IN THE UNITED STATES COURT OF FEDERAL CLAIMS
OFFICE OF SPECIAL MASTERS

N RE: CLAIMS FOR VACCINE INJURIES RESULTING IN AUTISM SPECTRUM DISORDER, OR A SIMILAR NEURODEVELOPMENTAL DISORDER, Various Petitioners, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Autism Master File **PSC MOTION FOR RECONSIDERATION: TOXICOLOGY REBUTTAL TESTIMONY AND EVIDENCE** **Special Master George Hastings**

MOTION

Petitioners move the Special Masters 1) to reconsider their orders of June 17 and July 3, 2008 barring rebuttal testimony regarding toxicology issues in the "thimerosal only" test cases, and 2) for leave to file a brief rebuttal report from petitioners' toxicology expert witness Dr. Vas Aposhian.

FACTS

Respondent represented during the hearings of the Mead and King "thimerosal only" test cases that two of its expert toxicologists (Drs. Clarkson and Magos) would not appear to testify and be cross-examined during the hearing of the general causation evidence in those test cases between May 12 – 30, 2008. Instead, respondent said the two witnesses would appear during the hearing of the third test case scheduled for the week of July 21 – 25, 2008. Respondent did call another toxicologist, Dr. Jeffrey Brent, to testify during the Mead and King hearings.

At the conclusion of Dr. Mumper's testimony on May 16, petitioners indicated that they intended to offer rebuttal testimony in response to all of respondent's toxicology testimony—Dr. Brent, Magos and Clarkson—at one time, in July, after Drs. Magos and Clarkson testified.

Petitioners, in fact, proposed that *all* of their general causation rebuttal be presented at one time in July after Drs. Magos and Clarkson testified, not just toxicology rebuttal. Respondent objected, and a lengthy discussion on the record ensued. (See, Transcript, pp. 1678-1690). No ruling on the scope, timing, or sequence of rebuttal was made at that point.

When the hearing reconvened on May 19, the rebuttal issue came up again. (pp.2039-2047). In those discussions, respondent indicated that while it maintained its objection to petitioners' presenting all of their rebuttal testimony on all issues in July, there was no problem with petitioners presenting all of their *toxicology rebuttal only* in July:

> "Now, if Dr. Aposhian wants to wait until July to put together his rebuttal to Dr. Brent as well as any potential rebuttal he may have to Drs. Clarkson and Magos, that's not beyond what respondent believed that's the procedure the court had in mind in the first place."
>
> Transcript, p. 2040.

This approach would mean that petitioners wouldn't have to call Dr. Aposhian on two different occasions; that is, at the conclusion of the Mead and King hearings, and again at the conclusion of Drs. Magos' and Clarkson's appearances in July. This is what petitioners now propose to do through the presentation of Dr. Aposhian's brief rebuttal report. Petitioners do not object to respondent's filing of a sur-rebuttal if needed.

Further discussions regarding the scope and timing of rebuttal testimony followed the discussion on May 19, as counsel for the parties conferred and the Special Masters discussed the issue off the record, with counsel, in chambers.

During these discussions, petitioners understood the contentious issue to be whether rebuttal testimony in July would include non-toxicology topics, and particularly whether petitioners would offer rebuttal testimony from Dr. Kinsbourne or any other witness concerning neurology issues generally. After respondent's representations on May 19 that putting on rebuttal to Drs. Magos, Clarkson and Brent in July was not a problem, petitioners were focused on resolving the question of whether rebuttal relating to other topics and witnesses should proceed in July, or at the conclusion of the May hearings. Petitioners' recollection of the off-record discussions is that the Special Masters decided to require that all non-toxicology rebuttal

testimony would be presented at the conclusion of the May hearings, but that Dr. Aposhian could testify in rebuttal in July regarding all toxicology issues, including those raised by Dr. Brent, a position consistent with the discussion on the record on May 16. Petitioner agreed that any rebuttal testimony on issues other than toxicology would need to be presented during the final week of the Mead and King hearing.

