UNITED STATES
COURT OF FEDERAL CLAIMS

IN RE: CLAIMS FOR VACCINE INJURIES RESULTING IN AUTISM SPECTRUM DISORDER, OR A SIMILAR NEURODEVELOPMENTAL DISORDER	)	
FRED AND MYLINDA KING, PARENTS OF JORDAN KING, A MINOR, Petitioners,	)	
v.	)	Docket No.: 03-584V
SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	)	
GEORGE AND VICTORIA MEAD, PARENTS OF WILLIAM P. MEAD, A MINOR, Petitioners,	)	
v.	)	Docket No. 03-215V
SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	)	

REVISED AND CORRECTED COPY

Pages: 1456 through 1693/1775

Place: Washington, D.C.

Date: May 16, 2008

HERITAGE REPORTING CORPORATION
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IN THE UNITED STATES COURT OF FEDERAL CLAIMS
OFFICE OF SPECIAL MASTERS

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SECRETARY OF HEALTH AND HUMAN SERVICES,	)	
Respondent.	)	

Courtroom 402
National Courts Building
717 Madison Place NW
Washington, D.C.

Friday,
May 16, 2008

The parties met, pursuant to adjournment, at 9:05 a.m.

BEFORE: HONORABLE GEORGE L. HASTINGS, JR.
HONORABLE PATRICIA E. CAMPBELL-SMITH
HONORABLE DENISE VOWELL
Special Masters

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C O N T E N T S

WITNESSES:	DIRECT	CROSS	REDIRECT	RECROSS	VOIR DIRE
For the Petitioners:					
Elizabeth Mumper	--	1460	1625	1669	--

E X H I B I T S

RESPONDENT'S EXHIBITS:	IDENTIFIED	RECEIVED	DESCRIPTION
Trial Exhibit:			
2	1563	--	Letter from N.Y. Department of Health
3	1566	--	Letter from N.Y. Department of Heath, 2006

P R O C E E D I N G S

(9:05 a.m.)

SPECIAL MASTER CAMPBELL-SMITH: We are back on the record. I understand from counsel there are no preliminary matters to be addressed. We are in a position to resume with the cross-examination of Dr. Mumper.

Good morning, Dr. Mumper. You continue to be under oath.

Whereupon,

ELIZABETH MUMPER

having been previously duly sworn, was recalled as a witness herein and was examined and testified further as follows:

CROSS-EXAMINATION (Resumed)

BY MR. JOHNSON:

Q Good morning, Doctor.

A Good morning.

Q When we wrapped up yesterday, we were talking about porphyrin testing, and I want to go back to that issue just briefly.

This is not the first time you've testified as an expert in a case involving autism and mercury, is that correct?

A That is correct.

Q You testified in a case called Blackwell v. Sigma Aldridge in Baltimore, is that right?

A That's correct.

Q And do you remember giving a deposition in that case?

A Yes, I do.

Q And that was in January of 2007?

A Yes.

Q And you also testified at an evidentiary hearing held in August of 2007, is that right?

A That's correct.

Q At the hearing, the evidentiary hearing, do you remember being asked about laboratory results for biomarkers that you look for in your patients to determine if they are harmed by mercury?

A I'm sure that I was, but it's been so long that I would appreciate it if you would flash the testimony up.

Q We'd be happy to.

And do you remember testifying, or actually why don't you just read the highlighted portion of your testimony.

A "Probably the most helpful test to me now is a porphyrin test, and the reason I like the porphyrin test is that it actually looked at the impact of ethyl

mercury and other heavy metals on body biochemistry and body physiology."

I realize now I should have specified the impact of mercury because it does not distinguish ethyl from other forms.

Q Is it now your testimony that the porphyrin test is not the most helpful test for determining if your patients are harmed by mercury?

A As the science as evolved, we have continued to use other measures also. What we have now that I didn't know about back then are also some inflammatory markers in urine specimens that look for co-existing inflammation, that, in addition, is very helpful, but I do continue to use the porphyrins quite a bit, yes.

Q You mentioned testing for inflammatory markers in urine. Who does that testing?

A Dr. Nataf's lab does that testing, but we are also able to get it from Metametrix and other labs probably that I don't know of.

Q The porphyrin test is a test that you are still ordering in your practice, is that right?

A That's correct.

Q And it's a test that you are still using to make clinical decisions regarding your patients treatment and care, is that accurate?

A Yes, using it in context with the most important part, which is the history of the child and the clinical appearance of the child.

Q You testified yesterday that you could not tell me how long after an exposure you would still consider the porphyrin test to be reliable.

A That's correct.

Q If you're using the test to make treatment decisions, you must have some idea of when the test is medically appropriate, wouldn't that be correct?

A When I use the test, I am looking at the impact of a particular child with regard to impact on a very crucial biochemical pathway at the time that I get that child. I don't have the advantage of getting him or her at a time that I can choose with regard to any exposures, and often I am trying to figure out what the exposures are.

So, I think we have already established that I'm not a toxicologist, so I don't want to venture beyond my area of expertise to speculate about how that test is constructed in terms of the question that counsel asked me. But I am still able to use it, I think, as a clinically valuable tool as long as I use it in the context of the individual patient, the clinical history, and other supporting laboratory data

that might be consistent with what I see on the porphyrin test.

Q You wouldn't order that test unless it was medically appropriate or clinically indicated, is that accurate?

A Unless my judgment was that that was the case, yes.

Q And how long after an exposure, in your judgment, would you personally not order a porphyrin test based on a belief that it would no longer be a reliable measure of, or reliable evidence that your patient was harmed by mercury?

A I do not have that number and do not wish to speculate about it.

Q So you are unwilling to provide me with a timeframe on that question?

A No. I'm willing to provide you with any information that I feel is within the realm of my expertise as an clinician. It seems to me that I'm being asked to venture into territory that we've already established through DOJ's help is not my area of expertise, i.e., toxicology and laboratory science. So, I wish to be able to confine my testimony to areas of clinical expertise.

Q But you do basic clinical decisions based on

your interpretation of the those test results, is that right?

A That is correct.

Q Doctor, you included in your William Mead report a paragraph that discussed neuroinflammation, and this is on pages 7 to 8 of your report.

SPECIAL MASTER HASTINGS: In which case?

MR. JOHNSON: In the William Mead case.

SPECIAL MASTER HASTINGS: In the Mead case.

THE WITNESS: Can you tell me the bolded title on that page because mine isn't paginated?

SPECIAL MASTER CAMPBELL-SMITH: Analysis of William Mead's Clinical and Laboratory Evidence --

THE WITNESS: Thank you.

SPECIAL MASTER CAMPBELL-SMITH: -- With Regard to the Medical Literature.

MR. JOHNSON: Thank you, Special Master. That's it.

THE WITNESS: Okay. Thank you.

BY MR. JOHNSON:

Q All right. And in that paragraph you refer to the Vargas article, correct?

A That is correct.

Q Okay, and that's Petitioner's Master List No. 69.

You also cite to the Burbacher study in that paragraph for a statement regarding persistent inorganic mercury in the brain. Is it your opinion that persistent inorganic mercury in the brain causes neuroinflammation?

A It is my opinion that persistent inorganic mercury has the capacity to cause neuroinflammation in the brain.

Q Is it your opinion that persistent inorganic mercury in the brain causes neuroinflammation?

A It's my opinion that persistent inorganic mercury is consistent with causing neuroinflammation.

Q When you gave your deposition in the Blackwell case you were asked about the Vargas article. Do you recall that?

A Not specifically, but I'm sure that I was.

Q Okay. And at that time, in January of last year, you testified that you did not rely on the neuroinflammation work for your opinion, is that correct?

A At that --

Q And right now for the record we are showing you a page from your deposition, and the question that you were asked was, "Do you rely in any way in any of Dr. Zimmerman's work for your opinions in this case?"

Now, Dr. Zimmerman was a co-author on the Vargas article, is that right?

A That's correct.

Q Okay. And if you could just read what your answer to that question was.

