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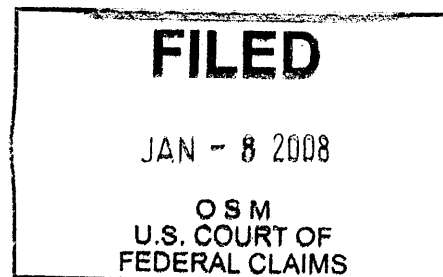
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November 30, 2007

ORIGINAL



VIA UPS DELIVERY

Clerk
United States Court of Federal Claims
717 Madison Place, NW
Washington, D.C. 20005

Re: In Re: Claims for Vaccine Injuries Resulting in Autism Spectrum Disorder, or a Similar
Neurodevelopmental Disorder v. Secretary of Health And Human Services
Autism Master File
Our File No. 054500 - Omnibus Autism Proceeding

Dear Clerk of Court:

Enclosed for filing in the case file captioned above is the original and two copies of the medical expert report of petitioners' expert Elizabeth A. Mumper, M.D. in the compensation claim of Alexander Krakow. This report is submitted both in support of petitioners' theory that exposure to the mercury contained in certain pediatric vaccines was a substantial contributing cause of some or all of the injuries at issue in the Omnibus Autism Proceeding, and in support of Alexander's individual claim. Petitioners anticipate relying on this testimony in hearings on "test cases" currently scheduled for May 2008.

A copy of this expert report is simultaneously being filed in Krakow v. Secretary of Health and Human Services, Case No. 1:03-00632-UNJ.

Very truly yours,

A handwritten signature in black ink, appearing to read "T. B. Powers". The signature is fluid and cursive, with a long horizontal stroke at the end.

Thomas B. Powers
Attorney at Law

Enclosures

cc: John Fabry, Esq., Williams Kherkher Hart Boundas, LLP (via UPS and email)
Vincent J. Matanoski, Esq., U.S. Department of Justice (via UPS and email)
Special Master George Hastings, US Court of Federal Claims (via UPS and email)
Special Master Denise Vowell, US Court of Federal Claims (via UPS and email)
Special Master Patricia Campbell-Smith, US Court Federal Claims (via UPS and email)
Staff Attorney, Joseph Lowe, US Court of Federal Claims (via email)

Professional opinion about the role of thimerosal containing vaccines In the case of Alexander Krakow

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Qualifications:

Background and Qualifications:

MD 1980 Medical College of Virginia
Internship in Pediatrics 1980-81, University of Massachusetts
Residency in Pediatrics 1981-83, University of Virginia
Chief Residency in Pediatrics 1983-84, University of Virginia
Private Practice Pediatrics 1984-1989, Lynchburg, VA
Director of Pediatric Education 1989-2000, Lynchburg Family Practice Residency
CEO, Advocates for Children, 2000-present
Medical Director, Autism Research Institute, 2005 – present
Founder, RIMLAND Center, 2007

Experience in Treating Children with Autism Spectrum Disorders

During my pediatrics residency I received the usual and customary training in how to care for children with neurodevelopmental disorders. Since developmental and behavioral problems were a special interest of mine, I attracted a population of such patients during my time in private practice and while serving as Director of Pediatric Education at a University of Virginia affiliated family practice residency program.

In 2000, I established Advocates for Children to help meet what I perceived as unmet needs in my community for the increasing numbers of children with neurodevelopmental and behavioral disorders. We have just opened The RIMLAND Center, which will serve as a training facility for clinicians interested in learning about the medical problems of children with autism. We have over 3000 patients, and care for children not only in central Virginia, but also from other states and countries. At least 500 of our patients have autism spectrum disorders and other neurodevelopmental disabilities. A large proportion of our general pediatric patients have chronic diseases.