Petitioner understands that the Special Masters then made a record of the May 19 in-chambers conference at the beginning of the day on May 20, and that in describing their ruling the Special Masters explicitly stated that rebuttal in July would be "strictly" limited to the testimony of Drs. Clarkson and Magos, and not to any other testimony. Petitioner agreed that the Special Masters accurately described the conference. Petitioners apparently misunderstood or misconstrued the Special Masters' June 20 recorded decision, however, because petitioners were still under the impression that Dr. Aposhian could return in July to present rebuttal testimony to Drs. Brent, Magos and Clarkson, and therefore petitioner did not arrange for Dr. Aposhian to appear at the conclusion of the Mead and King hearing to testify regarding Dr. Brent.

After the King and Mead hearings concluded, respondent announced that it would not call either Dr. Magos or Dr. Clarkson to testify, denying petitioner the opportunity to cross-examine the two witnesses. Respondent has also moved to withdraw the already filed reports of Drs. Magos and Clarkson. In an order on July 3, 2008, the Special Masters concluded that petitioners would not be permitted to offer any additional rebuttal testimony on general causation issues during the third test case hearing because respondent was not making two toxicology experts (Drs. Clarkson and Magos) available to testify, refusing to allow them to be cross-examined, and because respondent was moving to "withdraw" the reports of those two witnesses already filed as evidence in the omnibus proceeding.

ARGUMENT

Petitioners should be allowed leave to file a rebuttal report by Dr. Aposhian because the information in the report is reasonably necessary for the Special Masters to consider in evaluating the extensive toxicology evidence in the general causation inquiry. Petitioners submit

that the misunderstanding or error of their counsel in not bringing Dr. Aposhian to the stand in rebuttal of Dr. Brent's testimony in May should not be allowed to prejudice the presentation of important evidence that Dr. Aposhian would have offered if he had appeared in May. Petitioners attach a copy of Dr. Aposhian's rebuttal report as Exhibit 1 to this motion as an offer of the proof that would be adduced if leave is given to file the report.

Petitioners should also be allowed to present this evidence because respondent's late withdrawal of the witnesses, and their highly irregular motion to withdraw the reports that already are evidence in the proceeding, is a thinly-veiled attempt to deprive petitioners of the opportunity to develop the facts of these cases through cross-examination. Upon cross-examination of Drs. Magos and Clarkson, petitioners would likely elucidate testimony admitting (based on the literature published by each of the witnesses) that: Hg++ is toxic to brain cells (Clarkson 2005 PMRL 0026), the human brain to blood ratio is 6.0 (Magos CV, Respondent's Ex Z, #158 on publications list), the macaque brain to blood ratio is only 2.6 (Clarkson 2005 PMRL 0026), the infant macaques had blood levels comparable to human infants in the Pichichero and Stajich studies, and therefore, it is reasonable to conclude that brain levels of Hg++ in some human infants are in the same range that ignited neuroinflammation in the adult macaques.

While unable to pursue these issues upon cross-examination of witnesses that petitioners have prepared for several months because respondent is making the witnesses unavailable, petitioners should, in the interest of fairness, be permitted to complete the toxicology record in these proceedings by admitting into evidence Dr, Aposhian's rebuttal report.

For these reasons, the Specials Masters should reconsider their orders of June 17 and July 3 and allow Dr. Aposhian's report into evidence in this proceeding.

DATED this 28th day of July, 2008.

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CERTIFICATE OF SERVICE

I hereby certify that on July 28, 2008, I served the foregoing **PSC MOTION FOR RECONSIDERATION: TOXICOLOGY REBUTTAL TESTIMONY AND EVIDENCE** on the following individual(s):

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By UPS, next business day delivery.

Petitioners specifically authorize the Court and the Office of Special Masters to post this document, and any attachments or exhibits thereto, on the Court/OSM website, expressly waiving any confidentiality as to the contents of these materials. Petitioners expressly wish to publicly disclose this filing in any other forum designated by the Court or the OSM.