A "I have read Dr. Zimmerman's work, so it becomes a body of knowledge that I have used to formulate my opinions. But I don't specifically recall any particular thing that he has as an individual contributed that I rely on for the opinion in the Blackwell case," which was a very different type of case.

I'm sorry. I should clarify for the Court. The last clause that I said was clarification and not reading directly from the record.

Q The Blackwell case did involve an allegation that thimerosal-containing vaccines, among other exposure, contributed or caused a child's autism, is that right?

A That's correct.

Q And in fact in the deposition you were asked if you disagreed with Dr. Zimmerman on any other points, and you testified that you disagreed with his conclusions, primarily his lack of concern with environmental issues and thimerosal toxicity, is that

correct?

A First of all, at the time that I did that deposition, I had relatively limited experience with Dr. Zimmerman's body of work with regard to neuroinflammation other than having read the Vargas paper.

Secondly, I had met him at the Autism Treatment Center and at the Autism Treatment Network meeting, and at that time, based on a comment that I had heard him say in the public forum, it did not seem that he shared some of the concerns that my colleagues and I did. I had very limited exposure to him so I don't know, in fact, if that was really the case or just my interpretation about what he said.

But as time has gone on I have re-read his work. I had not re-read his work just prior to this deposition since it was not part of the emphasis in that particular case. So as is my practice, I was not wanting to comment about details that I did not recall.

Q Since hearing Dr. Zimmerman make the comment that led you to believe that he didn't have concerns about thimerosal causing autism, have you spoken to Dr. Zimmerman since that time?

A Only a couple of weeks ago. One of my

patients who had been a normal baby up until his 18-month shots had had a cardiac arrest and a seizure on the day of the shots. He was air-lifted to the local university and the parents were told that the cardiac arrest and the seizures could not have had anything to do with his immunizations.

He subsequently went on to develop autism and a very recalcitrant seizure disorder for which the neurologists at the University of Virginia were not able to help him with. Ultimately he came to me for management, and he had seizures that were so bad that I had to put him on a vagus nerve stimulator, which did decrease the amount of his seizures from several hundred per day to a relatively smaller amount, somewhere in the range of 10 to 25.

About three weeks ago, he died, and he died in his sleep, which I presume was due to perhaps an unrecognized seizure that I had not been able to control.

So I asked the family if they would consider donating his brain for analysis, and because at that time I had read Dr. Zimmerman's work, I had just within the last few weeks read of his work in the Poling case. I had had the opportunity to look at his body of work and the folks at Hopkins in more detail.

I called him and asked him if he would work on Dillon's brain, and he said that what I should do is to have the brain donated to the Autism Tissue Brain Bank, and that he and his colleagues, as well as other scientists who had interest in this area, would be glad to work on it. That has actually been my only conversation with Dr. Zimmerman since I saw him at the Autism Treatment Network since, I think, 2004.

Q I take it the discussion did not involve Dr. Zimmerman's thoughts on whether thimerosal causes autism, is that accurate?

A The conversation I had about my dead patient?

Q Yes.

A That is correct.

Q So you have heard nothing from Dr. Zimmerman since the time that you heard him make the comment that led you to believe that he does not have any concerns about thimerosal causing autism, you're heard nothing from him since that time that would cause you to think that he's changed his mind on that issue, is that right?

A I haven't heard anything from him directly, no.

Q And you would agree that the Vargas article

you refer to does not mention thimerosal or mercury as a cause of a neuroinflammation, is that correct?

A I would agree.

Q You have talked about neuroinflammation in some of your talks at Defeat Autism Now conferences, is that right?

A That's correct.

Q And according to your CV, you gave a talk in Jacksonville, Florida, in late January 2007, and I want to show you a cover slide from a presentation and ask you if this is --

A Yes.

Q -- the presentation that you gave.

A Yes.

Q On the slide from that presentation titled "Vargas Research" --

A I'm sorry. Can you tell me the date of that lecture again?

Q I believe it was late January 2007. I can be more specific.

A Okay. That's close enough. I just needed to know the year.

Q It was about the same time that you gave your deposition in the Blackwell case.

A Yes.

Q And you note on the slide titled "Vargas Research" that the Vargas team "found evidence against an immune response mounted primarily against brain or agents within brain."

Does that mean they found evidence against that autoimmune process?

A I'd like with the permission of the Special Masters to expand a little bit on this slide.

The first point was no activation of adaptive immunity, which is the sort of classic B or T-cell infiltration or immunoglobulin deposition.

The second point is raising the possibility that the innate immune response may have been mounted primarily against brain or agents within the brain at that time. That was some evidence against at that point in time as science continues to march forward.

Q And I believe my question was, is it your understanding that Vargas found evidence against an autoimmune process and autism?

A No.

Q And then on a later slide in that same presentation you indicate that neuroinflammation may be secondary to GI inflammation. Is that right?

A That's correct.

Q So, at least when you gave this presentation

it was not your hypothesis that inorganic mercury in the brain was causing neuroinflammation, is that correct?

A The slides says there may be a link between primary GI inflammation and secondary CNS immune activation and tissue injury. I still believe that to be the case. There is growing evidence to suggest that toxicity through byproducts or intermediates of diet and gut bacteria also play a role in abnormal CNS function. I also believe that to be true.

Q But there you're referring to playing a role in abnormal CNS function and you don't say neuroinflammation, is that right?

A That is correct.

Q Have you changed your hypothesis since January of 2007?

A I have continued to expand my knowledge since January of 2007.

Q Have you adopted Dr. Kinsbourne's model as to his mechanism that he's proposed in this case?

A I agree with Dr. Kinsbourne's model as proposed in this case.

Q Did you adopt his model after reading his report that he prepared for this case?

A I fear that there is a tendency to

oversimplify here, that we either have to believe (a) or (b) or (c), and that they are all mutually exclusive. My work as a clinician and my work with these scientists relies on a model where neuroinflammation may be a final common pathway that can result from various mechanisms, depending on the situation of the child, the vulnerability of the child at the time of whatever the insult is.

I count myself among those who want to remain open to the idea of following the science wherever it goes, and as best we can determine, as my colleagues from all these different fields -- gastroenterology, toxicology, immunology -- they each contribute their body of information to inform this body of work.

So, just as I hope that all of us will have a better understanding of this in 2009 as we do now, I think we have a better understanding of it in 2008 than I did at the time of this lecture.

Q Prior to reading Dr. Kinsbourne's report, had you seen his specific model that proposes persistent inorganic mercury in the brain causing neuroinflammation leading to excess glutamate levels, had you seen that model anywhere before reading his report in this case?

A I had not seen that prior to Dr. Kinsbourne's report as articulated by him.

Q And you would agree that Dr. Kinsbourne's specific hypothesis as stated in his report has not been published, is that correct?

A I'm not aware that that's been published.

Q And it has obviously then not been peer reviewed, is that correct?

A I would assume that is correct.

Q And you would agree that it was generated for purposes of litigation?

A No, I would not agree.

Q And why would you disagree with that statement?

A Because I have no way of knowing what Dr. Kinsbourne's mental processes were when he generated that report. I would be surprised, based on my limited knowledge of him which only began after this trial, that he would be so motivated.

Q But you don't know one way or the other what motivated --

A That's correct.

Q -- Dr. Kinsbourne?

And I believe you said that it is still your opinion that gut inflammation can cause

neuroinflammation, is that right?

A I do think that in certain kids gut inflammation can play a contributing role. There is actually published science to support that.

Q And what science would that be?

A One of the papers is cited in my expert report by Vojdani and colleagues looking at dietary antigens. Another body of work is by Dr. DiNucci at New Jersey Medical School looking again at dietary antigens. Another body of work would be the work of Paul Ashwood and his immunology colleagues at the MIND Institute. Another body of work would be my clinical patients who have been endoscoped in my practice by my local gastroenterologist, and show evidence of inflammation. Another body of work would be the Walsh paper which looked at inflammation of the gut and secondary effects on the brain. And on the spur of the moment that is what I can give you.

Q Would Dr. Wakefield's work be included in that body of science?