Krakow - case summary:

- During the first 18 months of life, Alex appeared to be developing normally as documented in the medical records. He was noted to have ~10 words by 16-18 months of age.
- His medical problems included mild valvular pulmonic stenosis diagnosed in early infancy, with reassuring echocardiogram at day 6 of life, and follow-up at 18 months by cardiology with diagnosis of “very mild valvular pulmonic stenosis, otherwise good health”
- Allergies were first suspected at 6 months of life
- He had at least 5 courses of antibiotics prior to age 2 years, given for ear infections and protracted upper respiratory infections
- He was seen several times for loose and/or foul smelling stools and/or acute gastroenteritis prior to the age of 2 years
- At his 2-year check up, there was concern that his speech was not progressing, and that he had lost some words. Hearing was tested soon after and documented to be normal.
- He received flu shots in November 2001 and December 2001, and soon after was noted to have red fingers, act listless, and develop hypotonia. He lost speech completely and was unable to grip items as before.
- During the 6 months after his 2-year birthday, parents noted gaze aversion and unresponsiveness when his name was called. They consulted Dr. Marvin Boris in June 2002, who began investigating possible reasons for the developmental and speech regressions and recommended therapeutic strategies to deal with his medical and developmental problems.

Summary of expert medical opinion about the case of Krakow

- My professional opinions in this case are based on a careful review of Alexander Krakow’s complete medical records, my clinical experience with the medical problems associated with regressive autism, my role teaching doctors how to evaluate and treat children with autism, and my extensive review of the medical literature with regards to regressive autism, mercury toxicity, and the interaction between genetic predispositions and environmental factors.
- My review of the literature, clinical experiences with hundreds of children with regressive autism and conversations with researchers as a result of my role at the Autism Research Institute have led me to agree with the expert reports about causation prepared by Dr. Vas Aposhian, Dr. Richard Deth and Dr. Sander Greenland in this case.
- In my best professional judgment, with a reasonable degree of medical certainty, taking into account the specific medical facts about this particular child and applying my general knowledge obtained as described above, it is more likely than not that the thimerosal in the childhood

vaccines Alexander Krakow received was a substantial contributing factor to his neurodevelopmental problems.

Analysis of specific facts in the case of Alex Krakow with regard to thimerosal and its effects:

Thimerosal exposure:

- Based on my review of this patient's immunizations records, and given the limitations that some of the dates on my copy were difficult to read, it appears that Alex received 212.5 mcg of ethyl mercury by the age of 2 years via thimerosal containing vaccines given according to the usual and customary schedule
- He received his first dose of Hepatitis B vaccine containing thimerosal at birth
- He received two additional 25 mcg of ethyl mercury via doses of thimerosal containing flu vaccine in November 2001 and December 2001
- The combination of thimerosal containing vaccines, including the flu vaccines, resulted in a cumulative ethyl mercury exposure of approximately 262.5 mcg by the time Alex was 25 months old

In my best medical judgment, review of Alex Krakow's medical case is consistent with an increased vulnerability to the toxic effects of thimerosal exacerbated by co-existing antibiotic use.

Clinical evidence:

- Alex had received at least 5 courses of antibiotics during the first 2 years of life.
- He had several documented episodes of diarrhea in proximity to the administration of thimerosal containing vaccines

In addition, we have evidence that Alex Krakow had impaired methylation biochemistry and deficient detoxification ability with improvements in clinical status when his methylation biochemistry, cellular redox status and detoxification systems were improved.

Laboratory evidence of impairments in detoxification and impact of mercury on Alex's physiology:

- A thiol profile documented adenosine of 0.563, S-adenosine-homocysteine of 10.35, cysteine of 134.85, methionine of 23.35, and glutathione of 4.36. Thiol biochemistry is crucial for the body to handle heavy metal exposures; poor thiol biochemistry would be expected to impair the child's ability to handle mercury and other heavy metals.

- Alex was demonstrated to be heterozygous for 2 mutations (C677T and A 1298-C) in the methyltetrahydrofolate reductase enzyme, which is important in methylation biochemistry functioning, and could be a contributing factor to difficulty handling mercury exposures via thimerosal containing vaccines.
- A porphyrin profile obtained on August 9, 2006, after 3 years of interventions designed to improve his detoxification abilities and remove mercury and other heavy metals from his system, demonstrated a pattern interpreted by the lab as “moderate mercury toxic effect.” At a time when the normals for precoproporphyrin were 2-5 nmol/gram of creatinine, the patient demonstrated a precoproporphyrin of 22 nmol with urine creatine of 649. Precoproporphyrin is a marker specific for mercury; no other substance is known to cause an elevation.

