

CoMED INC.

14 REDGATE COURT,
SILVER SPRING, MD 20905-5726

Review of recent publications that show Thimerosal-preserved vaccines are major cause of current chronic-condition ‘autism’ epidemic

by Paul G. King, PhD, CoMeD Science Advisor, Science Advisor to the National Coalition of Organized Women (NCOW), and a Science Advisor to StS and other groups

Recent publications by several research groups, and myself, in several areas have all combined to further establish that Thimerosal-preserved vaccines, *which have not been banned from use in the USA and are widely used in much of the rest of the world today*, are a major causal factor in the epidemics of chronic childhood medical conditions, especially “autism”, which have incidence rates today of at least 1 in 100 (e.g., children with a diagnosis of an autism spectrum/pervasive development disorder [ASD/PDD]) and, in some cases, exceed 1 in 10 (e.g., asthma), but, *prior to the mid 1970s*, were either unknown in the USA (e.g., childhood type 2 diabetes) or were rare (i.e., having an incidence rates of about 1 in 10,000 or less; e.g., “autism”).

Similarly, *but displaced forward by more than a decade in time*, in New Delhi, India, *prior to 2000*, ASD/PDD (autism spectrum disorder/pervasive developmental disorder) symptoms were rare – typically only occurring in children who were vaccinated abroad until after the Indian pediatricians began recommending, in 2000, the addition of triple-dose Thimerosal-preserved Hib (Haemophilis influenza B) and Hep B (hepatitis B) vaccination programs to the existing Thimerosal-preserved triple dose DTP (diphtheria toxin, tetanus toxin and pertussis toxins) vaccination program recommended by the Government of India.

Moreover, the apparent “doubling” in ASD/PDD incidence that occurred in a New Delhi, India nursery school’s population after these Indian pediatricians reduced the recommended completion date for the 9-Thimerosal-preserved vaccine doses (3 DTP, 3

Hep B, and 3 Hib) from 6 months in 2000 to 3.5 months in the mid-2000s.

Hopefully, the abstracts, keywords, and brief notes by this reporter for each of the 9 articles that are important but, *for the most part*, have been ignored by the major media and many “autism groups” (e.g., Autism Speaks) that apparently have no interest in informing the public of the scientifically sound evidence linking Thimerosal-preserved vaccines to ASD/PDD and other childhood neurodevelopmental, developmental, and behavioral disorders, syndromes, and diseases that were unknown (e.g., childhood type 2 diabetes) or rare (e.g., asthma) but are common today.

However, this reporter would be remiss if he did not again warn the reader that no one, *other than the researchers conducting the study (not necessarily the persons writing the articles) and God*, know whether or not a published account, *peer-reviewed or sworn to, or not*, is a sound, valid, straightforward recounting of the observations made, the studies performed, and the outcomes reported.

Further, this reporter reminds the reader that independent reviews of many of the recent published peer-reviewed articles bearing on pharmaceutical safety and effectiveness issues have found that the studies reviewed were:

- Ghost written by other than the named authors;
- Perverted by those conducting them;
- Designed not to study what the article claimed to have studied;
- Statistically manipulated to reduce the certainty in the adverse effects below the legal level of concern
- Misleading or false in their design, execution and reporting because the study was never actually conducted but was fabricated by an author;

- Otherwise flawed; and/or
- Some combination of the preceding.

With the preceding caveats in mind, this reporter will now present those published articles that directly or indirectly establish Thimerosal-preserved vaccines are major causal factors for the epidemics of childhood chronic medical conditions (childhood disorders, syndromes and diseases) that beset the American public and the world today.

In general, this reporter will add underlining and/or double underlining to emphasize the important points in each publication.

Further, as with all of this reviewer's publications, should any reader find significant factual errors in this editorial, then please send the author (at paulgkingphd@gmail.com) your proposed changes along with e-mail attachments that contain copies of the published documents that provide the proofs needed to substantiate your claims.

Then, as has been the case in the past, after verifying the validity of your concerns, the confirmed factual errors will be corrected and an appropriately "revised document" posted as widely as the initial document was.

If you find spelling, grammar or textual errors, please also send them in so that this document can be appropriately revised and reposted as an "updated document".

Finally, this reviewer has suggested corrections in red bracketed text (e.g., [correction]) in instances where such are required or seem to be important to include.

With the preceding in mind, this reviewer/reporter will begin with the paper that was published on-line in June of 2010.

1. Kern JK, Geier DA, Adams JB, Geier MR. A biomarker of mercury body-burden correlated with diagnostic domain specific clinical symptoms of autism spectrum disorder *Biometals* 2010, published online “09 June 2010”; DOI 10.1007/s10534-010-9349-6.

[\[http://www.springerlink.com/content/100146/?Content+Status=Accepted&sort=p_OnlineDate&sortorder=desc&v=condensed&o=10\]](http://www.springerlink.com/content/100146/?Content+Status=Accepted&sort=p_OnlineDate&sortorder=desc&v=condensed&o=10)

Abstract The study purpose was to compare the quantitative results from tests for urinary porphyrins, where some of these porphyrins are known biomarkers of heavy metal toxicity, to the independent assessments from a recognized quantitative measurement, the Autism Treatment Evaluation Checklist (ATEC), of specific domains of autistic disorders symptoms (Speech/Language, Sociability, Sensory/ Cognitive Awareness, and Health/Physical/Behavior) in a group of children having a clinical diagnosis of autism spectrum disorder (ASD). After a Childhood Autism Rating Scale (CARS) evaluation to assess the development of each child in this study and aid in confirming their classification, and an ATEC was completed by a parent, a urinary porphyrin profile sample was collected and sent out for blinded analysis. Urinary porphyrins from twenty-four children, 2–13 years of age, diagnosed with autism or PDD-NOS were compared to their ATEC scores as well as their scores in the specific domains (Speech/Language, Sociability, Sensory/Cognitive Awareness, and Health/Physical/Behavior) assessed by ATEC. Their urinary porphyrin samples were evaluated at Laboratoire Philippe Auguste (which is an ISO-approved clinical laboratory). The results of the study indicated that the participants’ overall ATEC scores and their scores on each of the ATEC subscales (Speech/Language, Sociability, Sensory/Cognitive Awareness, and Health/Physical/Behavior) were linearly related to urinary porphyrins associated with mercury toxicity. The results show an association between the apparent level of mercury toxicity as measured by recognized urinary porphyrin biomarkers of mercury toxicity and the magnitude of the specific hallmark features of autism as assessed by ATEC.

