignity_in_danger_developmenta.html

RESEARCH:

If research is based on constantly renewed alarm-ism, it is flawed and unethical.

It leads some forms of research down a narrow path only collecting statistics on what you expect to find, not considering greater complexities and overlaps.

Fear always closes the mind. Once something is labeled disorder you only go looking for disorder.

Externally observed Statistics will almost always be contradicted by lived experiences because they did not take enough variables into account.

Genetic research is not going to find much, we are 99% similar to apes and other life forms already. All it will eventually find is that we share the same variables as everyone else, only ours can have wider ranges or narrower ranges. There is no high or low, there is simply a long list of variables with their own ranges. It also can not predict brain adaptation.

Excluding Co-Occuring conditions, Neuroscience is the most ethical science to pursue so long as it remains simply about understanding the human brain. That benefits many conditions and injuries, it may benefit in education or other adaptive communication interfaces.

EDUCATION:

If a student is only interested in one class but does poorly in others, let them stick with what they're interested in and go for mastery instead of test scores. The idea that certain knowledge must be drilled in at a certain grade is flawed, as then most will only focus on getting good grades, not actually thinking about how to use that knowledge.

Listen to TED talks by Salman Khan(not the actor) and Jacob Barnett.

They're on the right track. The classroom environment is irrelevant to education, herding children like livestock just isn't a good idea.

College is at least about self-directed education, but only after a having the mind dulled.

Psychology/PTSD:

Psychological consequences are vastly underestimated. Both in language used and how you treat someone. Whether you think they are listening or not. This can greatly stifle development as well or lead to PTSD that will be a big problem for all involved.

If self worth is destroyed in the process, no treatment is going to matter. Psychological recovery may take decades if it ever arrives at all.

THERAPY/INTERVENTION:

When you condition a mind that requires intense focus and complex pattern recognition to learn to not use it, or do not allow the time for it to be used; you potentially stifle that mind from further growth.

Unresponsiveness is not a sign of not thinking. Drilling the same action over and over for hours, such as touching noses serves no true educational value. Some skills can develop more naturally at a different pace, some slower and some faster. Don't waste so much time on trivial drills and allow some actual self-directed activities to build on. Don't worry about making a child ready for a certain school at a certain age, if it is forced it is stifling.

There is usually a reason behind "unwillingness" whether it can be communicated or not.

Despite the fact low expectations are defied all the time, it draws in predatory marketing that promises there is hope only if you do this "intensive" program or treatment. Look to the first person officially diagnosed. Stop setting low expectations for brains you haven't even looked at. What is externally observable should not be considered a reliable indicator.

ORPHANAGE:

The simple truth is that some really only want "normal" kids, I would never advocate for an orphanage or child services unless it was designed by Autistic people. It is far better than filicide of unwanted children.

CHECKS AND BALANCES:

Since when does a big corporate CEO starting a charity get his organization recognized by government as a central authority on Autism issues world wide in record time just because of wealth and marketing influence? It was is that one group became the only government representation despite the fact there has been a rights movement that began in the 1980s.

This was not a democratic process, I certainly never voted to have someone direct government spending to research AWAY from Rights and Accommodations. This is precisely why the "support cliff" still exists, why there are not better more informed services and why a major UN topic was "basic human rights not being met" or "discrimination the rule, not the exception" a couple years ago.

I also didn't vote for them to represent me at the Vatican and convince the Pope to prioritize cures above all else. I do not recall the part where Jesus said "Let he who is unlike me cease to exist". Often how people treat you when they think you're suffering is worse than suffering itself.

Using language of fear such as "Kidnapped" "Burden" "Tsunami" "Epidemic" to keep the focus on research has resulted in enforced poverty for those blocked by lack of accommodations.

This is why doctors do not treat family members, emotional attachment clouds objectivity. The sense of urgency causes them to forget the consequences of their actions nor consider other possibilities.

Research agendas should always be based on actual needs, not alarm-ism set by a big businessmen.