Analysis of this case in relation to the literature:

- Infants are born at risk: 1 in 6 children born today is predicted to have blood levels of mercury high enough to impair neurological development (Stern 2005, Ref. # 0131¹).
- Antibiotics potentiate mercury toxicity (Rowland, 1984, Ref. # 0187)
- Mercury has myriad manifestations of toxicity: Mercury is the classic prototype demonstrating the ability of heavy metals to have myriad manifestations of toxicity depending on the biochemical individuality of the victim, route of exposure, dose effects and synergistic toxicities (Blaxill, Redwood et al. 2004, Ref. # 0259).
- Normal infants immunized per routine recommendations can meet criteria for acute mercury toxicity: The CDC has defined mercury poisoning as a blood mercury level greater than 10 mcg/L (2005). The Stajich study looked at normal infants after hepatitis B vaccination. One infant developed a post vaccine mercury level of 23.6 mcg/L, which meets CDC criteria to qualify as a case of acute mercury poisoning (Stajich, Lopez et al. 2000, Ref. # 0249). The presence of such high blood levels is consistent with significant inter-individual variability.

Clinical evidence compatible with damage from mercury:

- Mercury is an immune disrupter, upsetting the balance between TH1 immunity, which defends the body against bacteria, viruses and fungal infections and TH2 immunity. Alex has clinical documentation of food allergies and low levels of immunoglobulins.
- Mercury has been documented to induce autoimmunity. Alex developed several different autoimmune diseases. He developed autoimmune

¹ All reference numbers are taken from Petitioners’ Master Reference List of Medical Literature, Nov. 2007. CD-ROMS containing copies of all articles with a linked index are on file with the court. A copy of the list is attached to this report.

thyroiditis, for which he had to be supplemented with prescription thyroid replacement. The thyroid gland is particularly susceptible to thyroid damage. He demonstrated an autoimmune response to his own myelin, the nerve sheaths that are crucial for processing neurologic messages.

- On November 20, 2002, Dr. Krigsman reviewed the results of Alex's intestinal biopsies with the parents. He demonstrated multiple reactive hyperplastic lymphoid follicles in the ileal mucosa and nodular or diffuse inflammatory infiltrates of lymphocytes in the colon. His gastric and esophageal mucosa were unremarkable. Immunohistochemical studies of this pathology are consistent with an autoimmune process.
- Mercury has been documented to damage DPPIV (dipeptidyl peptidase), which is a digestive enzyme responsible for breaking down gluten (in wheat products) and casein (in dairy products). Alex had documented laboratory evidence of intolerance to casein, and evidence of clinical improvements on the gluten free casein free diet. In the absence of other reasons, such as celiac disease, to explain his intolerance to gluten and casein, my best medical judgment is that his mercury exposure was a substantial contributing factor.
- The clinical picture described in which Alex had red fingers, listlessness, new onset hypotonia, unresponsiveness to name, loss of previous ability to grip and complete loss of speech is consistent with direct neurotoxic effects of mercury and consistent with classic descriptions of mercury toxicity.
- Dr. Boris documented by physical exam his hypotonia and diagnosed chronic inflammatory neuropathy, which is consistent with the effects of mercury on the developing nervous system as documented in the literature cited in this report.
- When nutritional and medical interventions directed towards enhancing his body's detoxification pathways were undertaken, these symptoms improved.