WILLIAMS LOVE O'LEARY & POWERS, P.C.

Michael L. Williams
Of Attorneys for Petitioners' Steering Committee

Supplemental Report in lieu of rebuttal testimony:

Mercuric mercury in the developing brains of young children exposed to thimerosal-containing vaccines.

US Federal Court of Claims, Vaccine Trial

July 3, 2008

by

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EXHIBIT 1
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I. Introduction

The major purpose of this report is to rebut the claim of Dr. Jeff Brent that there would be insufficient amounts of Hg^{++} in the brains of infants exposed to TCV's to trigger a neuroinflammatory process, and that the amount of Hg^{++} from breast milk would result in greater amounts of Hg^{++} than that derived from TCV's. In so doing, I will present to the court the estimated concentration of mercuric mercury in brains of human infants who received thimerosal-containing vaccines. In addition, other relevant facts will be presented.

It is well known to biochemists and toxicologists that mercuric mercury (also designated as Hg^{2+}, or sometimes described as inorganic mercury) is chemically very reactive in the brain of human infants that have received thimerosal-containing vaccines. In addition, mercuric mercury has a strong affinity for and combines with thiol (sometimes called sulfhydryl or -SH) groups of brain proteins resulting in the inhibition of crucial enzymes or of proteins that are part of the brain's structure and cytoarchitecture (Nordberg et al., 2007, PMRL0213).

When thimerosal-containing vaccines are administered to humans the thimerosal is quickly biotransformed in the tissues to ethylmercury which is then converted by oxidation to mercuric mercury (Clarkson and Magos, 2006, PMRL0035).

In the case of the brain, once the ethylmercury enters the brain it is either released from the brain or converted to mercuric mercury in the brain (Burbacher et al., 2005, PMRL0026). Ethyl mercury is released from the brain at a rate faster than is methyl mercury but the conversion in the brain of ethyl mercury to mercuric mercury occurs more rapidly than the conversion of methyl mercury to mercuric mercury.

When the amount of Hg in blood and brain of primates and humans is determined in controlled experiments,, wide variations among individuals, often by an order of magnitude, have been found in every study done (Burbacher et al., 2005, PMRL0026).

II. Brain Mercury Levels

Burbacher et al., compared the blood and brain mercury levels in infant monkeys exposed to methyl mercury and vaccines containing thimerosal. The $t_{1/2}$ values for ethyl mercury blood levels in infant monkeys (Burbacher et al., 2005, PMRL0026) and humans (Pichichero et al., 2002, PMRL0223, and 2008, PMRL0497) are quite similar.

Stereologic and autometallographic studies of the brains of adult monkeys chronically exposed to methyl mercury (Vahter et al., 1994, 1995, PMRL0060, PMRL0064) demonstrated that the persistence of inorganic mercury in the brain was associated with an increase in the microglia in the brain but the number of astrocytes decreased. Mercuric mercury can cause gliosis (Davis 1994, PMRL0183). The effects in the adult monkeys were associated with brain inorganic mercury levels on average five times higher (Charleston et al., 1994, 1995, 1996, PMRL0033, PMRL0032, PMRL0116) than those associated with the infant monkeys (Burbacher et al., 2005, PMRL0026).

The total Hg in brain of the MeHg fed infant monkeys was about 105 ng/g at peak, and dropped only to about 90 at the end of the experiment. At that point in time the average Hg^{++} concentration was about 6 or 7 ng/g, but was below detection limits in 8 of 17 monkeys. The text calculates that only about 6-10% of total Hg was converted to Hg^{++} in these animals.

Dr. Brent argues that because there was still almost 90 ng/g of organic MeHg in the infant monkey brains at end of experiment, much would still be converted to Hg^{++}. However, if the conversion rate is only 6-10%, then the total converted would be at most 5-9 more ng/g of additional Hg^{++} once the conversion of MeHg to Hg^{++} was fully made in the brain.