Take measures to mitigate such impact from taking place again.

BACKGROUND/INSIGHT:

Delayed speech, moderately severe sensory, parents were told I'd never tie my shoes, etc. I did well in science and reading classes(only because I wanted to read comic books), but ultimately I came to a school that refused to follow my IEP back in the early 1990s, instead choosing to threaten and blame my parents. When the school did agree I needed "help", their solution was to "Warehouse" me in an empty room for the day with no teachers or classes.

I developed PTSD as a result of a build up of negative experiences and lack of accommodations. I was triggered by anything school related; including the sight of a school bus. I also developed intense self hatred and suicidal ideations, believing myself to be a burden to my parents. That is partly due to the fact my father was a very successful scientist until he and my mother had to go to war with the public education system trying to get them to uphold my rights. Because of my "self image", I lost my innate intense focusing abilities to a large degree. Even though I could speak, I did not have the language to communicate what was happening to me as my only frame of contextual reference was how "normal" children were treated by the school.

Then some years later in early 2000s, I inadvertently started using Intense Focusing and Pattern recognition but because it was unusual for me, I was loaded up with psyche meds and recommended for a group home. Nikola Tesla describes it best "I do not think there is any thrill that can go through the human heart like that felt by the inventor as he sees some creation of the brain unfolding to success... such emotions make a man forget food, sleep, friends, love, everything."

Then around 2007 I started hearing more and more people talking about the "epidemic" that was me. I took comfort in the fact there was a large charity working to prevent people like me from existing, as their language reinforced self-hatred to the point that I felt it was my moral duty to society to stop existing. Several years later I finally overcome all of that due to accidentally using Intense Focus and Pattern Recognition while thinking of a very complex simulation in my mind and charting notes on a whiteboard. I became far more articulate, able to communicate and self-advocate. But again, it was a mysterious change and as a precaution; more psyche meds. I ultimately realized that in all my efforts to condition myself to "socialize" and "blend in"; that I had conditioned myself not to even use my innate abilities. Trying to boil complex designs into general language always lacks context and attention to detail, including this. Now I focus on taking care of both of my increasingly disabled parents.

I survived, but at a cost so high that others do not survive intact if at all. I see too many falling into the same traps, patterns upon patterns of preventable losses. I see the same and much worse happening. Things that would be illegal if Rights had been a focus years ago. Too many are trapped in poverty because the alarm at the sight of disabled children causes you to forget that we grow up to be adults that will mostly just want the same opportunities to succeed but may need better accommodations to do that.

Good luck.

Marian Dar

April 26, 2017

Thought you might want to know about this upcoming event. Attached are a flyer and tech specs for group and/or individual links. Please forward the information to family, friends and community members you think may be interested.

Please understand, the April 26 autism symposium that I mentioned in my email is *an initiative of and collaboration between a private party (family) in lower Ct (the Hilibrands) and a religious non-profit. (UJA, NY).*

In NW Ct (Southbury) we are only marketing and offering a venue/satellite viewing of the program -- to increase awareness and attendance for the event locally.

I have sent our flyer to more distant family, friends and communities to increase exposure and suggest that they consider setting up local venues also, or view at home if that is preferable, so more are able to benefit from and learn more about the issue and opportunities.

Also, possibly seeing some tangible and worthwhile product like this might spur others to attempt the same.

Good viewing!

JEWISH FEDERATION of WESTERN CONNECTICUT
and Brownstein Jewish Family Service
invite you to join us as we livestream from UJA Federation, NY
10th Annual Hilibrand
AUTISM SYMPOSIUM
“Fostering Independence for
Young Adults with Autism Spectrum Disorder”
A Focus on Self-Advocacy, Housing and Entrepreneurship

Wednesday, April 26 from 8:30am–4:00pm
Harry and Jeanette Weinberg Community Function Hall
444 Main Street North, Southbury, CT 06488