Clinical evidence of improvement with medical treatments, including those directed at removing mercury and improving the body's natural detoxification and immune mechanisms:

- When Dr. Boris evaluated Alex in June of 2002 and began treating him with interventions designed to improve his detoxification pathways and treat his medical problems, improvements in his developmental status was documented in sequential office notes. Specifically, Dr. Boris tracked improvements in behavior, social interactions and language over time.
- Alex showed improvements in GI and developmental status when begun on the gluten free casein free diet.
- Dr. Boris prescribed methylcobalamin injections, designed to improve methylation biochemistry and parents reported behavioral improvements in his behavioral, social and speech profile noted on many sequential office visits.

- On October 10, 2002, Dr. Boris prescribed chemet (DMSA, a chelating agent approved by the FDA for treating lead toxicity and demonstrated in Dr. Nataf's studies to lead to improvement in porphyrin profiles of children with documented mercury toxicity). He documented in his medical records that he discussed the mercury protocol with the family.
- On April 27, 2003, evaluation of the urine for toxic metals showed extreme excretion of mercury (15 with normal unprovoked <3) and lead (48 with normal unprovoked <15), providing evidence of excretion of mercury in response to therapeutic strategies being used by Dr. Boris.

Laboratory evidence of immune dysregulation, endocrine disruption and autoimmunity:

- Anti-myelin antibodies: CNS myelin IgG titers 1:32 (titers <1:8 expected), CNS myelin IgA titers 1:64 (titers < 1:8 expected). This is compatible with a toxic encephalopathy.
- Positive myelin basic protein antibodies reported by Dr. Boris compatible with autoimmune neuropathy
- Autoantibodies to thyroid: antimicrosomal antibodies 1:1600 (normal <1:100) and antithyroglobulin antibodies 1:400 (normal < 1:100). This is a dramatic production of autoantibodies against a vital gland to regulate metabolism.
- Thyroid peroxisomal antibodies 12.4 (normal <2)
- Thyroid stimulating hormone elevated on several occasions. Patient required treatment with thyroid replacement.
- IgG level low at 635 (normal 673-1734) on one occasion and 605 on another. At other times, was low on certain IgG subsets.

Analysis of Alex's clinical and laboratory evidence with regard to the medical literature.

- Intermittent larger doses of mercury as given in vaccine injections bypass the normal protective mechanisms found in the gut that are designed to protect against oral exposures. Children have not had the opportunity to evolve mechanisms to protect against injected ethylmercury. My best medical judgment, based on clinical experience and studying the medical literature is that injected thimerosal in bolus doses is associated with more risk of toxicity than a chronic low-dose daily intake of oral mercury (Grandjean and Jorgensen 2005, Ref # 0210).
- Numerous mechanisms of thimerosal toxicity have been demonstrated Thimerosal is metabolized to thiosalicylate and ethylmercury, which is taken up by organs and degraded to Hg²⁺ (Qvarnstrom, Lambertsson et

al. 2003, Ref. # 0246). Thimerosal is documented to cause DNA damage (Baskin, Ngo et al. 2003, Ref. # 0253) and inhibit mononuclear phagocytosis (Rampersad, 2005, Ref. # 0211).

- Pathologic brain injury has been documented in response to thimerosal. Subclinical mercury poisoning induced experimentally in monkeys (levels less than 50 mcg mercury/kg body weight/day) demonstrated pathologic brain changes including decreased numbers of astrocytes and increased activated microglia without any noticeable clinical manifestations (Charleston, Body et al. 1996, ref. # 0116). Recent autopsy studies of autistic brains demonstrated activation of microglia and the innate immune system, but not adaptive immunity. Burbacher demonstrated in primates that injected organic mercury was associated with persistence of inorganic mercury in the brain (Burbacher, Shen et al. 2005, Ref. # 0251)
- Patients with autism have been demonstrated to have increased oxidative stress [James, 2004, Ref. # 0005]. "Oxidative stress (endogenous or environmental) may contribute to the development and clinical manifestations of autism" [James, 2006, Ref. # 0049]. Alex had this testing done and treatments directed to improving his oxidative stress and disordered thiol biochemistry.
- Metabolic perturbations are very common in children affected by the recent autism epidemic. James and colleagues described fundamental abnormalities in methylation and transulfuration biochemistry in autistic children when compared to neurotypical control children. Autistic children had low methionine, low cysteine, low reduced glutathione, increased oxidized glutathione, and abnormal redox ratios. Normalization of the redox ratios occurred with nutritional supplementation, including methylcobalamin, betaine and folinic acid (James, Cutler et al. 2004, Ref. # 0005). These findings were later confirmed in a larger cohort of autistic children compared to neurotypical children (James, Melnyk et al. 2006, Ref. # 0049). My clinical experience has validated the methylation and transulfuration abnormalities described by Dr. James, and our patients demonstrate clinical improvements when we utilize strategies to support methylation biochemistry, as occurred in Alex.
- Glutathione deficiencies impair ability to excrete thimerosal. Impaired methylation biochemistry leads to glutathione deficiencies, which are present in the vast majority of our autistic patients and over 75% of autistic children (James, Cutler et al. 2004, Ref. # 0005). Since glutathione is such a crucial intracellular anti-oxidant, has vital roles for detoxification function, modulates T cell function, and helps regenerate intestinal epithelium, treatment strategies designed to normalize the ratio of reduced to oxidized glutathione often lead to clinical improvements, and was part of Alex's therapeutic regimen.