Keywords Toxicity, Mercury, CARS, ATEC, ASDs, Asperger’s, Autism”

Simply put, this study demonstrated that there was a linear correlation between the levels of the mercury-associated urinary-toxicity indicating porphyrins and the degree of impairment observed in 24 children with a diagnosis of an ASD. Thus, the child’s mercury-toxicity burden is shown to correlate with the degree of impairment observed as measured by the ATEC Test and various ATEC Subscales.

2. Kern JK, Geier DA, Adams JB, Mehta JA, Grannemann BD, Geier MR. Toxicity Biomarkers in Autism Spectrum Disorder: A Blinded Study of Urinary Porphyrins. *Pediatrics International* 2010. Accepted Article: “09 June 2010”; doi: 10.1111/j.1442-200X.2010.03196.x.

[\[http://www3.interscience.wiley.com/journal/123573454/abstract\]](http://www3.interscience.wiley.com/journal/123573454/abstract)

“Abstract

Background: Recent studies suggest children diagnosed with an autism spectrum disorder (ASD) have significantly increased levels of urinary porphyrins associated with mercury (Hg) toxicity, including pentacarboxyporphyrin (5cxP), precoproporphyrin (prcP), and coproporphyrin (cP), compared to typically developing controls. However, these initial studies were criticized because the controls were not age- and gender-matched to the children diagnosed with an ASD.

Methods: Urinary porphyrin biomarkers in a group of children (2-13 years of age) diagnosed with an ASD (n=20) were compared to matched (age, gender, race, location, and year tested) group of typically developing controls (n=20).

Results: Participants diagnosed with an ASD had significantly increased levels of 5cxP, prcP, and cP in comparison to controls. No [statistically] significant differences were found in non-Hg associated urinary porphyrins (uroporphyrins, hexacarboxyporphyrin, and heptacarboxyporphyrin). There was a significantly increased odds ratio for an ASD diagnosis relative to controls among study participants with precoproporphyrin (odds ratio=15.5, p<0.01) and coproporphyrin (odds ratio=15.5, p<0.01) levels in the second through fourth quartiles in comparison to the first quartile.

Conclusion: These results suggest that the levels of Hg-toxicity-associated porphyrins are higher in children with an ASD diagnosis than controls. Although the pattern seen (increased 5cxP, prcP, and cP) is characteristic of Hg toxicity, the influence of other factors, such as genetics and other metals[,] cannot be completely ruled-out.

Key words: toxicity, mercury, Hg, heavy metal, ASD, autism, porphyrins”
[Underlining emphasis added.]

Simply put, this cases-matched-controls study demonstrated that there was a elevation in the levels of the urinary-mercury-toxicity-indicating porphyrins in children with a ASD diagnosis as compared to the levels of those urinary-mercury-toxicity-indicating porphyrins in the matched (age, gender, race, location and year tested) control children studied. There was no similar elevation for the other urinary porphyrins in either the case or the control groups. To be fair, the researchers reported:

“Although the pattern seen ... is characteristic of Hg toxicity, the influence of other factors, such as genetics and other metals[,] cannot be completely ruled-out”.

3. King PG. Parallels in New Delhi, India: An Epidemic Induced By Added Doses Of Thimerosal-Preserved Vaccines. Published on-line 11 July 2010.

[\[http://dr-king.com/docs/100711_ParallelsinNewDelhiIndia_AnEpidemic_b.pdf\]](http://dr-king.com/docs/100711_ParallelsinNewDelhiIndia_AnEpidemic_b.pdf)

The importance of this report is that it clearly established that the year 2000 addition of six (6) Thimerosal-preserved vaccine doses (3 each for Hep B and Hib) to the three doses of Thimerosal-preserved DTP vaccines that New Delhi, India children were already receiving in their pediatrician-recommended vaccination program increased the observed incidence of “ASD”-symptomatic children from <1 in about 2200 to 2 to 4% when the completion date for the Thimerosal-preserved DTP and added Hib and Hep B vaccinations was “by 6 months of age”.

That the Thimerosal-preserved vaccines are the casual factor is confirmed by a doubling in the incidence of affected children to about 5 to 8 % after the Indian pediatrician reduced completion age for the nine Thimerosal-preserved vaccine doses from 6 months to 3.5 months.

Thus, the principal source of the observed post-vaccination mercury-poisoning symptoms and post-vaccination clinical diagnosis outcomes in this instance is the Thimerosal-preserved vaccines is supported by the reality that these New Delhi, India children and their families are vegetarians – they do not eat fish.

As we shall see, this reality also impacts the other reported study involving children in India.

4. Priya MDL, Geetha A. Level of Trace Elements (Copper, Zinc, Magnesium and Selenium) and Toxic Elements (Lead and Mercury) in the Hair and Nail of Children with Autism. *Biol Trace Elem Res* 2010, published online July 2010 (11 pages).
[\[http://www.unboundmedicine.com/medline/ebm/record/20625937/abstract/Level of Trace Elements Copper Zinc Magnesium and Selenium and Toxic Elements Lead and Mercury in the Hair and Nail of Children with Autism \]](http://www.unboundmedicine.com/medline/ebm/record/20625937/abstract/Level_of_Trace_Elements_Copper_Zinc_Magnesium_and_Selenium_and_Toxic_Elements_Lead_and_Mercury_in_the_Hair_and_Nail_of_Children_with_Autism)