SPEAKERS INCLUDE:
Patrick Bardsley, Spectrum Designs Foundation, NY
Retail operation, manufacturer
Dr Judah Koller, PsyD, Hebrew University, Jerusalem
“Early symptoms, signs and intervention...”
Dr. Kathy Matthews, Faison Center, Richmond;
Kennedy Kreiger, Baltimore
Lifespan services for individuals and families
Marjorie Madfssis
“Yes She Can...”
Jesse Saperstein
Self-Advocate, Transition to Adulthood
Jonah Zimiles

Bookstore owner and vocational work site

Parents, professionals, advocates and other interested community members are encouraged to attend and learn more about the latest research and innovative program models. Light refreshments will be provided.

CO-CHAIRS: Marian Dar, Meredith Hettler B-JFS

Kindly RSVP by Monday, April 24th

203-267-3177, ext. 340 or RSVP@Jfed.net

www.jfed.net

Instructions for Organizations/Communities Presenting a Webinar for:

The 10^h Annual

UJA-Federation of New York Hilibrand Autism Symposium

April 26, 2017

For more information about the conference, please visit our online event program description:

<http://www.ujafedny.org/event/view/annual-hilibrand-autism-symposium>

To stream the 10th Annual UJA-Federation of New York Hilibrand Autism Symposium, point your browser to the following URL:

Please note the page will not be live until the morning of the symposium.

www.ujafedny.org/hilibrand-symposium-livestream/

The conference begins with registration at 8.30 a.m. and lasts till 4.30 p.m. (EST)

If you go to the URL address now, you will find links to more information about the symposium. The streaming will be live on the day of the conference.

If you are watching the event as part of a bigger group: You will also need a projector, an adaptor (from computer to projector), a screen and speakers in order to project this in a large format for all individuals to see at your organization.

If you are watching the event on your own computer: You can just go to the live stream sight and type in the URL.

PLEASE NOTE: As we are creating a master list of partners who are streaming for the day, please confirm your participation by emailing Beth Rosenberg at bbethr@gmail.com if you would like your organization to be included. For the latest updates on the conference please make sure to email Beth so that she knows your community is participating.

If you have specific problems on the day of the event, please email bbethr@gmail.com.

Minimum Technical Requirements

Browser

Adobe Flash Player 10.0.32 or above

Internet Explorer 8.0 or above, Firefox 2.0 or above, or Safari 3.0, or Chrome 4.0 or above

Microsoft Windows XP SP2, Microsoft Windows Vista, Macintosh OS X v10.4 or above, or Linux

256 megabytes (MB) of RAM - 512 MB recommended

JavaScript and Cookies must also be enabled.

Super VGA (800 x 600) or higher resolution

16-bit sound card

Speakers/headphones

Internet Connection Speed

For a reliable viewing experience at medium quality we recommend a downstream connection speed of at least 700Kbps.

Viewers can select a higher (up to 2Mbps for HD) or lower (198Kbps for low) video quality on the player depending on the connection speed.

You can test your connection speed [here](#) -- we recommend testing several times as bandwidth can fluctuate.

Sincerely,
Marian Dar

Maurine Meleck

April 26, 2017

STOP the autism epidemic. Stop vaccines.

April 18, 2017

Thank you for the opportunity to submit written comments.

The Autistic Self Advocacy Network (ASAN) remains deeply concerned about the allocation of autism research funding in the federal government and the overall direction of autism research. The latest data available, from the Office of Autism Research Coordination and IACC in 2012, found that only a tiny fraction of total autism funding from the National Institutes of Health (NIH) went to research on service effectiveness (2%) and even less (only 1% of the total) went to research on outcomes across the lifespan. Research on autism is still disproportionately focused on early diagnosis and intervention, biology, and medical treatment of autistic traits. 55% of NIH research funding went to research on the biology of autism or to the causation of autism.

While we appreciate the increased interest in health and long-term outcomes for autistic people reflected in the IACC's 2015 Summary of Advances and 2013 Strategic Plan, that interest will not bear fruit as long as research funding for these subjects is limited. ASAN is committed to working with federal agencies to ensure that their research gathers information that will lead to a better quality of life for autistic people.