A I am influenced by Dr. Wakefield's work. That is correct.

Q Is there a reason that you didn't mention his work?

A He has ironically just returned from a very

intensive investigation, and I am in this courtroom instead of being at the IMFAR meeting where some of his current science will be presented on this very day.

Q And since you bring up IMFAR, I noted that you've mentioned that you're here instead of being there, and I just wanted to say I hope you didn't miss it on our account because we had actually agreed that you could testify next week to allow you to go.

A And next week I'm in Chicago educating doctors, but I do appreciate that. Thank you.

Q Sure. Doctor, what clinical evidence of neuroinflammation can you point to in William Mead's case?

A By the very definition, neuroinflammation does not have good peripheral markers. This is the biggest problem for us as clinicians because, to the best of my knowledge, the body of work that looks at neuroinflammation actually makes a point of saying that peripheral markers are difficult to find, and certainly clinically available peripheral markers are, to my knowledge, nonexistent at this point. Let me give you an example.

In the Vargas paper, they looked at CSF, and all the tools that I would have available to me as a

clinician -- glucose levels, protein levels, cell counts, culture -- would not be informative. The things that they found that were abnormal are these very exquisite immunologic markers that are not typically available in community labs.

So, for example, they found that interferon gamma was vastly, vastly increased. I want to say it was like 200-fold increase, and so that was part of what they used to look at the issue of innate versus adaptive immunity, and so that in a clinical research setting was very helpful to them, but I have no way of getting that.

When we find high inflammatory markers, the ones we have clinically available frequently don't tell us where the inflammation is, and so I am left with wondering if it's neuroinflammation or gut inflammation or potentially other systems in the body that are affected.

So, we are forced to try to help these children without the lab evidence that would help us determine if they do have neuroinflammation, and John Green back in 2001 was even more hampered because the laboratory evidence available for him then was even more restrictive. That was seven years ago now. So it's entirely true that we don't have good markers

peripherally to determine this, and that's one of the big problems.

Q So the short answer to my question is that you can't point to any clinical evidence of neuroinflammation in William Mead's medical records, is that right?

A That is correct.

Q And the same would be true for Jordan King, is that correct?

A That is correct.

Q Doctor, your training is in general pediatrics, is that right?

A That's correct.

Q And you do not have subspecialty training in any particular area, is that right?

A That's correct.

Q You're not a neurologist?

A That's correct.

Q And do you have any formal training in neurological disorders?

A No.

Q And you're not a clinical child psychologist, is that right?

A That's correct.

Q Okay, and you're not a psychiatrist, is that

right?

A That's correct.

Q Your only board certification is in pediatrics, is that correct?

A That's correct.

Q What training do you have in diagnosing autism?

A The training that I have received as being a general pediatrician, and the things I learned when I was asked to write a book chapter for medical students on developmental and behavioral pediatrics, which included autism, and on immunology and allergies.

Q In your practice, what method do you use to diagnose a child with an ASD?

A I actually request that patients are independently diagnosed. My preference is that they go to a place like the TEACH Center in North Carolina, which is not too far from me, or one of the university centers, or to a neuropsychiatrist. My preference is that they have an ADOS or an ADIR to formally make the diagnosis, and that they undergo speech evaluations and motor evaluations, and psychological testing and intellectual testing.

Then in my practice I basically then just determine by using the records that I am given to see

if those diagnoses seem to be appropriate.

Q So you do not independently diagnose ASDs, you just confirm other professionals' diagnoses, is that correct?

A That's correct, and then I go on to try to take care of the kids medically.

Q What is your definition of regressive autism?

A A case in which there is a clearly documented time of normal development, followed by a clear loss of developmental milestones, and then the emergence of autistic symptoms.

Q Let's start with the period of normal development. How do you confirm normal development prior to regression?

A I look at records supplied by the primary care physician, and I take a very careful history from the parents, and I ask the parents to bring, when available, baby books or other contemporaneous documentation of their children's landmarks.

There are very clearly delineated month by month developmental markers in language, gross motor, fine motor, and social skills, and primary care physicians, whether they are pediatricians or family physicians, are taught to ask those questions at each

visit and do a record, so we are able to document normal development in that way.

Q Do you ever use videos?

A In my clinical practice?

Q Yes.

A I am given videos by parents, but it is very time-consuming for me to review the videos, so I typically do not use that if I have all these other measures that I've described.

Q Now I want to talk about the documentation of regression. Are there objective measures that you use to determine that a child actually regressed?

A One objective measure is whether or not they lost words. Another objective measure is whether or not they lost motor skills. Another objective measure is whether or not they developed stereotypic repetitive behaviors. Another objective measure would be whether or not they lost social reciprocity.

Q Is that all?

A I think so.

Q Okay. Let's start with losing words.

A Okay.

Q First of all, how many words must a child have had before the regression?

A I do not have a specific number. It

obviously depends on the age of the child and the developmental stage of the child.

Q Let's assume a 15-month-old child. How many words would you expect that child to have?

A Anywhere between eight and 15 typically, but if there is a child who developed language late, and only had three words, and then at some point in the future lost all of those words, that would be very concerning to me.

So again, I'm going to resist the idea that we can on a clinical basis make a definition of regressive autism by an arbitrary number.

Q How about for a 21-month-old child, how many words would you expect that child to have?

A That's very variable. I would expect -- the normal classic milestone is two-word phrases at 18 months of age. Some kids at 21 months have 50 to 100-word vocabularies. Some kids may have 25 to 50-word vocabularies.

Q Does it matter at all to you how the child is using the words?

A Yes.

Q In what way?

A If a child is only using a word to repeat or is doing it over and over and over, that is concerning

to me. A more normal pattern at 21 months would be a child who is using words to name different objects.

Q And how many words must a child lose for you to consider it a regression?

A I do not have a specific number of words. Again, it depends on what their developmental status was, and then that they lost words.

Q In terms of the other areas, the motor skills, the stereotypic behaviors and the social reciprocity, are you relying primarily on the parent reports in order to determine their progress, the child's progress in meeting those milestones and engaging in those behaviors prior to the regression?

A I'm sorry. Could you say that again? I missed part of your question.

Q Yes. In addition to the language, you have identified motor skills, stereotypic behaviors and social reciprocity as three other objective measures that you look at to determine whether regression had occurred. And my question was whether you were relying primarily on parent reports of the child's behavior prior to the regression to confirm that none of that existed prior to the alleged regression.

A I do place a huge amount of weight on the parents' report, but we also have these

contemporaneously-generated documents by the pediatrician, and typically when I get a referral and have the pediatric records sent, I see a pattern where the pediatrician is checking off, you know, coos, babbles, jargons, saying mama, dada, has eight words, putting two words together, and then at some point the records start saying things like no words, lost words, not talking the way he used to. That, to me, is reliable evidence in addition to the parents' history.

Q And all those examples you just gave dealt with language, and I was actually talking about the other measures, the motor skills, the stereotypic --

A Oh, I'm sorry.

Q -- behaviors and the social reciprocity.

A So my -- sorry.

Q Are those things normally noted in the medical records?

A That is correct.

Q Okay.

A So my same statements about language would also apply to motor, and in fact with motor there are two different streams of development. One would be gross motor. One would be fine motor. So when you look in the records, you will see things for gross motor like rolled over at four months, sat up at six

months, pulled up at nine months, began walking around a year. And for fine motor you will see things like hands midline to mouth at four months, transfers object at six months, pincer grasp at one year, pointing around one year. So that is typically recorded in the pediatrician's record, as well as remembered fairly well by parents when their child is young. It's more difficult, you know, years later.

So we did motor. Now the next one was social reciprocity?

Q Sure.

A Okay. So social reciprocity at two weeks the baby should smile at the mom and look at her -- I'm sorry -- should look at the mom in the eye. At six weeks the baby should start smiling. There should be interactive play that continues through the first year of life, and later on it includes things like picking up the hands when they see the mom walk into the room so they can be picked up, and involves around nine months of age to 12 months of age these reciprocal games like peek-a-boo, for example.