“Abstract Autism is a multi-factorial pathology observed in children with altered levels of essential and elevated levels of toxic elements. There are also studies reporting a decrease in nutritional trace elements in the hair and nail of autistic children with healthy controls; moreover, bioelements have been shown to play an important role in the central nervous system. Therefore, the purpose of the present study was to assess the levels of trace elements like copper (Cu), zinc (Zn), magnesium (Mg), and selenium (Se) and toxic elements like mercury (Hg), and lead (Pb) in the hair and nail samples of autistic children and to evaluate whether the level of these elements could be correlated with the severity of autism. The subjects of the study were 45 autistic children with different grades of severity (low (LFA), medium (MFA), and high (HFA) functioning autism) according to Childhood Autism Rating Scale, n=15 children in each group and 50 healthy children (age and sex matched). The boys and girls ratio involved in this study was 4:1, and they were 4-12 years of age. The study observed a valid indication of Cu body burden in the autistic children. The children with different grades of autism showed high significance (p<0.001) in the level of copper in their hair and nail samples when compared to healthy controls. The level of Cu in the autistic children could be correlated with their degree of severity (more the Cu burden severe is autism). The study showed a significant elevation (p<0.001) in the levels of toxic metals Pb and Hg in both hair and nail samples of autistic children when compared to healthy control group. The elevation was much pronounced in LFA group subjects when compared among autistic groups MFA and HFA. The levels of trace elements Mg and Se were significantly decreased (p<0.001) in autistic children when compared to control. The trace element Zn showed significant variation in both hair and nails of LFA group children when compared to control group and other study groups. The significant elevation in the concentration of Cu, Pb, and Hg and significant decrease in the concentration of Mg and Se observed in the hair and nail samples of autistic subjects could be well correlated with their degrees of severity.

Keywords Autism . Hair . Nail . Trace elements . Degrees of severity”

Simply put, this is a case-matched-control study of the comparative levels of key “trace” metals, both beneficial (Cu, Mg, Se, and Zn) and toxic (Pb and Hg), in the case group to those in the matched control group.

In addition, the correlation between the degree of severity (as assessed by CARS evaluations) in the “autism” group and these metals was assessed.

The samples assessed were nape of the neck hair and fingernail clippings.

The observation of a 4:1 male-to-female ratio in the case group again points to a causal factor that is related to the child’s sex in a manner that males are several times more likely to be affected than females – mercury poisoning is one of the few types of poisoning that exhibits this type of sex ratio

Here it is again important to remember that dietary mercury from fish is not a contributor to the elevated levels of mercury observed because the children and their families do not eat fish, fish-meal-fed animals, or fish-derived foods as a general rule.

Thus, given the preceding, Thimerosal (vaccine-mercury) from Thimerosal-preserved vaccines is the most logical major post-natal source – though there may be contributions from the child’s mother or mother’s milk when the child is breastfed, in instances where the mother has “silver-mercury” (amalgam) fillings or, typically in older children, the placement of amalgam dental fillings in the child.

In general, statistically significant between-group differences were observed for cases and the matched controls for all of the metals measured when the cases were classified by CARS as Low-Functioning (LFA) and between the LFA and the High-functioning (HFA) cases.

In addition, a highly statistically significant ($p < 0.001$) correlation was found between all measures in the case group and their CARS scores.

Based on this reviewer's assessment of the data, it would seem that, between hair and nail samples, that the copper (Cu), Magnesium (Mg), Selenium (Se), Zinc (Zn) essential elements and Lead (Pb) and mercury (Hg) toxic elements and the Cu/Zn ratio (not reported but discussed) in nail samples should be used as the basis for an independent measure of severity in assessing neurodevelopmental impairment in children with an ASD diagnosis independent of the children's mercury body burden (which should still be assessed using a valid urine porphyrin profile analysis).

Since there are ISO-17025, CLIA-certified clinical laboratories that have an ICP-MS system set up to assess the levels of the range of elements accessible to an ICP-MS system using a single prepared nail sample, it would seem that a follow-up study would explore all of the available elements in nail samples to assess the correlation of all of the available metals as a measure of severity in those diagnosed with an ASD as well as a discriminator between those who are not in the ASD spectrum and those who are.

Since only the Pb levels in the LFA group were statistically significant compared to the controls in the nail samples, while the Hg levels were statistically significant for the hair and nail samples in all case groups (LFA, MFA and HFA), these results seem to point to a probably much higher degree of mercury intoxication in the children with an ASD diagnosis than lead intoxication. However, the appropriate urinary porphyrin profile analyses and red-blood-cell Hg and Pb levels would be needed in the case group using fresh urine, blood, and nail samples to assess how the levels being excreted match

both the body burden (as measured by the urinary porphyrins) and the circulating levels of Pb and Hg (as measured by the levels in the patient's red blood cells).

Further, the most important, in the USA, toxic metal that was left out is arsenic and a full ICP-MS scan would shed light on this missing piece of the puzzle.

Finally, the researchers closing remarks are important to relate here:

“The limitation of the study is that we have measured the levels of only few trace elements like Cu, Zn, Mg, and Se and toxic elements like Pb and Mg, whereas there are also other essential trace elements which may be just or even more important for life and other toxic elements which may be just or even more threatening to life. So the study can be extended to evaluate the other trace and toxic elements in autistic children with different grades of severity.”

5. Majewska MD, Urbanowicz E, Rok-Bujko P, Namysłowska I, Mierzejewski P. Age-dependent lower or higher levels of hair mercury in autistic children than in healthy controls. *Acta Neurobiol Exp* 2010, **70**(2): 196–208. [<http://www.ane.pl/pdf/7024.pdf>]

“An association between autism and early life exposure to mercury is a hotly debated issue. In this study, 91 autistic Polish children, male and female, 3-4 and 7-9 years old, were compared to 75 age- and sex-matched healthy children with respect to: demographic, perinatal, clinical and developmental measures, parental age, birth order, morphometric measures, vaccination history, and hair mercury content. In demographic and perinatal measures[,] there were no consistent differences between the autistic and control groups. Autistic children had a significantly greater prevalence of adverse reactions after vaccinations and abnormal development than controls. Between 45 and 80% of autistic children experienced developmental regress[ion]. Autistic children significantly differed from healthy peers in the concentrations of mercury in hair: younger autistics had lower levels, while older – higher levels than their respective controls. The results suggest that autistic children differ from healthy children in metabolism of mercury, which seems to change with age”.

Key words: autism, mercury, hair, thimerosal, vaccines, development

Abbreviations: THIM – thimerosal”

Simply put, this is another case-matched-control study where the matching has been extended to address multiple potentially confounding factors (age- and sex-matched healthy children with respect to: demographic, perinatal, clinical and developmental measures, parental age, birth order, morphometric measures, vaccination history, and hair-Hg content).