Lifespan and Quality of Life

Research being done on autism across the lifespan is still woefully insufficient and limited, especially research on the employment practices, services, health care, and supports that help us manage any co-occurring conditions that improve our quality of life. This is true despite the results of recent studies which have consistently found that autistic people have a much higher mortality rate than the general population and are at higher risk for suicide and injury.¹ There is an urgent need for research that illustrates ways to address the discrepancy.

In its 2015 Summary of Advances, the IACC highlighted a study on autism and health which found that autistic adults have higher rates of seizures, obesity, sleep disorders, high blood pressure, and many different mental health conditions including depression, anxiety, and schizophrenia.² Given this, it is crucially important that more research be done on how autism interacts with other conditions, and which forms of preventative and behavioral health care best help an autistic person in managing co-occurring conditions.

¹ Ann Griswold, "Large Swedish study ties autism to early death," Spectrum News, December 11, 2015, <https://spectrumnews.org/news/large-swedish-study-ties-autism-to-early-death/>; Susan Scutti, "Children with autism 40 times more likely to die from injury, study says," CNN, March 21 2017, <http://www.cnn.com/2017/03/21/health/autism-injury-deaths-study/index.html>; Autistica, Personal tragedies, public crisis: The urgent need for a national response (last visited March 20, 2017) (2016): <https://www.autistica.org.uk/wp-content/uploads/2016/03/Personal-tragedies-public-crisis.pdf>.

² Croen L.A, Zerbo O., Qian Y., Massolo M.L, Rich S., Sidney S., Kripke C., The health status of adults on the autism spectrum, *Autism*. 2015 Oct; 19(7):814-23.

The IACC should also invest in research that examine the accessibility of health care. Many health clinics, hospitals, and community health centers are cognitively and/or physically inaccessible to autistic people. Many could be made accessible with minor modifications or reasonable accommodations that could be determined through research on the accessibility of health care.

Some studies hypothesized that a lack of *disability-competent* primary and preventive care could be responsible for increased mortality. There are very few primary care providers that are able to understand and effectively treat persons with complex medical conditions, mental health conditions, and developmental disabilities. Many doctors also have pre-existing biases about the capabilities of people with developmental disabilities. The IACC could potentially look at these barriers to disability-competent health care and at the best strategies for reducing these barriers.

Future research should also examine the interactions between autism and the medications ordinarily used for any of our commonly co-occurring conditions. Many autistic people have reported to us that they experienced unexpected adverse effects from the medications they took to address their physical and mental health conditions.

Supports and Services

The 2012 data indicates that research into new support services and service effectiveness continues to be low-priority. What little research does exist appears to prioritize early interventions in autistic infants and very young children rather than the effectiveness of support services for school-age autistic children, transition-aged youth, and autistic adults (as suggested by the IACC's 2013 Strategic Plan and as indicated by the articles it selected in its 2015 Summary of Advances). We want the focus of autism research to broaden to supports and services that help autistic people of all ages integrate into our communities.

Autism research should strive to determine what can aid autistic people alive today, rather than what causes autism or how to eliminate autism before it appears.

We approve of the IACC's interest in the successful employment of autistic adults and their transitions to adulthood, as shown by the 2013 Strategic Plan and the 2015 Summary of Advances. However, existing studies focus on the "cost" of employing an autistic person and unnecessarily reexamine what we know to be true: that autistic people are much less likely to be employed or engaged in post-secondary education. More research needs to be done on vocational rehabilitation and supported employment services that give autistic adults access to competitive integrated employment in our communities.

In addition, we urge the IACC to encourage research into the assistive technology used by autistic people and what types of technology are most effective for us. Many autistic people rely on text-to-speech devices and other forms of AAC to communicate, but little research has been funded regarding assistive technology for communication. In fact, many autistic people have been denied access to the communication technologies that work best for them in certain contexts (such as schools) *because of* the lack of a research base on the communication form.