So again, these are things most parent at the time are able to tell you, and most doctors are noting those signs in their records. So, you are also, I think, able to document fairly clearly when

the doctor starts saying, you know, no longer looks at mom, or eye contact with parent diminished, or not using gesture to be lifted. Those are all signs of social reciprocity.

Then I think the last thing --

Q Let me interrupt you. Does wanting to be held, is that also a sign of social reciprocity?

A Wanting to be held is a tough one because babies differ in how much they like that. Most babies do like to be held, but if you have a child that likes being held and cuddled, and then quits liking that, I would include that as a loss of that behavior.

Q Okay.

A And then are we doing stereotypic or repetitive next?

Q That's right, let's do that one.

A So, typically, even though infants will do some things over and over when they are playing with a toy. They typically have a broad range of interests. So for example, a baby who is still lying on his back and you put one of those little swing set kind of toys where it dangles, the various objects, usually you're able to get the baby to play with one, and then another, and then go back to another one.

When you see the emergence of stereotypic

behaviors, the story I hear is, you know, I bought him all these toys and he used to play with everything, but now all he wants to do is line up his trains, or you know, he used to love playing with his BRIO set, but now all he wants to do is flip the light switch off and on over and over and over, or we can't get him to -- he used to eat with a fork and spoon. Now all he wants to do is eat with his hands over and over and over again. That's actually a little bit of a bad example because it also includes motor, but you get the point.

Q All right. I think that covers it.

Let me ask you this. In your practice do you use the term "clearly regressive autism"?

A I do.

Q How is that distinct from just regressive autism?

A Let me get some data for a second. I essentially am using those terms in my clinic interchangeably because when I say "regressive autism", I want there to be a situation in my mind where it was a clear regression as I've articulated, and clearly that's a clinical judgment.

I think I mentioned yesterday that in my population I actually see a lot of those patients

because of referral bias, but we also try to very clearly document the kids who have no regression, and that works out to be about 35 percent of my patients.

So, for me to call it regressive autism, I really want it to be clear, and one of the reasons for that is it helps me inform my judgments about how I'm going to treat that child because a differential diagnosis in my mind for a clear regression is quite different from the differential diagnosis for something that is either present from birth or there is no clear regression, so that the possibility at least has to be entertained that maybe there were signs that could have been missed by the parents because they were understandably in a state of denial that something bad could be happening to their baby.

Q So in any case or for any patient in which the symptoms either appeared early or gradually, you would not consider that a case of regressive autism, is that right?

A That's correct.

Q Doctor, you testified that the DAN model that you employ is collaborative, is that right?

A That's correct.

Q And the parents are involved in the process?

A That's correct.

Q And you testified, I believe, that you view your particular expertise as in taking histories, is that right?

A That's correct.

Q Prior to preparing your report in this case, did you interview William Mead's mother or father?

A No, I did not.

Q And did you interview Jordan King's mother or father?

A No, I did not.

Q So you did not take your own histories prior to preparing your reports in these cases?

A That's correct, and that's why I was so glad to have the opportunity to hear them here a couple days ago.

Q Did you personally evaluate Will Mead prior to preparing your report?

A No, I did not.

Q Did you perform a physical examination of him?

A No, I did not.

Q And did you personally evaluate Jordan King prior to preparing your report?

A No, I did not.

Q And I take it you did not perform a physical

examination of Jordan either, is that right?

A That's correct.

Q And I believe you said yesterday that you didn't review any of the videos that the family provided until last Thursday, is that right?

A That's right.

Q So would it be fair to say that your opinions as stated in your report were based on a review of the medical records?

A At the time I was asked to generate the report, that is absolutely correct.

Q Okay. In the Blackwell case, you only testified on the issue of general causation, is that right?

A You know, I don't remember, to tell you the truth.

Q You don't remember whether you offered an opinion as to whether the plaintiff in that case, whether his autism was specifically caused by mercury?

A You know, I'm sorry. I really don't remember much of that day.

Q Okay. We can refresh your recollection. We'll show you the portion of the transcripts from your deposition where you were asked whether you would be offering an opinion as to the plaintiffs, and if

you could just read what you answered to that question.

A I said, "Not for a specific case because I would never render such a specific opinion without the opportunity to evaluate the child, interrogate the parents, do a physical exam, and review the laboratory data."

Q Doctor, what makes these cases different than the Blackwell case?

A I guess part of the difference is that I know John Green well and I understand how he practices, and how careful he is, and he's actually been doing this work longer than I have. So when I see his physical exam and am able to read his notes, and see how he's thinking, I am better able to make a judgment.

And my understanding in this case is that I was being asked to provide an expert opinion as opposed to testifying as the treating physician. So I thought that in the vaccine court that was a distinction, and the role I was being asked to play here was not that of the treating physician, but as an expert in clinical pediatrics.

Q Do you believe that your opinions in these cases are subject to a lower standard than what you

would apply in your clinical practice?

A Is there a way you can rephrase that question?

Q The answer that you just provided was that the reason that you didn't feel it was necessary to do these things that you said in the Blackwell case, you would be required to do in order to offer the opinion that you've offered is that you weren't being offered here as a treating physician but rather as an expert.

And so I'm asking if you believe that your opinion as an expert is subject to a lower standard than what you would apply in your clinical practice.

MR. POWERS: Just a clarification. Is it the opinion in the reports or the opinion that she's testified to?

BY MR. JOHNSON:

Q I wasn't aware that there was a distinction, but let's start out with the report.

A It was my understanding that in vaccine court the idea was to make it a nonadversarial, family friendly funded compensation mechanism to which I have been contributing since the eighties in order to identify and compensate children where there was biologic plausibility that vaccines, some component might have contributed to their health problems.

In a case like the Blackwell case, I actually resisted for many years doing cases in the other court system because that clearly is an adversarial situation, and the stakes are very high. So I would say that I was under the idea that there was a different standard, not lower standard, I think carries a disparaging tone, but I thought that vaccine court and civil court were different situations.

Q Do you believe the decisions in vaccine cases should be based on reliable science?

A I do.

Q You're relying on notations in the medical records for your conclusion that William Mead and Jordan King experienced regression, is that correct?

A I'm sorry. Say that again.

Q You're relying on notations in the medical records for your conclusion that William Mead and Jordan King experienced regression, is that right?

A At the time of the report, yes. At the time of my testimony, I also have the advantage of having heard the parents and seen the videos.

Q Okay. So you're now also taking into consideration those other facts that were not available to you when you prepared your report?

A That's correct.

Q In your opinion, when did William Mead's regression occur?

A It appeared to emerge between 15 and 18 months of age.

Q And what are you relying on for that calculation of the timeframe when regression occurred?

A The medical records that noted his milestones and loss thereof.

Q When did, in your opinion, Jordan King's regression occurred?

A Emerging between 15 to 20 months.

Q And again if I could just ask you what you were relying on for that calculation.

A At the time of this, the medical records.

Q Is there a specific record in either case that you can point me to that you found particularly compelling or conclusive on that issue?

A We showed, I thought, yesterday the records from the pediatrician that showed before and after. You know, before the skill was there, after it wasn't. I also relied on Dr. Green's initial intake on both children which I don't have the exact page reference for, but I'm sure Scott could find it if we need to do that.

Q Okay. And we don't need to. I was just

wondering off the top of your head if you knew.

Doctor, does it matter for your opinions in these cases when the regression occurred?

A All the regressions that -- well, let me not say "all" in medicine because there is always the exception that proves the rules. The pattern that we see clinically is that the regressions are typically in the second year of life.

Having said that, I've also seen regressions in children, and this is atypical for the diagnosis of autism, but I have had children develop autistic type behaviors after the age of three, but for purposes of the classic picture I would say between -- somewhere in the second year of life or thereafter.

Q For the children who develop autistic behaviors after the age of three, would they be diagnosed as autistic?

A No, they would not be able to be diagnosed as autistic because the criteria as originally set out require under the age of three, not over.