Using the same analysis on the TCV infant monkeys, they had 16ng/g of Hg^{++} on average, with about 8 ng/g of organic Hg left at the end of experiment; if 34% were converted (the conversion % for ethyl mercury to Hg^{++} as found by Burbacher et al, 2005), that would add another 3 ng to the 16, leaving around 19 ng/g on average.

Now, since the human brain/blood ratio is 2.3 times higher than for monkeys (per Magos 1987, PMRL0666 as cited in Burbacher 1990, PMRL0224): the formula is 6.0/2.6=2.3), then the 19 is multiplied by 2.3 = 44 ng/g.

We are now already to a level about 73% of the amount of Hg necessary to set off neuroinflammation in the adult monkeys, which was as low as 60 ng/g—see table Table 2 in Vahter 1994 "Speciation of Hg in Primate blood and brain." (PMRL0060)

See also the text on p. 203 of Charleston 1994 (PMRL0033), "Increases in the number of reactive glia . . .", where it says that even the I-Hg-fed monkeys, which had the lowest levels of Hg^{++} in brain, still had lots of reactive microglia (left hand column); note that in animal 82177, one of those fed HgCl, the level of Hg^{++} was only .06 micrograms/g, or 60 ng/g, very, very close to the projected human value for TCV's. (To convert mcg/g to ng/g, one multiplies the number of mcg's by 1000; hence, .06 x 1000 = 60.)

It is likely that the rapidly developing human brain from birth to 1.5 years of age is more sensitive to neuroinflammation than a mature adult brain, just because the microglia and astrocytes are so involved in the orchestration of the complex and rapid growth of connections. In other words, just because the adult monkeys showed no symptoms despite confirmed chronic, active neuroinflammation there should not be an assumption the same kind of neuroinflammation in an infant's developing brain is harmless. One would expect that the developing brain of an infant would have more developmental processes and events occurring than the adult brain. It needs to be remembered that in the Minamata methylmercury spill disaster, severely damaged central nervous systems occurred in children born to mothers without symptoms.

III. Brain cumulative inorganic mercury levels based on USA children from Pichichero et al, 2002

I have used the only human infant ethylmercury blood data we have after TCV's, from the two Pichichero studies (PMRL0223, PMRL0497) and the Stajich study (PMRL0249), to calculate

what the likely concentration of Hg^{++} in the brain of human infants would be. Those calculations are set out in Table 1 of this supplemental report.

The estimated uncorrected cumulative brain inorganic Hg content is 5.2 X 7 = 36.4 ng inorganic Hg/g brain tissue plus an injection-collection correction factor ***of 20% or giving a corrected estimated cumulative value of 43.7ng inorganic Hg per g brain tissue. 7.3 ng

It needs to be clearly understood that the basis for the conclusions of the below data in the following table is:
a)- thimerosal is metabolized to ethyl mercury.
b)- ethyl mercury is metabolized to mercuric mercury
c)- mercuric mercury is tenaciously held in the brain for years.
The above statements can be verified in the following references: Clarkson TW and Magos L. 2006 (PMRL0035); Burbacher et al., 2005 (PMRL0026); *Handbook Of The Toxicology Of Metals* edited by Nordberg, Fowler, Nordberg and Friberg, Academic Press 2007 (PMRL0213).

Table 1- BRAIN INORGANIC AND TOTAL MERCURY LEVELS AT IMMUNIZATON TIMES 2 MONTHS AND 6 MONTHS
(This TCV schedule does not include flu vaccine)