Beyond communication, many autistic people use assistive technology such as specialized timers, apps and online programs to support executive function, memory, travel and navigation, independent living, decision making, community integration, education, and employment. Research examining the various

uses of these technologies and how to improve them could potentially benefit all autistic people. We could also benefit a great deal from research that examines how to make commonly-used internet services (such as Google Suite and Facebook) more cognitively accessible to people with disabilities. Research on this subject could potentially connect many more people with developmental disabilities with the wider social world.

Diagnostic Disparities and Prevalence

African-American and Hispanic autistic children continue to go under-diagnosed, as do girls on the spectrum. In addition, girls who receive an ASD diagnosis are more likely than boys to have been identified as having an additional disability, and children of color diagnosed with ASD are more likely than white children to have been identified as having an additional disability. This suggests that there are many undiagnosed autistic girls and autistic children of color who are currently either assigned a different disability level or who are being missed entirely due to not being male and white.

More research is needed to determine what factors are causing these diagnostic disparities, identify methods of combating bias, and to identify and develop diagnostic best practices which increase accuracy and ensure that all children are able to exercise their rights and access needed services. Research is also needed to develop effective diagnostic practices and tools for autistic adolescents and adults, many of whom go undiagnosed because clinicians are not trained in identifying autism in these populations.

Proposed Changes to the IACC Strategic Plan

ASAN continues to have profound concerns about the Questions the IACC uses to direct its research prioritization under its Strategic Plans. The current Questions (as reflected in IACC's 2013 Strategic Plan) are unnecessarily and excessively focused on the prevention and potential causes of autism. As part of its efforts to set the agenda for federal autism research, we encourage the IACC to revise the Questions used to direct research goals in the Strategic Plan. The new Questions should reflect the increasing scientific consensus that autism cannot and should not be "cured" or "prevented." Instead, the Questions should emphasize the need for research into: the services and supports that best enable autistic people to live self-determined lives; autism across the lifespan; co-occurring medical conditions and access to equitable medical care; identifying and addressing disparities within the autistic community; and other priorities identified in conjunction with the autistic community.

Question 3 should be limited to the "What Caused This to Happen?" component of the question, which should be changed to: "What Environmental Factors Have an Impact on our Quality of Life?" Question 3's "What Caused This to Happen?" component primarily focuses on environmental conditions more likely to "cause autism," but we find this research does not help autistic people. Instead, the IACC should fund research into the aspects of an autistic person's environment and society that may lead to physical or psychological harm to the autistic person.

There is no need for a separate Question component on prevention when Question 2 already addresses neurological development in autism. Attempting to research how to "prevent" autism devalues the lives of autistic people and has not led to improvements in our quality of life. Questions 4 and 5 should remain the same, but should be redirected towards the development of services and supports that aid autistic people across the lifespan. These questions are currently focused on interventions that attempt

to “reduce” characteristics of autism or eliminate autistic traits in the very young, rather than the supports many autistic people of all ages might need to live full lives.

We agree with the goals of Question 6 but believe that they should not be addressed in a single question. Lifespan issues for autistic people should be included as part of all the Questions IACC investigates. Lifespan issues, such as quality preventative and behavioral care for autistic people at all ages, supports and services, and employment issues, are the most pressing subjects of interest to autistic people.

Question 6 may be more effective if split into three Questions or sub-Questions: one on employment outcomes, one on physical and behavioral health, and one on population-level longitudinal studies of autistic adults, as exist for adults with other developmental disabilities.

Working with the Autistic Community

We implore the IACC to promote research into the issues that are important to the stakeholders most affected: the autistic community. The autistic community is asking for research into the issues that many of us face, including motor planning difficulties, skill loss, struggles with nutrition, atypical response to medications, unusual pain perception, and difficulty with executive functioning. Additionally, the experiences of autistic people during common life events such as pregnancy, parenting, and aging have not been studied. Many autistic people have asked us for such information in order to understand what to expect as they grow older or start a family, but little to no research is available.