In addition, a two-level design (GROUP I; children 3-4 years of age and GROUP II, children 7-9 years of age) was adopted to ascertain if there were differences between the age groups. In general, all of these children (case and control) had the “same” (*not statistically different*) vaccination exposures by 2 years of age, except for the females in the 7-9 group (who, on average, received about four (4) less vaccine doses by 2 years of age than either the male or the female controls).

The differences seen are strictly associated with the diagnostic difference (“ASD excluding Asperger’s” for the case children and “normal” for the controls) and apparently not to any other factor.

The paper’s Table III and Figure 1 shown below clearly summarize the real differences between the cases and the controls as follows

Table III

| Comparison of combined groups of autistic and control children | | | |
|--|---------------------------------|---------------------------------|-------------------|
| | Autistic (M+F) Groups I + II | Controls (M+F) Groups I + II | <i>p</i> |
| <u>Vaccine complications (%)</u> | <u>20.4*</u> | 6.5 | <u>p=0.009</u> |
| <u>Abnormal development (%)</u> | <u>40.9*</u> | 3.9 | <u>p<0.001</u> |
| Caesarian or pathological birth (%) | 32 | 29 | NS |
| Epilepsy (%) | 5.5 | 1.2 | NS |
| Potential Rh conflict (%) | 8 | 12 | NS |
| Genetic load (%) | 12 | 5 | NS |

Nonparametric measures: Comparison of nonparametric measures between combined groups of autistic and control children. Information about epilepsy, potential genetic load and potential Rh conflict is based on parental interviews. M = males, F = females. Statistically significant differences are denoted by (*).

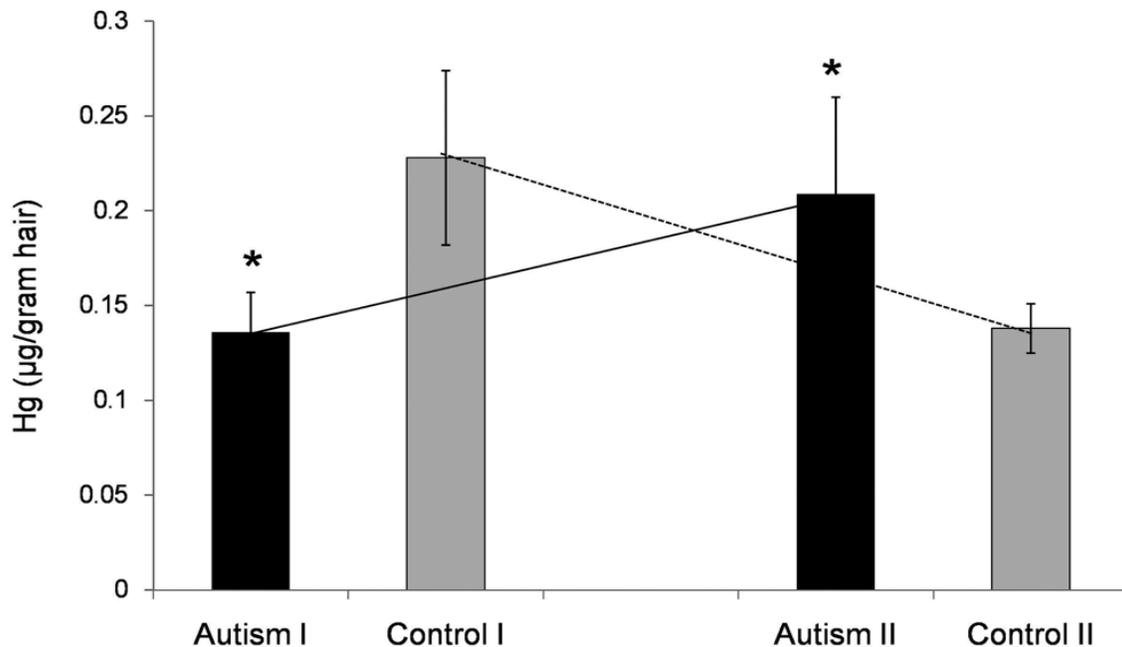


Fig. 1 Different levels of mercury in hair of autistic and healthy children from age groups I and II. The histogram shows mean values ± SEM. Statistically significant differences between autistic and control groups are denoted by (*), (p=0.01). Crossing lines point to divergent developmental trends of change in hair mercury levels between the autistic and control groups.

Significantly, there was no statistically significant difference between cases and controls attributable to: a) “Caesarian or pathological birth”, b) “Epilepsy”, c) “Potential Rh conflict”, and d) “Genetic load”.

The only parameters that were significantly different were: 1) Vaccine complications (20% in the overall “autism” case group; and 6.5% in the overall control group – a 3-fold difference) and 2) Abnormal development (40.9 % in the case group; 3.9 in the control group – a 13.6-fold difference).

In addition, as shown on the article’s “Fig. 1” above, in both age ranges (3-4 and 7-9), the average hair-mercury levels were significantly different between the case and control sets.

In addition, as the researchers reported, the controls had an average hair-mercury level that apparently decreased as the Polish children matured; while the average hair-mercury level in the case groups increased as the Polish children matured.

The researchers’ conclusion was:

“Autistic and healthy children differ in prevalence of abnormal development, frequency of adverse reactions to vaccinations and concentrations of mercury in hair, which change with development. The data indirectly imply vaccinations and mercurials as potential factors in autism pathogenesis.”

Further, since:

- ◆ Thimerosal-preserved vaccines (DTP, Hib and Hep B) have not been removed from the early childhood vaccines given by age 2;
- ◆ The Polish vaccination schedule has remained similar to the US vaccination schedule in the late 1990s with respect to Thimerosal-preserved vaccines; and
- ◆ Most all of the children in the study were vaccinated similarly,

the factors causing the differences seen must be related to:

1. Differences in the genetic make-up/susceptibilities of the damaged children as compared to the apparently undamaged controls and,
2. From the differences in the changes in the hair-mercury excretion patterns, some aspects of the differential toxicity of the mercury that is not initially excreted by the cases at the same rate as by the “fully” matched controls.

Since the vaccination histories for the cases and the controls are “matched”, it is not the vaccinations per se or the level of Thimerosal exposure per se that account for the significant differences seen but rather the significant differences reflect:

- How poorly some Polish children handle vaccination, in general, and/or, *based on the hair mercury data,*
- How poorly these children handled sub-acute mercury detoxification of the mercury in the multiple doses of Thimerosal-(49.55% mercury by weight)-preserved vaccines that they received by two years of age.