- Autistic children demonstrate mercury toxicity. One recent prospective study of 115 children with autism demonstrated porphyrinuria when compared to 119 control children [Nataf et al., 2006, Ref. # 0065]. When compared to the control group, children with autism had a mean increase of 2.6-fold ($p < 0.001$) in urinary coproporphyrin. A subgroup of these autistic children underwent oral chelation therapy with DMSA which resulted in a significant reduction in mean urinary coproporphyrin and precoproporphyrin ($p = 0.002$), indicating that the urinary porphyrin elevation was not genetic in nature but due to the toxic metals removed [Nataf, 2006, Ref. # 0065]. This test was not widely used in the autism community at the time Alex began treatments for the mercury toxicity diagnosed by Dr. Boris, so we do not have the advantage of knowing what the impact of mercury toxicity on his porphyrin pathways was prior to chelation.
- Abnormal immune responses to dietary proteins and brain cells: Vojdani et al. demonstrated immune responses to dietary proteins, gliadin, and cerebellum peptides in children with autism. A sub-group of patients with autism produced antibodies against Purkinje cells and gliadin peptides, providing further evidence of a link between the gut, brain and immune system (Vojdani, O'Bryan et al. 2004, Ref. # 0094). Alex's intestinal problems and improvements when he was begun on gluten free casein free diet raise concern that he is in this clinical category.
- Improvements argue for environmental components. Reported cases of improvements or recoveries from autism have been published in the academic literature [Mundy, 1997, Ref. # 0145; Dawson, 2003, Ref. # 0154; Fein, 2005, Ref. # 0150; Kelley, 2006, Ref. # 0144]. Alex's improvements argue against purely genetic causes for his autism and make thimerosal exposure more likely as a contributing factor.

Absence of alternative explanations for Alex's developmental regression:

Alex received a thorough evaluation for alternative genetic and infectious explanations for his neurodevelopmental deterioration including but not limited to:

- Evaluation for MeCP2 abnormalities (Rett) – normal
- Prader-Willi/Angelman genetic testing – normal
- MRI of the brain – normal
- Guanidinoacetate methyltransferase deficiency – normal
- Herpes simplex titers – negative
- HHV 6 titers - negative

Thimerosal effects:

Referring to Dr. Vas Aposhian's report submitted to the court:

- He cited Pichichero's work on non-autistic children and Burbacher's work with primates as evidence for deposition of mercury in the brain after thimerosal containing vaccines.
- He reported that Pardo and Vargas documented the presence of neuroinflammation with activation of the brain's innate immune system.
- He explained the concept of developmental windows of increased vulnerabilities to toxins.
- He articulated the concept that there are variable vulnerabilities to exposure to mercury, based on other modifying factors and genetic predispositions, citing the fact that not all children exposed to mercury containing teething powders developed Pink disease
- He reviewed the toxicokinetics of thimerosal.