The allocation of autism research funding has a real impact on the lives of autistic people. For that reason, ASAN believes that this funding should be allocated with the input and involvement of the autistic community. We urge the IACC to promote the involvement of autistic adults in grant review and other aspects of the research process, including through the use of Participatory Action Research models.

Again, ASAN appreciates the opportunity to provide comments on the important issue of autism research. For more information on our comments, please contact Julia Bascom, Executive Director of ASAN, at jbascom@autisticadvocacy.org.

Written Testimony
Provided by Lisa Wiederlight, Executive Director, SafeMinds
April 18, 2017

Dear Dr. Gordon:

Please find below my written testimony, on behalf of SafeMinds, a national 501 (c)3 organization based in Baltimore. A group of parents dedicated to identifying the environmental causes of the autism epidemic started our organization 17 years ago, and we remain steadfast in our resolve to promote environmental causation research, and to increase the safety, health, and independence of people with autism.

In a recent interview, Dr. Joshua Gordon stated that it is the IACC's priority to ensure that "the federal effort on autism accounts for the needs of individuals with autism and their families." The IACC has not achieved this, as even the most basic human need—personal safety—is the largest concern the autism community faces.

Two reports in 2017 have highlighted the increased mortality of people with autism:

The Swedish study cited by the publication *Autistica* found that people with autism die on average 18 years earlier than the general population; and that those with autism and learning disabilities die more than 30 years prematurely.

The Columbia University study featured in the *American Journal of Public Health* found that "Individuals with autism appear to be at substantially heightened risk for death from injury."

Injuries can be prevented. Rather than talking solely about research that may or may not find answers that will help future generations, SafeMinds is asking the IACC to prioritize addressing the documented increase in mortality for persons with autism.

This national crisis must be addressed by research, education, training, and evaluation of outcomes. It will require the active support of legislation, such as Kevin and Avonte's Law to prevent and address wandering in people with developmental disabilities. The autism community is clamoring for urgent action. There is no better reason to seek actionable answers. Fewer people with autism will die. The lifespan of people with autism will increase. The number of families who will suffer from preventable tragedies will decrease.

Sincerely,

Lisa Wiederlight

Gail Elbek

April 26, 2017

Interesting...as you know while U.S. allowed to saturate fetus, infants and children contains several estrogenic hormone disruptors! It is truly unforgivable that NIMH ignores hundreds of soy studies confirming the soy photo-poisoning of developing body and brain!

Why! Why do you care nothing of hundreds of valid soy study facts?

Share the toxic soy studies. How many more hundreds is NIMH waiting for?

[Http://illegalfda.blogspot.com](http://illegalfda.blogspot.com)

I will appreciate your reply,

Gail Elbek

On Apr 19, 2017, at 1:21 PM, National Institute of Mental Health <nimh@public.govdelivery.com> wrote:

[Estrogen Alters Memory Circuit Function in Women with Gene Variant](#)

Hormone-gene interaction may underlie sex/individual differences in mental disorders

Fluctuations in estrogen can trigger atypical functioning in a key brain memory circuit in women with a common version of a gene, NIMH scientists have discovered. Brain scans revealed altered circuit activity linked to changes in the sex hormone in women with the gene variant while they performed a working memory task.

[Read More >>](#)

Carol P. Fedorchak

April 26, 2017

I have a son with Asperger's Syndrome. He is 16 and is in a public high school where few staff understand his problems . He recently has had a breakdown and has been in a hospital and now is home going to school half days and an Intensive Hospital Program in the afternoon . He is exhausted all the time and has started refusing to attend school. He isn't eating well and that is causing health problems I tried before high school to have him placed in a specialized program to have his special needs met but the school district won't accept his case and pay dollars to educate him as well as he needs. He is very bright ,has abilities(plays music in school band) but socialization is and has been difficult for him. I am a single Mother and need help and support with him.