Taken together, the findings indicate that the Thimerosal-preserved vaccines still being administered in Poland to children from birth to 2 years of age are key factors in “autism pathogenesis”.

6. Geier DA, Audhya T, Kern JK, Geier MR. Blood mercury levels in autism spectrum disorder: Is there a threshold level? *Acta Neurobiol Exp* 2010, **70**(2): 177–186. [<http://www.ane.pl/pdf/7022.pdf>]

“Mercury (Hg) may significantly impact the pathogenesis of autism spectrum disorders (ASDs). Lab results generated by Vitamin Diagnostics (CLIA-approved), from 2003-2007, were examined among subjects diagnosed with an ASD (n=83) in comparison to neurotypical controls (n=89). Blood Hg levels were determined by analyzing Hg content in red blood cells (RBC) using cold vapor analysis, and consistent Hg measurements were observed between Vitamin Diagnostics and the University of Rochester. Adjusted (age, gender, and date of collection) mean Hg levels were 1.9-fold significantly (P<0.0001) increased among subjects diagnosed with an ASD (21.4 µg/L) in comparison to controls (11.4 µg/L). Further, an adjusted significant (P<0.0005) threshold effect (>15 µg/L) was observed for Hg levels on the risk of a subject being diagnosed with an ASD in comparison to controls (odds ratio=6.4). The weight of scientific evidence supports Hg as a causal factor in subjects diagnosed with an ASD.

Key words: Asperger, autistic, body-burden, neurodevelopmental, PDD, mercury]”

Simply put, this is another case-matched-control study.

Here the matching factors used were age, gender and date of collection.

The results from this research clearly establish that the average level of mercury in red blood cells is significantly elevated above the controls’ average level.

In addition, based on the results found, a child with a red-blood-cell mercury level above the lab’s blood-test’s level of 15 micrograms per liter has a significantly higher statistical risk (odds ratio=6.4; *where an odds ratio of “2” or more is considered legally significant*) of being diagnosed with an ASD than a child with a lower level of red-blood-cell mercury.

Finally, all should carefully consider the researcher’s conclusions concerning their finding here:

“The present study indicates that subjects diagnosed with an ASD have, on average, significantly higher levels of Hg in their blood than controls. The neurotoxicity of Hg is

well-established, and it is known that even small amounts of Hg can cause neurological injury similar to the brain pathology found in subjects diagnosed with an ASD (...). In addition, recent research indicates subjects diagnosed with an ASD have a decreased detoxification capacity for Hg (...). The weight of evidence provided by a variety of different studies offers a compelling argument for the hypothesis that Hg is a causal factor in the neuropathology reported in subjects diagnosed with an ASD.

It is recommended that further research should be conducted to evaluate the consistency of the present results with those in other populations of subjects diagnosed with an ASD. Investigators should also examine the potential correlation between elevated Hg and other potential markers of adverse effects in subjects with an ASD diagnosis, and physicians should consider treatment options that may be available for Hg-intoxication in subjects diagnosed with an ASD.”

Based on the findings reported in the other articles, in this review set, and previous articles, it is clear that Thimerosal-preserved vaccines have been clearly established as a major factor in the “autism” epidemic.

Moreover, *because Thimerosal-preserved flu shots are still the predominate vaccines in terms of doses and are still allowed to be administered to pregnant women and children from 6 months of age until 18 years of age*, Thimerosal-preserved vaccines will continue to be a major factor in the USA, if not the major factor, in “autism” and many other of the post-1988 rising epidemics of childhood chronic diseases, disorders and syndromes that were unknown or rare in the 1970s.

7. Geier DA, Kern JK, Geier MR. The biological basis of autism spectrum disorders: Understanding causation and treatment by clinical geneticists. *Acta Neurobiol Exp* 2010, **70**(2): 209–226. [<http://www.ane.pl/pdf/7025.pdf>]

“Autism spectrum disorders (ASDs), also known as pervasive developmental disorders (PDD), are a behaviorally defined group of neurodevelopmental disorders that are usually diagnosed in early childhood. ASDs disproportionately affect male children. Mercury (Hg), a heavy metal, is widespread and persistent in the environment. Mercury is a ubiquitous source of danger in fish, drugs, fungicides/herbicides, dental fillings, thermometers, and many other products. Elevated Hg concentrations may remain in the brain from several years to decades following exposure. This is important because investigators have long recognized that Hg is a neurodevelopmental poison; it can cause problems in neuronal cell migration and division, and can ultimately cause cell degeneration and death. Case-reports of patients have described developmental regressions with ASD symptoms following fetal and/or early childhood Hg exposure, and epidemiological studies have linked exposure to Hg with an elevated risk of a patient being diagnosed with an ASD. Immune, sensory, neurological, motor, and behavioral dysfunctions similar to traits defining or associated with ASDs were reported following Hg intoxication with similarities extending to neuroanatomy, neurotransmitters, and biochemistry. The sexual dimorphism of ASDs may result from synergistic neurotoxicity caused by the interaction of testosterone and Hg; in contrast, estrogen is protective, mitigating the toxicity of Hg. Mercury exposure may significantly increase androgen levels, and as a result, patients diagnosed with an ASD may significantly benefit from anti-androgen therapy. Finally, the clinical geneticist has a wealth of biomarkers to valuate and treat patients diagnosed with an ASD.

Key words: autistic, estradiol, ethylmercury, merthiolate, methylmercury, Thimerosal[, testosterone, androgens]”

Simply put, this article starts by introducing the reader to the history of “autism” in the USA and closes by noting:

“These epidemic rates for ASD diagnoses in the US have apparently coincided with a sharp rise in fetal and infant exposures to mercury (Hg) ...”

Next, the article reviews the history of mercury exposure and closes with:

“All told, researchers have reported that Hg exposures in early childhood from both potential environmental and vaccine sources resulted in some infants receiving in excess of 350 µg Hg during the first 6 months of life. It was estimated that about 50%

of the total Hg doses to which some infants were exposed came from routinely recommended Thimerosal-containing childhood vaccines. The cumulative exposure resulted in infants receiving doses of Hg in excess of Hg exposure limits established by the US EPA, US CDC, US Food and Drug Administration (FDA), and Health Canada during key developmental periods during the first year of life ...”.