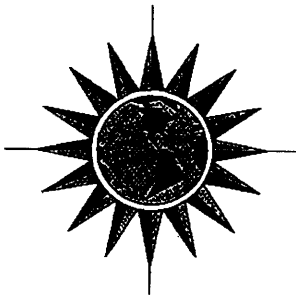
Referring to Dr. Richard Deth's report submitted to the court:

- He described the detrimental effects of thimerosal on cellular redox status and glutathione levels.
- His experiments demonstrated the potent inhibition of neuronal methionine synthase by thimerosal at concentrations far below the plasma level of one thimerosal containing vaccine.
- He reported that thimerosal is known to be toxic to human cortical neurons, and induces apoptosis (programmed cell death).
- He described how thimerosal interferes with cellular production of glutathione, which is a crucial mechanism for the body to deal with heavy metal toxicity.
- He explained how thimerosal induces oxidative stress and interferes with sulfate metabolism, which is crucial for getting rid of toxins and heavy metals.

Summary:

- In my best medical judgment based on my understanding of the medical literature some of which is cited above and my clinical experience, Alex is a child whose neurodevelopmental problems were exacerbated by mercury exposure in vaccines.
- Delayed manifestations of neurotoxicity as evidenced by emerging symptoms of autism many months after his exposure to mercury in vaccines are consistent with the pattern of developmental toxins.
- He seemed to acutely deteriorate after the age of 2, coincident with receipt of 2 flu vaccines a month apart. This is consistent with reaching a toxic tipping point, in my judgment.
- Clinically, he fits the picture of a child with genetic predispositions which acted together with environmental triggers to develop neurologic impairments.

- Thimerosal reduces cellular glutathione, which is the body's major intracellular anti-oxidant, and serves vital roles in maintaining the gut epithelium, preserving immune function, and enabling adequate detoxification. Alex demonstrated problems in all these areas.
- Thimerosal has devastating effects on methylation biochemistry, which ironically is the main way the body attempts to deal with heavy metal toxicity. My clinical experience in conjunction with my understanding of the published works of Drs. James and Deth, lead me to have grave concerns about the clinical consequences of thimerosal exposure in this child with impaired methylation biochemistry.
- Alex had documented mercury exposures via thimerosal and demonstrated mercury excretion after clinical measures to enable him to get rid of some of his mercury body burden. He had clinical problems compatible with the expected effects of thimerosal toxicity, especially with his dramatic autoimmune problems. He showed clinical improvements in his developmental status following measures to treat his oxidative stress, detoxification systems, and immune functioning, leading me to conclude that thimerosal was a substantial contributing factor in the development of his neurodevelopmental and language regression.



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In the case of Alexander Krakow:

The opinions expressed in this report are held by me to a reasonable degree of medical and scientific likelihood. I reserve the right to supplement this report in light of any additional scientific or medical literature that may be published during the pendency of this claim in the NVICP, or in light of any relevant change in Alex Krakow's medical condition.