TIME	VACCINE	Hg Dose mcg	Cum. Dose mcg	Blood Hg nmol/L	Blood Hg ng/ml	Brain Hg ng/ml - *Total	Brain Hg ng/ml - *Inorganic
Birth	HepB #1	12.5	12.5				
1 mo.	HepB #2	12.5	25				
2 mos.	DTP #1	25					
	HIB #1	25	75	**20.55	4.12	24.7	8.4
4 mos.	DTP #2	25					
	HIB #2	25	125				
6 mos.	DTP #3	25					
	HIB #3	25					
	HepB #3	12.5	187.5	**6.9	1.4	8.4	2.0
12-18	DTP #4	25					
	HIB #4	25	237.5				
4-5 yrs	DTP #5	25					
	HIB #5	25	287.5				
Sum of brain mercury based on only 2 month value and 6 month value above						33.1	10.4
The average, ng /ml, of the above 2 and 6 months values for **inorg** Hg							5.2
***Corrected estimated cumulative value of 43.7ng inorganic Hg per g brain tissue							43.7

**For these calculations the infant blood mercury levels after vaccination were taken from Pichichero et al., 2002 (PMRL0223). A brain/blood ratio for mercury in the human of 6.0 was used (Magos, 1987, PMRL0666); and from Burbacher et al., 2005 (PMRL0026) 34% was used as the percentage that inorganic Hg was of the total Hg in the brain of infant monkeys receiving TCVs.*

***Highest value from taken from Pichichero et al., 2002 (PMRL0223). . However, the collection of blood in this infant took place five days after vaccination, so the blood levels had to be higher on days 1 –2 than when measured; this is also true of all the blood measurements in the study—they were taken on average several days after*

vaccination. Therefore, to use the data in the Pichichero study will lead to a significant underestimate of the total Hg in blood, and thus in the proportion going into the brain, in the day or two after vaccination when blood levels are highest. Thus an injection-collection correction factor is necessary, and I think a reasonable estimate would be to project that blood levels at their peak post vaccination would be about 20% higher..

****Vaccines were given at 7 different times. Therefore estimated cumulative brain inorganic Hg content is 5.2 X 7 = 36.4 ng inorganic Hg/g brain tissue plus an injection-collection correction factor of 20% or 7.3 ng giving a corrected estimated cumulative value of 43.7ng inorganic Hg per g brain tissue.*
I have assumed throughout that 1 ml brain tissue = 1 gram brain tissue. Such an assumption was obviously made in the Burbacher et al., 2005 (PMRL0026).

IV. Brain cumulative inorganic mercury levels based on USA children from Pichichero et al, 2008

If the same sort of calculations are done using the highest blood concentrations of the Pichechero 2008 paper (PMRL0497) the uncorrected, incremental brain **inorganic mercury** concentrations of the highest outliers are:
Newborns.....17.1 ng/ml brain tissue
2 month olds.....10.2
6 month olds.....10.0

The final corrected values are shown in Table 2.

Table 2 High end of the doses of Hg^{++} in human infant brains

TIME	*Blood – total Hg	Brain Total Hg	Brain inorganic Hg^{++}	**Corrected Brain inorganic Hg^{++}
New born	7.9 ng/ml	47.4 ng/ml	17.1 ng/g	20.5 ng/g
2 mos.	5.0 ng/ml	30.0 ng/ml	10.2 ng/g	12.2 ng/g
6 mos.	4.9 ng/ml	29.4 ng/ml	10.0 ng/g	12 ng/g
Cumulative corrected brain inorganic Hg				44.7 ng/g

**For these calculations the infant blood mercury levels after vaccination were taken from Pichichero et al., 2008 (PMRL0497). A brain/blood ratio for mercury in the human of 6.0 was used (Magos, 1987, PMRL0666); and from Burbacher et al., 2005 (PMRL0026), 34% was used as the percentage that inorganic Hg was of the total Hg in the brain of infant monkeys. However, these values are from Argentinian children who received TCVs according to a different vaccination protocol than USA children (Pichichero et al., 2008).*

*** see legend of table 1 for definition of corrected value.*

The paper by Vahter 1994 "Speciation of Hg in Primate blood and brain." (see table 2)states that 60ng of Hg^{++}/g (or inorganic Hg/g) will cause neuroinflammation in the brain. This is remarkably close to the 43.7 and 44.7 ng inorganic Hg that we have determined independently.