This paper then discusses mercury distribution and persistence following exposure and closes with:

“Some researchers have described that Hg may have the potential to remain in the brain from several years to decades following exposure (Sugita 1978).”

Next, this paper briefly reviews the biological plausibility of the mercury-inducing symptoms used to diagnose an ASD (autism spectrum disorder) and closes with the telling statement:

“Finally, a scientific consensus statement developed by the Collaborative on Health and the Environment’s Learning Developmental Disabilities Initiative (2008) on environmental agents associated with neurodevelopmental disorders declared there was no doubt Hg exposure causes learning and developmental disorders including conditions such as ASDs”.

Then, the article proceeds to discuss in some detail the independent and reviewable evidence of a link between Thimerosal (49.55% mercury by weight) exposure from Thimerosal-preserved vaccines and children having an ASD diagnosis. This extended discussion of the mercury-ASD link closes with:

“Furthermore, Schweikert and others (2009) undertook an evaluation in the US, on a state by state basis, of ASD prevalence among 3 to 5 year-old children from 2000 to 2006 and environmental Hg exposure levels from 1996 to 2006. These investigators observed that Hg concentration in the environment among children 1 year-old or younger had a significant association with ASD prevalence three years later.”

Turning from direct and indirect human evidence of a mercury-autism link, the authors next briefly discuss various animal models of “ASD” symptoms or conditions induced by mercury exposure from injected Thimerosal-preserved vaccines (mouse, rat, and hamster).

Next, the authors reviewed the clinical evidence of susceptibility and toxicity that children with an ASD diagnosis exhibit and closes with:

“...potentially vulnerable sub-populations need to be identified and evaluated independently because large population epidemiologic studies do not have the sensitivity to detect minor high-risk subpopulations”

The article then proceeds to discuss the cellular mechanisms by which mercury exposure induces the symptoms that are used to diagnose an ASD.

From there, the authors turn to an extensive discussion for the several-fold excess of males to each female who has an ASD diagnosis in terms of not only mercury exposure but also hormonal system effects and hormonal system dysregulation by mercury compounds. This extensive discussion closes with:

“In putting these pieces together, environmental exposures (particularly Hg exposure) that adversely effect HST and the transsulfuration pathway can cause a cyclical biochemical interaction pattern to develop between the transsulfuration and androgen pathways that directly correlates with the biochemistry observed in those with an ASD diagnosis. As expected, this interaction pattern and androgen elevations are consistent with the behavioral/physical traits associated with or defining those who have an ASD diagnosis”

Next, the authors discuss how one should understand the treatment of hormonal imbalances in children and adults having an ASD (or related behavioral disorder) diagnosis. This discussion closes with:

“In evaluating the effects of leuprolide acetate administration to patients diagnosed with an ASD, investigators have described their clinical experience following its administration to nearly 200 patients with an ASD diagnosis. Leuprolide acetate administration significantly lowered androgen levels and resulted in very significant overall clinical improvements in socialization, sensory/cognitive awareness, and health/physical/behavior skills, with few non-responders and minimal adverse clinical effects to the therapy. It was also observed that leuprolide acetate administration resulted in significant clinical ameliorations in hyperactivity/impulsivity, stereotypy, aggression, self injury, abnormal sexual behaviors, and/or irritability behaviors ...”.

Unfortunately, perhaps because of article size limitations, other than briefly mentioning both cyproterone acetate, and spironolactone, the authors did not review their

use of these drugs in the treatment of hormonal imbalance in those with an ASD or related diagnosis.

Finally, in their conclusions, the authors present their conclusions and close with:

“Overall, it is apparent that many patients diagnosed with an ASD have significant medical conditions that require evaluation and potential treatment. Table 1 summarizes the specific types of biomarkers that a clinical geneticist may employ to help evaluate and treatment patients diagnosed with an ASD It is clear that as additional research is done, careful attention will be needed by the clinician to incorporate new testing and treatment options for the benefit of their patients on the autism spectrum.”

Since the authors’ “Table I” presents a fairly comprehensive overview of the tests for and some of the prescription drugs that are available to “treat” many of the damaged biological pathways, that reviewer has provided a transcribed version of the original as shown on the following page:

Table 1 [as in the text not “T” as at the top of the actual table]

| A summary of clinically available lab testing and clinically available drugs to treat such conditions among biomarkers associated with ASDs | | |
|---|--|--|
| Autism Biomarker | Clinical Laboratory Testing [LabCorp Test#] ¹ | Clinical Treatment Options |
| Porphyrins | Random Fractionated Urinary Porphyrins [120980] | Detoxification Therapy (DMSA, DMPS) |
| Transsulfuration | Homocysteine [706994], Cystathionine [911032], GSH [853002], Taurine [910844] | Methylcobalamin (vitamin B12), Folic Acid, Pyroxidine (Vitamin B6) |
| Oxidative Stress/ Inflammation | Oxidative Stress Panel (Catecholamine, GSH, Lipid Peroxides, GSH-Px, SOD) [853047], Neopterin [140335] | ALDACTONE® (Spironolactone) |
| Hormones | Testicular Function Profile II (FSH, LH, Testosterone, Free Testosterone) [035113], DHEA [004101], DHEA-S [004697], Androstenedione [004705], Androstane Diol Glucuronide [140442], Dihydrotestosterone [500142], Estradiol [140244], Estrone [004564], Total Estrogens [004549] | LUPRON® (Leuprolide Acetate), ANDROCUR® (Cypertyrone Acetate), ALDACTONE® (Spironolactone) |
| Mitochondria | Carnitine [706500], Pyruvic Acid [004788], Lactic Acid [004770], Ammonia [007054] | CARNITOR® (L-Carnitine) |
| Genetic | Blood Chromosomes [052019], Chromosome Microarray [510002], DNA Rett Syndrome [511180], Angelman/Prader Willi Syndrome Methylation Assay [511210], Fragile X Syndrome [510065], MTHFR [511238], APOE [822098] | Genetic Counseling (Prenatal, Pediatric, Predictive) |
| | | |
| <p>(APOE) Apolipoprotein E; (DHEA) Dehydroepiandrosterone; (DHEA-S) Dehydroepiandrosterone-Sulfate; (DMP) 2,3-Dimercapto-1-propanesulfonic acid; (DMSA) Meso 2,3-dimercaptosuccinic acid; (FSH) Follicle-stimulating hormone; (GSH) Glutathione; (GSHPx) Glutathione Peroxidase; (LH) Luteinizing hormone; (MTHFR) Methylenetetrahydrofolate reductase; (SOD) Superoxide Dismutase</p> <p>¹Laboratory testing described is available from the Laboratory Corporation of America (LabCorp), and is covered by most major insurance companies.</p> | | |