Elizabeth Mumper, MD
Elizabeth Mumper, MD

11/30/07
Date

PETITIONERS' MASTER REFERENCE LIST - REFERENCE NUMBER SORT

MASTER REFERENCE NUMBER	AUTHOR	TITLE	CITATION	DATE
0001	Halsey NA, Goldman L	Balancing Risks and Benefits: Primum non nocere Is Too Simplistic	Pediatrics;108:466-7	8/2/2001
0002	McRill C, Boyer LV, Flood TJ, Ortega L	Mercury Toxicity Due to Use of a Cosmetic Cream	Journal of Occupational and Environmental Medicine;42:4-7	1/1/2000
0003	Nierenberg DW, Nordgren RE, Chang MB, Siegler RW, Blayney MB, Hochberg F, Toribara TY, Cernichiari E, Clarkson TW	Delayed Cerebellar Disease and Death after Accidental Exposure to Dimethylmercury	New England Journal of Medicine;338 (23):1672-76	6/4/1998
0004	Andrews, N et al	Thimerosal Exposure in Infants and Developmental Disorders: A Retrospective Cohort Study in the United Kingdom Does Not Support a Causal Association	Pediatrics, Vol 114, No. 3, September 2004	9/1/2004
0005	James SJ, Cutler P, Melnyk S, Jernigan S, Janak L, Gaylor DW, Neubrandner JA.	Metabolic biomarkers of increased oxidative stress and impaired methylation capacity in children with autism.	Am J Clin Nutr. 2004 Dec;80(6):1611-7.	12/1/2004
0006	Costa LG, Aschner M, Vitalone A, Syversen T, Soldin OP	Developmental neuropathology of environmental agents.	Annu Rev Pharmacol Toxicol. 2004;44:87-110.	1/1/2004
0007	James SJ, Slikker W 3rd, Melnyk S, New E, Pogribna M, Jernigan S	Thimerosal neurotoxicity is associated with glutathione depletion: protection with glutathione precursors.	Neurotoxicology. 2005 Jan;26(1):1-8.	1/1/2005
0008	Humphrey ML, Cole MP, Pendergrass JC, Kiningham KK	Mitochondrial mediated thimerosal-induced apoptosis in a human neuroblastoma cell line (SK-N-SH).	Neurotoxicology. 2005 Jun;26(3):407-16.	6/1/2005
0009	Rimland B	Treatment Options for Mercury/Metal Toxicity in Autism and Related Developmental Disabilities: Consensus Position Paper	Autism Research Institute	2/1/2005
0010	Counter S, Buchanan L, Ortega F	Neurocognitive screening of mercury-exposed children of Andean gold miners.	Int J Occup Environ Health. 2006 Jul-Sep;12(3):209-14.	7/1/2006
0011	Blaxill, MF	What's going on? The question of time trends in autism	Public Health Reports, Nov-Dec 2004;119:536-551	1/1/2004
0012	Croen LA, Grether JK	Response: A Response to Blaxill, Baskin, and Spitzer on Croen et al. (2002), the changing prevalence of autism in California.	J Autism Dev Disord. 2003 Apr;33(2):227-229	4/1/2003
0013	Haley, B	Mercury Toxicity: Genetic Susceptibility and Synergistic Effects	Medical Veritas 2 (2005) 535-542	2/1/2005
0014	Heron et al.	Thimerosal Exposure in Infants and Developmental Disorders: A Prospective Cohort Study in the United Kingdom Does Not Support a Causal Association	Pediatrics; Vol 114; No. 3; September 2004:577-583	9/1/2004
0015	Hornig, M, et al	Neurotoxic Effects of Postnatal Thimerosal Are Mouse Strain Dependant	Molecular Psychiatry 2004:1-13	5/4/2004
0016	Hu, L et al.	Neutron activation analysis of hair samples for the identification of autism.	Poster presentation:Trans Am Nucl Soc 2003;89	1/1/2003
0017	Lorscheider et al.	Degenerative Neuron (Snail Brain Video)	U of Calgary Video	00/00/0000
0018	Windham GC, Zhang L, Gunier R, Croen LA, Grether JK	Autism spectrum disorders in relation to distribution of hazardous air pollutants in the San Francisco Bay Area.	Environ Health Perspect. 2006 Sep;114(9):1438-44.	9/1/2006
0019	Verstraeten, T	Letters to the Editor: Thimerosal, the Centers for Disease Control and Prevention, and GlaxoSmithKline	Pediatrics. 2004 Apr;113(4):932	4/1/2004