V. Additional Rebuttals of Dr. Brent's Testimony

Since my testimony in May, there have been some published studies that back up my opinions and which tend to refute those of Dr. Brent.

First, with respect to evidence for a mercury efflux disorder in autistic children, the Holmes et al (PMRL0237) studies indicating that autistic children have less mercury in their hair indicating that they have an efflux disorder has been confirmed recently by Adams et al., 2008 (PMRL0667).

Citations for evidence that autistic children have less mercury in their hair and bodies and support the concept that a mercury efflux disorder is involved in some or all autism cases.

1. **Holmes et al., 2003. (PMRL0237)**
2. **Hu et al., 2003 (PMRL0016)**
3. **Bradstreet et al., 2003 (PMRL0244)**
4. **Adams et al., 2007 (PMRL0138)**
5. **Adams et al., 2008 (PMRL0667)**

Adams et al., 2008 demonstrated that at hair mercury concentrations of below 0.55μg/g, children are 2.5 times more likely to manifest autism. This study was done in collaboration with people at the National Inst of Health. This supports the initial paper of Holmes et al., 2003 (PMRL0237) and Hu et al., 2003 (PMRL0016). All three studies plus the Bradstreet et al., report together show that autistic children tend to be slow excretors of Hg.

Dr. Brent and other DOJ witnesses criticized the Hornig mouse study (PMRL0015) model of thimerosal leading to autistic symptoms in animals, but since then there has been publication of another good animal model of EtHg toxicity from TCV's: the Peruvian hamster study by Laurente et al. (PMRL0668).

Dr. Brent also criticized the Bradstreet 2003 chelation challenge study (PMRL0224) for having no pre-challenge results and for not having any standard reference for post-chelation results. However, traditionally there have been two ways of doing challenge tests: (1) a control group also is given the challenge, or (2) a pre-challenge urine collection is begun on the subjects 6 hrs before the challenge. Experienced investigators know that when working with autistic children it is difficult to get a pre-challenge collection for 6 hrs and then give them the challenge and again collect urine, so they used the control protocol. This was a reasonable and valid way to do the study.

It is pertinent to note that Windham et al., 2006 (PMRL0018) in their conclusions "suggest a potential association between autism and estimated metal concentrations, and possibly solvents, in ambient air around the birth residence..." and that since my testimony in May, Windham et al. have refined their data to show a statistically significant correlation between distance from the Hg release point source and the rate of autism four years later. Windham et al., 2008 (PMRL0670).

Dr. Brent cited a study from Brazil for his claim that the average exposure to MeHg ingested from breast milk in the first six months was about 280 micrograms. About 95% of the MeHg is absorbed by the gut (266 micrograms). A certain percentage of this will be delivered to the brain. We are not certain as to how much because during this period the infant blood brain barrier is not in its mature barrier form. Note, however, that the Hg++ delivered to an infant's brain from TCV's would only add to the total Hg++ in the brain, thus making TCV's even more dangerous to infants whose mothers have MeHg in breast milk.

In summary, it is my opinion to a reasonable degree of scientific certainty, that in some infants receiving the normal schedule of TCV's in the mid 1990's in the USA, there would be sufficient concentrations of Hg++ deposited in their brains to trigger the same kind of neuroinflammation and other brain cell changes seen in the adult monkeys exposed to MeHg. Given the fact that many infants will already be exposed to some Hg++ in their brains from breast milk, and ambient air sources, it is even more likely that the additional amount of Hg++ from TCV's would push some kids over the toxic threshold.