8. Hewitson L, Lopresti BJ, Stott C, Mason S, Tomko J. Influence of pediatric vaccines on amygdala growth and opioid ligand binding in rhesus macaque infants: A pilot study *Acta Neurobiol Exp* 2010, **70**(2): 147–164. [<http://www.ane.pl/pdf/7020.pdf>]

“This longitudinal, case-control pilot study examined amygdala growth in rhesus macaque infants receiving the complete US childhood vaccine schedule (1994-1999). Longitudinal structural and functional neuroimaging was undertaken to examine central effects of the vaccine regimen on the developing brain. Vaccine-exposed and saline-injected control infants underwent MRI and PET imaging at approximately 4 and 6 months of age, representing two specific timeframes within the vaccination schedule. Volumetric analyses showed that exposed animals did not undergo the maturational changes over time in amygdala volume that was observed in unexposed animals. After controlling for left amygdala volume, the binding of the opioid antagonist [¹¹C]diprenorphine (DPN) in exposed animals remained relatively constant over time, compared with unexposed animals, in which a significant decrease in [¹¹C]DPN binding occurred. These results suggest that maturational changes in amygdala volume and the binding capacity of [¹¹C]DPN in the amygdala was significantly altered in infant macaques receiving the vaccine schedule. The macaque infant is a relevant animal model in which to investigate specific environmental exposures and structural/functional neuroimaging during neurodevelopment.

Key Words: rhesus macaques, Macaca mulatta, non-human primates, animal model, neuroimaging, PET, MRI, amygdala, opioids, ethyl mercury, thimerosal, neurotoxicity”

This primate case-control paper begins by introducing the reader to the region of the primate brain known as the “amygdale”, “a complexly interconnected limbic system structure located in the temporal lobe of the brain”.

That introduction closes with:

“The safety of the combined vaccine regimen per se, rather than that of individual vaccines or vaccine components, is an important aspect of vaccine safety that has not been examined. In order to more directly investigate the neurodevelopmental impact of the complete US pediatric vaccine schedule (1994-1999), our model examined structural and functional changes in the amygdala before and after vaccination in the developing infant primate brain. Longitudinal development and functional characteristics of the amygdala are reported in vaccinated (exposed) and unvaccinated (unexposed) animals, and data on the novel application of [¹¹C]DPN PET to the study of macaque central nervous system (CNS) development are presented”.

The article then discusses the methods it used for the study in general touching on “**Animal Assurances**”, “**Animal Husbandry**”, “**Study Design**”, “**Vaccine Dosing and Administration**”, “**Magnetic Resonance (MR) Image Acquisition**”, “**Positron Emission Tomography (PET)**”, “**Image Co-Registration and Volume-of-Interest (VOI) Determination**”, “**PET Data Analyses**”, and “**Statistical Analysis**” with varying degrees of specificity.

Next, the researchers discuss their findings under a heading titled, “**RESULTS**”. The specific results categories initially discussed are “**Volumetric analyses of the total brain**”, and “**Volumetric analyses of the amygdala**”, where the subcategories discussed are: “**Total Amygdala Volume**”, “**Right Amygdala Volume**” and “**Left Amygdala Volume**”.

Then, the authors’ results’ reporting turned to “**PET data analyses of region-specific [¹¹C]DPN binding in macaque brain**” and their findings clearly showed significant effects. Since figures speak volumes, this review has included the article’s “**Fig. 3**” here:

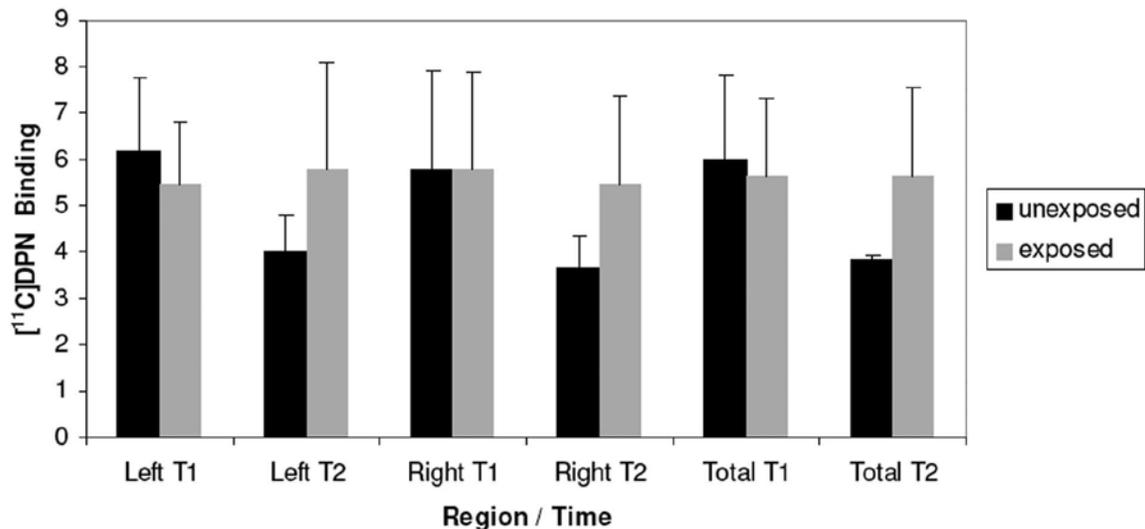


Fig. 3. [¹¹C]DPN binding potential (BPND) values in the amygdalae. [¹¹C]DPN binding potential (BPND) values (raw mean+1 SD) in left, right and total amygdalae at T1 and T2 in exposed and unexposed animals determined using the Logan Reference Plot and the cerebellar cortex reference region rLP(CER).