0020	Stratton K, Gable A, McCormick, C	Immunization Safety Review: Thimerosal-Containing Vaccines and Neurodevelopmental Disorders - ADVANCE COPY	ADVANCE COPY Report of the Institute of Medicine 2001	10/1/2001
0021	Parran DK, Barker A, Ehrich M.	Effects of thimerosal on NGF signal transduction and cell death in neuroblastoma cells.	Toxicol Sci. 2005 Jul;86(1):132-40. Epub 2005 Apr 20.	4/20/2005
0022	Luyster R, Richler J, Risi S, Hsu WL, Dawson G, Bernier R, Dunn M, Hepburn S, Hyman SL, McMahon WM, Goudie-Nice J, Minshew N, Rogers S, Sigman M, Spence MA, Goldberg WA, Tager-Flusberg H, Volkmar FR, Lord C.	Early regression in social communication in autism spectrum disorders: a CPEA Study	Dev Neuropsychol. 2005;27(3):311-36	1/1/2005
0023	Ashwood P, Wakefield AJ	Immune activation of peripheral blood and mucosal CD3+ lymphocyte cytokine profiles in children with autism and gastrointestinal symptoms	J Neuroimmunol. 2006 Apr;173(1-2):126-34. Epub 2006 Feb 21	4/1/2006
0024	Herdman ML, Marcelo A, Huang Y, Niles RM, Dhar S, Kinningham KK	Thimerosal induces apoptosis in a neuroblastoma model via the cJun N-terminal kinase pathway	Toxicol Sci. 2006 Jul;92(1):246-53. Epub 2006 Apr 19	4/19/2006
0025	Brown L	Thimerosal Induces Programmed Cell Death of Neuronal Cells via Changes in the Mitochondrial Environment	The UCI Undergraduate Research Journal	00/00/0000
0026	Burbacher TM, Shen DD, Liberato N, Grant KS, Cernichiari E, Clarkson T	Comparison of blood and brain mercury levels in infant monkeys exposed to methylmercury or vaccines containing thimerosal.	Environ Health Perspect. 2005 Aug;113(8):1015-21	08/00/2005
0027	Heyer NJ, Bittner AC Jr, Echeverria D, Woods JS	A cascade analysis of the interaction of mercury and coproporphyrinogen oxidase (CPOX) polymorphism on the heme biosynthetic pathway and porphyrin production.	Toxicol Lett. 2006 Feb 20;161(2):159-66. Epub 2005 Oct 7	2/20/2006
0028	Amin-Amin-Zaki L, Majeed MA, Greenwood MR, Elhassani SB, Clarkson TW, Doherty RA	Methylmercury poisoning in the Iraqi suckling infant: a longitudinal study over five years.	J Appl Toxicol. 1981 Aug;1(4):210-4.	8/1/1981
0029	Baron-Cohen S, Knickmeyer RC, Belmonte MK	Sex differences in the brain: implications for explaining autism.	Science. 2005 Nov 4;310(5749):819-23	11/4/2005
0030	de Bruin EI, Verheij F, Wiegman T, Ferdinand RF	Differences in finger length ratio between males with autism, pervasive developmental disorder-not otherwise specified, ADHD, and anxiety disorders.	Dev Med Child Neurol. 2006 Dec;48(12):962-5	12/1/2006
0031	Buyske S, Williams TA, Mars AE, Stenroos ES, Ming SX, Wang R, Sreenath M, Factura MF, Reddy C, Lambert GH, Johnson WG	Analysis of case-parent trios at a locus with a deletion allele: association of GSTM1 with autism.	BMC Genet. 2006 Feb 10;7(1):8 [Epub ahead of print]	2/10/2006
0032	Charleston JS, Body RL, Mottet NK, Vahter ME, Burbacher TM	Autometallographic determination of inorganic mercury distribution in the cortex of the calcarine sulcus of the monkey Macaca fascicularis following long-term subclinical exposure to methylmercury and mercuric chloride.	Toxicol Appl Pharmacol. 1995 Jun;132(2):325-33	6/1/1995
0033	Charleston JS, Bolender RP, Mottet NK, Body RL, Vahter ME, Burbacher TM	Increases in the number of reactive glia in the visual cortex of Macaca fascicularis following subclinical long-term methyl mercury exposure.	Toxicol Appl Pharmacol. 1994 Dec;129(2):196-206.	12/1/1998
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CERTIFICATE OF SERVICE

I hereby certify that on November 30, 2007, I served the foregoing **Expert Report Re Alexander Krakow from Elizabeth A. Mumper, M.D.** on the following individual(s):

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