Q In your opinion, how much thimerosal would a child need to be exposed to before it caused autistic regression?

A It depends on a huge number of factors, so I'm not able to give you a number.

Q Okay. Let's use the specific cases that we've got available to us as examples, and we will start with William Mead, and you state in your report that he received 187.5 micrograms of ethyl mercury by the time he was seven months old.

A Right.

Q And that is based on three Hepatitis B vaccines, three DTaP vaccines, and two Hib vaccines, is that correct?

A Yes.

Q Or I'm sorry, three Hib vaccines.

Do you know whether the Hib vaccines that William Mead received were from single-dose vials or multi-dose vials?

A Oh, actually that's a good point. I do not know.

Q Okay, because --

A So it's possible that the numbers I calculated are different. I mean, would have been different had I had that information.

Q Because am I correct that single-dose vials do not contain thimerosal, is that right?

A That is correct. So if there is evidence with the Court's permission that -- it's unusual for them to be single-dose vials, but if they were, I

would like the opportunity to revise the estimates downward based on that new information.

Q And it would be revised downward by 75 micrograms, is that correct?

A Yes.

Q And did you hear Dr. Deth testify? I believe you were here for his testimony the other day, is that right?

A I was here for his testimony.

Q And did you hear him testify that he believes there is a threshold concentration of inorganic mercury in the brain necessary to cause autism?

A Let me pull my notes on his testimony. Actually, at this point in time we don't have a way, I presume, of pulling the transcript up because it hasn't been done yet, is that correct? Okay.

Okay, what I see are notes that for neurologic symptoms, how much in brain concentration dependant upon effects, sub-nanomoler. I don't know if that was what you're referring to. Maybe you could give me the sentence that he said.

Q Let's go at it this way. You've stated that William Mead received 187.5 micrograms of thimerosal by the time he was seven months old, is that right?

A That is correct.

Q And that you've testified that his regression occurred between 15 and 18 months, is that right?

A That's correct.

Q And he didn't receive any other thimerosal-containing vaccines until he was 23 months, I believe, is that right?

A I think that is correct.

Q Okay. So at the time that the regression occurred according to you, he had received 187.5 micrograms of thimerosal.

A Or less.

Q Or less.

A Yes.

Q But let's assume that he received 187.5. In your opinion, is that amount sufficient to cause autistic regression?

A It depends on the amount of inorganic mercury that resulted in his brain or affected as yet undetermined systems. But to the best of my knowledge, that amount would be sufficient because we have no known or proven safe level of mercury, which is a known neurotoxin and has other bad effects. So yes, that would be a sufficient amount.

Q You say there is no safe level. Could one thimerosal-containing vaccine expose the child to enough ethyl mercury to cause regressive autism?

A I actually think that's theoretically possible depending on the situation of the child at the time. I'm concerned that if the child, for example, had birth trauma or was ill at the time or had oxidative stress or if you had a bunch of genetic predispositions as Jill James has outlined with regard to, you know, reduced folic carrier and transcavalimen to enzymes or COMT enzymes or MTHFR, or if there were other factors that we still don't even know about, that the vulnerability of the child at the time to me can play a huge role, and that's why I'm so reluctant to pick some number. So, I think it is theoretically possible that one TCV could harm a given child.

Q Am I correct that in the Blackwell case you actually testified that even trace amounts of thimerosal in vaccines could lead to autism, is that right?

A I may have said that. I don't recall specifically.

Q Do you believe that as you are sitting here today?

A I am very concerned about trace amounts even

as I sit here today.

Q Are you aware of any studies that show that trace amounts of thimerosal in vaccines cause any neurological disorders?

A Examinations in children or animals --

Q Any studies at all.

A -- or anywhere?

Q In trace amounts.

A No.

Q And do you know of any medical organization that would agree with the idea that trace amounts of thimerosal could lead to autism?

A No.

Q Doctor, is your opinion in this case that thimerosal-containing vaccines contribute to autism, is that limited to regressive autism?

A I actually -- I actually don't know the answer to that yet. I am still -- the pathology and my clinical experience tends to make me think that that's the case, but I'm also concerned about prenatal exposures to thimerosal, and so I'm concerned that for the kids whose mother got Baro, for example, with high does of thimerosal, or who got flu vaccines in pregnancy, that we may start seeing a shift in the pattern where because of that exposure during fetal

development we also could be affecting children that don't have a classic period of normal development.

But I think obviously the science is in its infancy here, so I can't be sure about whether it's just regressive or potentially also emerging symptoms within the first year, or even at birth.

Q As you know, one of the factors that the Special Masters are required to consider is whether the onset of symptoms occurs in a medically appropriate timeframe following vaccination.

A Yes.

Q In your opinion, what is a medically appropriate timeframe for the onset of regressive autism after a child receives a thimerosal-containing vaccine?

A Yes, this is a difficult issue because as the neuropathology as best we know at this point in time shows the neuroinflammation increases over time, and in a lot of the papers in animal models, which are obviously limited, there can be neuroinflammation documented and the animals aren't even particularly symptomatic at that time.

So, it's a little bit different in my mind from the classic vaccine injuries you all have had to make decisions on in the past because then we were

looking at things like an acute pertussis reaction where the onset was within seven days or so. Here our concern is that the seed is planted, and that it's a period of months or years, you know, as yet to be undetermined.

If the neuropathology shows that the inorganic mercury is going to last for decades, it's very hard to put a very tight timeframe on this. One of my concerns was when we originally evaluated vaccines for safety we tended to do very short follow ups looking for acute reactions, and unless there is something that I'm not aware of I'm not aware of studies that look beyond 21 days for acute reactions.

What we're concerned about is chronic reactions that are emerging over time, and emerging in different timelines, depending on the individual child. So, I really have a hard time putting an upper cap on the timeframe.

Q You just stated that it's your understanding that the neuroinflammation increases over time. Did I understand you correctly?

A Yes.

Q What's causing the neuroinflammation to increase over time?

A I have no idea, or let me clarify that

response. That is for a neuropathologist to determine, so I would defer to what is learned about this subject by them as time marches on.

Q By neuropathologists?

A Right, or others who have expertise in that field that goes beyond mine.

Q Doctor, in your report you discuss the concept of genetic susceptibility.

A I did.

Q Do you believe there is a genetic susceptibility to mercury?

A I believe there are many constellations of genetic susceptibilities that would render a particular individual more susceptible to mercury.

Q Does every child who has an ASD cause or contributed to by thimerosal have a genetic susceptibility to the effects of mercury?

A In the sense -- well, I actually don't know the answer to that question because, first of all, we have not obviously checked every child, and that's reaching beyond the science. But the state of the science at this point suggests some very intriguing possibilities about genetic abnormalities in biochemical pathways and enzyme function that would be expected to render children at greater risk.

So what they are, I think, remains to somewhat be determined. I try never to say, you know, always or never in medicine, so I would not want to say every child.

Q What markers do you look for to determine if a child has a genetic susceptibility to mercury?

A There are indirect markers and it depends a lot on the finances of the parents. One of the most helpful ways to try to evaluate that very difficult clinical problem is to look at methylation genomics. That involves single nucleotide polymorphisms that affect the biochemistry involving methylation and transsulfuration and glutathione production.

So, for the parents who are financially able to afford the tests or who are interested in doing those tests, we can look at things like methylenetetrahydrofolate reductase enzymes or methionine synthase-related enzymes, or the reduced folate carrier enzymes, or the transcobalamin II enzymes, or catecholamine O methyl transferase enzymes, or glutathione S transferase enzymes. Those are all in that pathway that impact on the body's ability to make glutathione and mobilize mercury and other heavy metals.

So, as Dr. James' work has shown, various

combinations of those genetic predispositions will lead to varying elevations in the odds ratio or the risk factor for that particular child. So, you know, sometimes it doubles the risk, sometimes it triples the risk, sometimes it's even more, but this field is really in its infancy, and I will much better be able to answer that question after Dr. James does her upcoming NIH study where she is able to look at huge numbers of kids.