Signed: H. Vasken Aposhian Date: 7/8/2008

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REFERENCES

PMRL #	AUTHOR	TITLE	CITATION	DATE
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
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
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
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.;	12/1/1994
0035	Clarkson TW, Magos L	The toxicology of mercury and its chemical compounds.	Crit Rev Toxicol. 2006 Sep;36(8):609-62	1/1/2006
0060	Vahter M, Mottet NK, Friberg L, Lind B, Shen DD, Burbacher T	Speciation of mercury in the primate blood and brain following long-term exposure to methyl mercury	Toxicol Appl Pharmacol. 1994 Feb;124(2):221-9	2/1/1994
0064	Vahter ME, Mottet NK, Friberg LT, Lind SB, Charleston JS, Burbacher TM	Demethylation of methyl mercury in different brain sites of Macaca fascicularis monkeys during long-term subclinical methyl mercury exposure	Toxicol Appl Pharmacol. 1995 Oct;134(2):273-84	10/1/1995
0076	Li Z, Dong T, Proschel C, Noble M	Chemically diverse toxicants converge on Fyn and c-Cbl to disrupt precursor cell function	PLoS Biol. 2007 Feb;5(2):e35	2/1/2007

0116	Charleston JS, Body RL, Bolender RP, Mottet NK, Vahter ME, Burbacher TM. ;	Changes in the number of astrocytes and microglia in the thalamus of the monkey Macaca fascicularis following long-term subclinical methylmercury exposure.; ;	Neurotoxicology. 1996 Spring;17(1):127-38. ; ;	4/1/1996
0138	Adams JB, Romdalvik J, Ramanujam VM, Legator MS. ;	Mercury, lead, and zinc in baby teeth of children with autism versus controls.; ;	J Toxicol Environ Health A. 2007 Jun;70(12):1046-51. ;	6/1/2007
0183	Davis LE, Kornfeld M, Mooney H, Fielder KJ, Haaland KY, Orrison WW, Cernichiari E, Clarkson TW	Methylmercury Poisoning: Long-Term Clinical, Radiological, Toxicological, and Pathological Studies of an Affected Family	Annals of Neurology;35(6): 680-6	6/1/1994
0213	Nordberg, GF, Fowler BA, Nordberg M, Friberg LT.	Handbook on the toxicology of metals,	3d edition, Academic Press 2007	
0223	Pichichero ME, Cernichiari E, Lopreiato J, Treanor J	Mercury concentrations and metabolism in infants receiving vaccines containing thiomersal: a descriptive study	Lancet;360(9347): 1737-40	11/30/2002
0237	Holmes AS, Blaxill MF, Haley BE	Reduced Levels of Mercury in First Baby Haircuts of Autistic Children	International Journal of Toxicology;22(4): 277-85	7/1/2003
0244	Bradstreet J, Geier DA, Kartzinel JJ, Adams JB, Geier MR	A Case-Control Study of Mercury Burden in Children with Autistic Spectrum Disorders	Journal of American Physicians and Surgeons;8(3): 76-9	6/1/2003
0497	Pichichero ME, Gentile A, Giglio N, Umido V, Clarkson T, Cernichiari E, Zareba G, Gotelli C, Gotelli M, Yan L, Treanor J.	Mercury levels in newborns and infants after receipt of thimerosal-containing vaccines.	Pediatrics. 2008 Feb;121(2):e208-14.	2/1/2008
0666	Magos L, Clarkson TW, Sparrow S, Hudson AR	Comparison of the protection given by selenite, selenomethionine and biological selenium against the renotoxicity of mercury	Archives of Toxicology;60(6): 422-26	8/1/1987
0667	Adams JB, Romdalvik J, Levine KE, Hu LW	Mercury in first-cut baby hair of children with autism versus typically-developing children.;	Toxicol Environ Chem 2008, May 2008 (Abstract)	5/1/2008
0668	Laurente J, Remuzgo F, Avalos B, Chiquinta J, Ponce B, Avendano R, Maya L	Neurotoxic effects of thimerosal at vaccines doses on the encephalon and development in 7 days- old hamsters.	Anales de la facultad de Medicina 2007;68(3):222-237	5/1/2008
0670	Windham G, Fenster L.	Environmental contaminants and pregnancy outcomes.	Fertil Steril. 2008 Feb;89(2 Suppl):e111-6;	2/1/2008