The authors then proceed to report on: “**[¹¹C]DPN binding is influenced by vaccine**

exposure and amygdala volume”, “Total Amygdala [¹¹C]DPN Binding”, “Left Amygdala [¹¹C]DPN Binding”, and “Right Amygdala [¹¹C]DPN Binding” in a series of brief paragraphs and to end by summarizing their “[¹¹C]DPN binding” findings as follows:

“In summary, at T1 there was a significant effect of exposure on total brain volume (exposed > unexposed), but no difference in amygdala volume between groups. Changes occurring between T1 and T2 include a differential change in total amygdala volume between groups (a significant decrease in unexposed animals compared with a non-significant increase in exposed animals) after adjustment for total brain volume, and an increase in [¹¹C]DPN binding in left amygdala compared with a decrease in binding in unexposed animals, after adjusting for amygdala volume.”

Having presented their results, the authors next discussed their results in a passage that ends with:

“If, for example, exorphines such as β -caseomorphine have a role in this model, either acting as partial or selective antagonists, or they exert a negative effect on endogenous opioidergic systems (LaBella et al. 1985), they might inhibit a maturational decline in opioid receptors and account for the sustained avidity of the amygdala for [¹¹C]DPN in exposed animals. How these effects could be potentially mediated by vaccine exposure is not known. Additional histologic and molecular analyses of the amygdala may provide mechanistic insights.”

Finally, these researchers present their conclusions as follows:

“In this pilot study, infant macaques receiving the recommended pediatric vaccine regimen from the 1990’s displayed a different pattern of maturational changes in amygdala volume and differences in amygdala-binding of [¹¹C]DPN following the MMR/DTaP/Hib vaccinations between T1 and T2 compared with non-exposed animals. There was also evidence of greater total brain volume in the exposed group prior to these vaccinations suggesting a possible effect of previous vaccinations to which these animals had been exposed. Because primate testing is an important aspect of pre-clinical vaccine safety assessment prior to approval for human use ..., the results of this pilot study warrant additional research into the potential impact of an interaction between the MMR and thimerosal-containing vaccines on brain structure and function. Additional studies are underway in the primate model to investigate the mechanistic basis for this apparent interaction.”

This reviewer can only note that these researchers’ concluding remarks are simply restating the findings of the observations in other papers as well as in human patient work-ups where prior adverse vaccination history most certainly had a significant negative impact on the outcomes observed following subsequent vaccinations.

9. DeSoto MC, Hitlan RT. Sorting out the spinning of autism: heavy metals and the question of incidence. *Acta Neurobiol Exp* 2010, 70(2): 165–176. [<http://www.ane.pl/pdf/7021.pdf>]

“The reasons for the rise in autism prevalence are a subject of heated professional debate. Featuring a critical appraisal of some research used to question whether there is a rise in cases and if rising levels of autism are related to environmental exposure to toxins (Soden et al. 2007, Thompson et al. 2007, Barbaresi et al. 2009) we aim to evaluate the actual state of scientific knowledge. In addition, we surveyed the empirical research on the topic of autism and heavy metal toxins. Overall, the various causes that have led to the increase in autism diagnosis are likely multi-faceted, and understanding the causes is one of the most important health topics today. We argue that scientific research does not support rejecting the link between the neurodevelopmental disorder of autism and toxic exposures”.

Key words: autism, autism prevalence, heavy metals, mercury, toxins”

In simple terms, this article addresses the “spinning”(slanting/distorting), *intentional or not*, of the probable causal links for the clinical symptoms that are used to diagnose an “autism spectrum disorder” to obscure the realities that there has been an epidemic increase in the incidence of children who have an ASD, related neurodevelopmental or related behavioral disorder, syndrome, or disease that has been caused, and is being supported, by the use of Thimerosal-preserved vaccines that, at a given level of sub-acute exposure, clinically mercury-poison some of those vaccinated, who are, for whatever reasons (genetic, dietary, medical or environmental), sufficiently susceptible to early developmental mercury poisoning by Thimerosal, an insidiously poisonous organic mercury toxin, still used as a preservative in many vaccines repeatedly administered to pregnant women and developing children.

Reviewer's Concluding Observations

Given the scientific evidence in all of the preceding case-matched-control studies and the other studies and review articles, it seems clear that Thimerosal (49.55% mercury by weight) used as a preservative in vaccines has been, and still is, the iatrogenic agent knowingly used by those, *who profit from an increasing percentage of our children's having one or more chronic diseases*, to intentionally injure an increasing percentage of our children to increase their customer base, their profit and/or their profit margins at the expense of the fiscal and physical health of ourselves and our children.

Given these additional findings, **it is time to demand that all those who have been, or are, involved in supporting the knowing use of Thimerosal as a preservative in vaccines without proof of safety to the standard “sufficiently nontoxic ...”** (more than 10 times below the lowest NOAEL for injected Thimerosal in the most susceptible segment of the human population), *as set forth in 21 CFR § 610.15(a)*¹, **be prosecuted under the “intent to defraud or mislead” provisions in 21 U.S.C. § Sec. 333(a)**².

¹ Compliance with 21 CFR § 610.15(a) for Thimerosal-preserved vaccines is an absolute nondischargeable duty that the makers of Thimerosal-preserved vaccines and other drugs have to perform or else their Thimerosal-preserved vaccines are adulterated drugs under 21 U.S.C. § 351(a)(2)(B). Further, the introducing or causing the introduction of such adulterated drugs into commerce is a Prohibited Act (see 21 U.S.C. § 331) and there are civil and criminal penalties that apply to all those who are, *in any responsible or accountable manner*, engaged in violating 21 U.S.C. § 331 Prohibited Acts (see 21 U.S.C. § 333 Penalties [for § 331's “Prohibited Acts” see § 331(a) “The following acts and the causing thereof are prohibited: (a) The introduction or delivery for introduction into interstate commerce of any food, drug, device, or cosmetic that is adulterated or misbranded”.]).

² 21 U.S.C. § 333(a)

“Violation of section 331 of this title; second violation; intent to defraud or mislead

(1) Any person who violates a provision of section 331 of this title shall be imprisoned for not more than one year or fined not more than \$1,000, or both.

(2) Notwithstanding the provisions of paragraph (1) of this section, (!1) if any person commits such a violation after a conviction of him under this section has become final, or commits such a violation with the intent to defraud or mislead, such person shall be imprisoned for not more than three years or fined not more than \$10,000, or both. (!1) So in original. Words ‘of this section’ probably should not appear.”