The things I've just mentioned are on, you know, the population we have studied so far. That may well change as we go into larger populations.

Q Now, you just mentioned a single nucleotide polymorphisms, is that right?

A That's correct.

Q And in shorthand, those are referred to as SNPs, is that right?

A That's correct.

Q So, I'm just going to call them SNPs because it's easier.

A I'd love to do that too. Thank you.

Q Okay. You have not personally done any research on SNPs, is that right?

A That is absolutely correct.

Q You're not a geneticist, is that correct?

A Absolutely correct.

Q So, you rely on the work of others for your opinions regarding which SNPs may or may not be associated with genetic susceptibility, is that right?

A I do.

Q You would agree that SNPs occur in a significant percentage of the population, is that right?

A I do.

Q They are not rare, is that right?

A We all have them, and that's what makes us -- you know, I might be more susceptible to have cancer, you might be more susceptible to have hypertension. We all have them. That's exactly right.

Q Okay. And specific SNPs vary across ethnic groups, is that right?

A That's also true.

Q And autism has an equal prevalence among ethnic groups or across ethnic groups, is that right?

A Is that true?

Q I'm asking you.

A I think there have actually been differences across ethnic groups, and I can't recall the numbers, but I was not aware that it was equal across ethnic

groups. I guess that's something we could put on our list to try to find out.

Q And not every person with a SNP is autistic, is that correct?

A Obviously correct.

Q You mentioned some studies that have been done by Jill James. Are you aware that Jill James recently published a study in which she looked at polymorphisms on two different genes, the CPOX and the ALAD gene?

A Oh, you know, I actually have not read that one. When did that come out?

Q It just came out recently. It's Respondent's Master List No. 430, and we can provide you with a copy.

A Okay.

Q Are you aware that Jill James and the other authors on the study looked at those genes because polymorphisms and the CPOX gene had been associated with elevated blood mercury levels, and polymorphisms in the ALAD gene had been associated with elevated blood lead levels, is that right?

A You know, I'm really very hesitant to comment about a complex paper that I have not read. Ordinarily, I would spend over an hour reading a paper

like this, so I don't know what the situation is when I'm being questioned about a paper I haven't read, but I would really rather not make opinions about it.

Q So you don't know --

A I don't know.

Q -- as you sit here that her hypothesis was that a higher percent of autistic children would carry both of those polymorphisms?

A I have not discussed this paper with her. She is a great colleague of mine, but I have not talked to her about this one so I don't know.

Q Okay. And you're obviously not aware that her study found that the frequency of the ALAD polymorphism was higher among autistic subjects but the frequency of the CPOX polymorphism was actually lower in autistic subjects than in controls.

A I did not know that but that does not surprise me at all because one of the things that's been so great about Jill is that she has always, as she has talked to us, talked about the limitations of small numbers, and the fact that the SNPs are found in a lot of patients, and that only until she can get the kind of NIH funding to do huge numbers will we be able to make definitive comments about the genomics of this.

Q And let me just direct your attention to the highlighted portions, and the second one in particular. Would you agree that at least in this study the authors concluded that lead, and not mercury, may be associated with autism?

A Oh, yeah. I would definitely acknowledge that because part of my concern in these kids that I tried to articulate in my report, and maybe did not do a good job of, is our concern for synergistic toxicities. It's clearly known that the lethal dose of two toxins put together tends to be higher than the individual lethal doses added linearly.

So, if you will recall in, I believe, both of the patients that are under review in this hearing they also were showing excretions of lead as well as mercury. So the co-existence of other toxicities is another risk factor in my mind, and so I maybe didn't clarify it enough, but when we're working up children for autism we also look at their lead history. We try to evaluate them for lead toxicity, and the porphyrins that we've talked about look for lead as well as mercury. The provoked urines that we do help the child excrete both lead and mercury, and the agent that we usually start out with is the one that's FDA approved for lead toxicity.

So, I have in no way in this hearing tried to negate or diminish the importance of other toxicities to neurologic impact on these children.

Q Doctor, you would agree that at this time there are no established biomarkers for genetic susceptibility to mercury, is that right?

A I would agree to that, yes. It's research that we need to do and we desperately need to develop biomarkers.

Q I want to talk now about the mercury testing in both of these cases, and I apologize because we're going to go through some of the same tests that you referred to yesterday, but I think it's going to be helpful to go through those again if you don't mind.

A Okay. For my preparation, will you have them on the screen?

Q Yes.

A Okay.

Q Yes, we will.

First of all, let me just ask you what testing methods do you believe reliably show mercury levels in a person, in a human?

A I don't think that we have a great test to reliably show the levels, and that's, I think, why we do a combination of the porphyrin testing, which will

show us impact of heavy metals on that crucial pathway, as well as trying to find the mercury or the lead or whatever it is.

The problem is is that the easiest way to find it, which would be a blood test, only works for acute toxicities. So when you're looking at either lead that is in the bones or mercury that is in the brain, the kidney, the fat and the liver, you have to use indirect measures, and the one that is useful is to do the provoked specimens and see if you can mobilize mercury to be excreted, implying that there is a body burden. But to my knowledge, it's very difficult to extrapolate from those tests to quantify a body burden.

Q In the answer that you just gave you referred to "we use these tests," and I was wondering when you use the term "we", who are you referring to?

A Primarily me and my colleagues that are affiliated with the Autism Research Institute.

Q So that doesn't include the general medical community, is that correct?

A No. I'm fairly certain that except in areas where naturopaths and holistic practitioners are better accepted than they are in my conservative community, it's not something typically in the general

medical community. That would be fair.

Q Haven't you stated in the past that blood, hair and unprovoked urine testing are not reliable tests for infantile exposure to mercury?

A Tell me that again.

Q Okay.

A Blood, yes.

Q Blood, hair and unprovoked urine are not reliable tests for infantile exposure to mercury.

A That is my belief.

Q Okay. And that is also stated in the DAN consensus statement, is that right?

A It may well be.

Q Okay.

A Again, it's been a long time since I worked on that. I think that was five or six or seven years ago.

Q Okay. So you helped write the consensus statement, is that correct?

A I reviewed it and signed off on it.

Q Okay. And the consensus statement indeed says that blood, hair and unprovoked urine tests are not good methods for measuring mercury/metal toxicity in autism, is that right?

A Yes.

Q And you testified just a moment ago that blood testing is only reliable for acute exposures. Is that right?

A That's correct.

Q So blood testing performed several weeks after an exposure would no longer be a reliable measure of the exposure, is that correct?

A That is my belief based on the studies of Pichichero and others who have looked at the fact that most seems to go away within about seven days, if I recall the number correctly.

Q What is your opinion on fecal testing?

A I have mixed feelings about fecal testing. One value is that theoretically you mobilize a lot of mercury and excrete it in the feces, and so some doctors will use that, and certainly back in the late nineties and early two thousands that was one of the only things that we had.

The more traditional way to look is through the urinary testing. So again, I think it's a matter of putting the various pieces of laboratory data, realizing the inherent limitations in most of them and putting it together with a clinical picture.

Q So there are limitations to fecal testing, is that correct?

A Yes, I'm sorry. That would have been a short way to say it.

Q Let's start and we'll switch up the order since it always seems William Mead gets to go first, we'll start with Jordan King's mercury testing.

A Okay. Just a second. Let me get his chart open. Okay.

Q All right. And let's start with the Jordan King Exhibit 1 at 46, and this is a hair test from March 29, 2000, is that right?

A That is right, but I don't recall that I used the hair test in my presentation because that is something that I don't feel that I can rely on.

Q Okay. And that's correct, you did not refer to this yesterday, and as you said, is that because you do not think that the hair tests are reliable?

A That is correct.

Q Okay. Did you hear Dr. Aposhian testify?

A No, I'm sorry, I did not.

Q Okay. Have you reviewed his report in this case?

A Gosh. Yeah, a long time ago back, probably in the late fall.

Q You're aware that Dr. Aposhian believes that there is a mercury efflux disorder that may be causing

certain children to have problems excreting mercury, is that correct?

A That is correct.

Q Have you personally formed an opinion about whether there is such a thing as a mercury efflux disorder?

A I do think that there are kids that have difficulty excreting and mobilizing mercury. The hair tests that I think Vas was referring to there are baby hair tests at time of first haircut, and that is a totally different situation from the situation that I had here where the child was already two.

I do think there is value in looking at the infant hair test because they are often obtained at a time when we have a documented ongoing exposure to mercury through thimerosal-containing vaccines, and some of the initial studies that have been done initially by Dr. Holmes and Dr. Haley, and then replicated at MIT were showing that there were some infants that didn't seem to be able to mobilize mercury into their hair, and yet they had this known exposure, and they were able to mobilize other known exposures like lead or antimony or arsenic or whatever.

So, in that subpopulation of kids when

you're looking at that specific timing, I think that hair analysis can be helpful, but not because mercury is high. In that case it's helpful because the mercury is low, and that, to the extent that hair is an excretory organ, and you know, it's not a primary excretory organ, so again that work has some limitations on it also.

But to the extent to which it functions to excrete mercury, having a low hair mercury in an infant at the time of a known exposure would be informative. These values, to me, later I don't feel like I can really rely on to make a judgment.

Q So any of the hair tests in either one of these cases you feel adds no support to your opinions in these cases?

A I think that would have to be true, yes.

Q Following this test, and we're now going to look at Jordan King Exhibit 12 at page 1, this is a blood test from April 24, 2000, and it revealed that his mercury levels were within the reference range, is that correct?

A Since we've already established my ability with math and birth dates, can someone tell me how old the child was here? Birth date on Jordan was in '98, September/October? Okay, September '97 to August

2000, so two years and -- two and six-twelfth years, okay. Now I'm ready.

Q Okay. You did not discuss this test result in your testimony yesterday, is that right?

A Yes, and the reason I did not is that the mercury levels in blood are so transient.

Q The next test that we were able to locate in the records is from May 1, 2000, and this is Jordan King Exhibit 1 at 45, and this is a post-provocation challenge, and you did discuss this result yesterday, and you noted that his results were high, is that right?

A That's correct. And tell me the date on this, did you say May 2000?

Q May 1, 2000.

A Okay.

Q Did Jordan have a pre-provocation baseline test done the previous day?

A I don't recall.

Q And this was a test that was ordered by Dr. Green, is that right?

A That's correct.

Q And Dr. Green is a member of the Defeat Autism Now, he's a DAN doctor?

A Yeah, we actually don't use the term "DAN

doctor" anymore for reasons that I articulated yesterday, but he is a member of our consortium, our group.

Q Okay. Doesn't the consensus statement say that for a post-provocation test a baseline should be done?

A And I suspect that what happened here is that a clinical decision was made in the context of clinical management not to continue to have the family have such high expenses. One of the great difficulties we have in trying to utilize these labs is that they frequently are out-of-pocket expenses for the family, and there are many, many times when we would prefer to have more laboratory information, but if the parent is buying supplements to restore the nutrition of their child, and buying medications, and having frequent doctor visits, in many states autism is carved out as a psychiatric diagnosis.

So that if you go to a doctor and you're autistic, you have much difficulty getting reimbursement for seeing the doctor, even if you went in because of, you know, diarrhea or, you know, whatever the case. If it's not coded very carefully, it's a huge financial burden on the family.

So knowing John the way that I do, I would

guess that he was trying to spare the family more expense.

Q And my question was actually whether the DAN! consensus statement --

A I'm sorry.

Q -- recommends that a baseline be done before a post-provocation test.

A We do recommend that.

Q And doesn't the fact that a baseline test was not done make the post-provocation results less reliable?

A It is suggested that a baseline urine sample be collected, followed by a provoked sample. This allows one to directly compare. Comparing with an unprovoked urine also helps if the person has abnormal creatine levels, and creatine is often found to be marginal, and low creatine can skew the urine analyte results to high levels.

So, I guess my question would be were there other times that Dr. Green had done a pre- and post-provocation such that he already had a pattern and was using this as more of a marker for his follow up to the therapies he was providing in order to see if he was getting effective excretion. That is how I would interpret that.

Q You've reviewed Dr. Green's records, is that correct?

A I have.

Q Did you see any such test in his records?

A My memory is that he did do a pre-provocation and a post-provocation specimen. I think we presented that yesterday as an example of how this process works, but I'll rely on Scott to help sort that out.

Q But at this point in time for this particular test had he up to this time performed a baseline sample test?

A I am not able to answer that without referring to the records. Perhaps we can find that during the break and I can address it later?

Q Sure. Okay, let's look at the next test and this is Jordan King Exhibit 1 at page 36, and this was a provoked fecal test done on May 2, 2000, and I believe this is one that you did discuss yesterday, is that right?

A Yes.

Q Are there other values that are elevated on this test besides mercury?

A Yes.

Q And what are they?

A Arsenic and lead and thallium and tungsten.

Q Are those values at all significant to your opinions?

A When we see other elements -- well, first of all, I don't usually use fecal testing in my personal practice, so I am talking a little bit out of scope of my clinical experience, and so I would like to do that with that caveat.

I'm sorry. I think I may have said lead was elevated, and it looks like it's actually nickel.

We look for potential environmental sources when we see things that are in the elevated range, and I rely, since I'm not a toxicologist, with the report that comes with the tests, to go through an environmental history with the family and say, do you have your child exposed to these potential sources.

We have not as yet identified, to my knowledge, thallium or tungsten or nickel as areas of potential concern, and I think the point here is that our feces are one of the ways that we get rid of environmental exposures, and so it's not unusual to me that we would find other elements in the stool.

Q Is it normal for children to be excreting elevated levels of arsenic, nickel, thallium and tungsten?

A Is it normal? I imagine that children weren't doing that as much before they were exposed to grocery store chicken that had arsenic in it, or some of the other environmental changes that have occurred with industrialization. It probably is very common in normal children. I would say it is not an ideal state that we were necessarily meant to live in.

Q Did you see anything in Jordan King's medical records that suggested to you any exposures that might explain elevated arsenic, nickel, thallium or tungsten?

A I can't recall now. In looking through John's records, I think that he personally would have probably asked about arsenic exposures, but I don't recall if that was recorded.

Q Okay, let's move to the next test which is Jordan King Exhibit 12 at 37, and this was a random urine test taken on May 23, 2000, is that right?

A I can't see the date but I'll be glad to take your word for it.

Q We'll try to blow it up for you.

A Yes. Yeah, no, it's fine. I take your word for it.

Q Okay. And this was not a test that you discussed yesterday, is that correct?

A That's correct.

Q Is there a reason that you didn't discuss this test?

A I was told that we were trying to present a tight case and that we would have limited number of slides, so I didn't choose to include it because it says no reference range was established. It didn't seem that it would be informative.

Q But the test results were essentially normal, is that correct?

A Yes, it is listed in the normal column.

Q And the next test that we can look at is Jordan King Exhibit 1, page 43, and this is a provoked fecal test done on June 19, 2000, is that right?

A Yes.

Q Okay. And you did not discuss this test result yesterday, is that right?

A I actually don't recall. We initially had a larger number of these tests and we ended up not showing all of them because, as you sat through it, some of my testimony was repetitive, so I honestly don't recall.

Q So this may have been one that you decided just wasn't important enough to include in the scaled down presentation, is that right?

A Perhaps. Yes.

Q Okay, let's look at the next test that we found in the records, and this is at Jordan King Exhibit 1, page 42, and this is another provoked fecal test. It's done on September 11, 2000, is that right?

A Yes.

Q Okay. And where the previous test was elevated, this test is no longer elevated, is that right, for mercury?

A That's correct.

Q Okay. And this was again a test that you didn't discuss in your presentation yesterday?

A And so I'm happy to do it now. There are a number of reasons that that could be the case. One possibility would be that at that particular point in time whatever Dr. Green had done was effective in decreasing the child's mercury burden.

Another possibility is that this was provoked, correct, Mr. Johnson?

Q Yes, it was.

A Another possibility was that the agent he chose at that particular time wasn't being effective. So, our pattern tends to be one where at times there is excretion, other times there is not. I wish I could say that it correlated well with our rational

approach to the children, but in my experience that's actually not even necessarily the case because sometimes some combination of things that we are doing seems to work better and we can't identify why.

One of the difficulties of us having scientific studies in our practice is is that we're trying to work on getting the kids better, so we're not as good at isolating the various factors.

Q All right, let's look at the next test. This one, I believe, is one that you discussed yesterday.

A Correct.

Q And this is a provoked urine test on December 19, 2000, and the cite is Jordan King Exhibit 1 at page 35?

A That's correct.

Q Okay. And this was a post-provocation test, is that right?

A Yes.

Q And again was any baseline performed prior to this test?

A I'm not certain, but since you're asking the question I'm thinking maybe it was not.

Q And on this test Jordan's tin level was elevated, is that right?

A That's correct.

Q Does the elevated tin level concern you at all?

A When we see elevated tin, we look for environmental exposures like whether they are getting it from juice boxes or toothpastes. I don't know much about tin toxicology, and I'm not aware that tin in itself has been demonstrated to be a huge problem in children, but just from a standpoint of trying to optimize the environment, that is usually the only intervention I do in my practice. Perhaps that will ultimately be proven correct, or I could be wrong.

Q Do you know how much toothpaste it would take to reach tin levels at this level?

A I really don't. I'm sorry.

Q And any idea how many juice boxes a child would need to consume to get this much tin?

A Absolutely no idea.

Q Okay. Let's look at the next test, which is at Jordan King Exhibit 7, page 36, and this is a hair test, correct?

A It is.

Q Okay. And as you testified earlier, you don't find hair tests particularly reliable, is that right?

A At the older ages, that is correct.
Q All right, the next test that we found was at Jordan King Exhibit 9, page 35, and this was from a different lab. The prior lab was Doctor's Data, I believe, and this is Great Smokies.
A Yes.
Q And this is another hair test, is that right?
A It appears to be so, yes.
Q Okay. So again the reason that you didn't discuss it yesterday was because you don't find the hair test to be reliable?
A That's correct.
Q Okay. Let's look at the next test, which is at Jordan King Exhibit 1, page 55.
A Okay.
Q And I believe this is one that you discussed yesterday.
A I did.
Q Okay. And this is a provoked urine test from Doctor's Data, is that right?
A Yes.
Q And the date on this is February 12, 2003, correct?
A Yes.

Q I believe in relation to this test you noted that seven times -- that the mercury was at seven times the top of the reference range. Is that right?

A That's right, and I hope I did the math right, yes.

Q Okay. And again no baseline was conducted before this test, is that correct?

A That's correct.

Q And here the amount of tin excreted was 20 times the top of the reference range, is that right?

A Yeah.

Q Okay. But do you have any concerns about that tin level?

A Well, as I just testified, I'm not aware of biological mechanisms about tin that concern me at this point in time. They may be there and I could be overlooking an area where I should be intervening with the kids, but I have not -- other than trying to control the environment, I have not been addressing tin in our patients.

Q Okay. The next test is at Jordan King, Exhibit 17, page 9. This is one from July 28, 2003, is that right? We just lost it. There we go.

A Yes, it appears to be so.

Q Okay. And this was actually a pre-

provocation urine test, is that right?

A On the left pre, and on the right post.

Q Okay. So we've got the post-provocation urine test on the right, is that correct, and that was from the next day?

A That's correct.

Q Okay. The pre-provocation test, mercury was in the reference range, is that right?

A That's correct.

Q And in the post-provocation test, the mercury levels were actually below the detection limit, aren't they?

A That's correct.

Q So they were lower after provocation?

A Yes.

THE WITNESS: And if I could ask the Special Masters, could I go off the record for a moment to discuss this?

SPECIAL MASTER HASTINGS: I'm not sure why we would want to discuss something off the record. Let me first, before we get to that, you've got two exhibits on two pages of the exhibit on the screen.

MR. JOHNSON: That's correct.

SPECIAL MASTER HASTINGS: Is that page 9, the pre-test, and page 16, the post-test?

MR. JOHNSON: Yes.

SPECIAL MASTER HASTINGS: Can you tell us, Doctor, what you're thinking, why you want to go off the record? I guess I don't understand.

THE WITNESS: Because I have some concerns about the reliability of this test, and since I am concerned about on the web and in an official document being perceived as saying something potentially uncomplimentary about a business, I am concerned about the legal consequences of me doing so for my organization.

SPECIAL MASTER HASTINGS: Well, I appreciate that concern. On the other hand, we have got evidence in an important court case here, and the government may be relying upon this evidence --

THE WITNESS: Okay.

SPECIAL MASTER HASTINGS: -- to disprove your theory.

THE WITNESS: Okay.

SPECIAL MASTER HASTINGS: I think we can't rely on any evidence if we were to go off the record and you were to talk to us.

THE WITNESS: Okay.

SPECIAL MASTER HASTINGS: I couldn't rely on that anyway. It wouldn't do you any good. So if you

think that this test thing is unreliable --

THE WITNESS: I didn't say that.

SPECIAL MASTER HASTINGS: -- I think you should tell us on the record.

THE WITNESS: Okay. In that case, it is my best medical judgment that this particular laboratory testing is unreliable.

BY MR. JOHNSON:

Q Does that include all testing from this laboratory?

A No. I would not say all testing from this laboratory.

Q Which tests from this laboratory would you find reliable?

A I'm not sure at this point in time. We are currently taking a split sample reliability approach to replication of these results. So until I have that data, I really cannot answer the question.

Q So Great Smokies is one of the labs that you have included in the research project that is ongoing in your practice? Is that what I'm hearing you to say?

A That's correct.

Q And at this time, based on the lack of results that you've got at this point, you wouldn't be

comfortable relying on any testing from Great Smokies, is that right?

A Not in a matter of this much importance, that's correct.

Q Thank you. So that was all of the test results that we were able to find for Jordan King, and it appears that out of all of the tests you cited four that were supportive of your opinion, is that correct?

A Correct.

Q Okay, let's talk about William Mead's mercury testing.

THE WITNESS: Could I ask the Special Masters if this might be an appropriate time to take a break since we're moving from one child to another?

SPECIAL MASTER CAMPBELL-SMITH: It is about 20 of roughly, 18 minutes before 11. It's a little earlier but because you're the one who is going to be doing the chatting, we are happy to go ahead now if that makes sense, and I guess we will give ourselves until 11:00.

THE WITNESS: Thank you so much.

SPECIAL MASTER CAMPBELL-SMITH: Okay, we are in recess.

(Whereupon, a short recess was taken.)

SPECIAL MASTER CAMPBELL-SMITH: We are back

on the record. Please be seated.

There have been some comments and observations about the warmth of the courtroom. I personally am enjoying it, but I understand --

(Laughter.)

SPECIAL MASTER CAMPBELL-SMITH: -- Special Master Hastings has demonstrated that we will invite counsel to remove your jackets if you are in danger of heat exhaustion, with that invitation and those others who are here.

I also understand that Mr. Mead is leaving this morning at 11:30, and I want to take the opportunity, as has been said a number of times during this proceeding, to thank you for your participation, for sharing your experience with us. I understand that Ms. Shirley will be here with us through the day, but we do want to thank you again for permitting your son's case to go forward in such a public way.

MR. MEAD: Thank you.

SPECIAL MASTER CAMPBELL-SMITH: With those preliminary matters, we will resume where we were which I believe is the cross, and with particular focus on William Mead.

MR. JOHNSON: That's correct, Special Master.

SPECIAL MASTER CAMPBELL-SMITH: Thank you.

BY MR. JOHNSON:

Q Dr. Mumper, we'll start with the first test that we were able to locate in the medical records for William Mead, and this is William Mead Exhibit 5 at page 5, and this is a blood test, correct?

A That is correct.

Q Okay. And this test is out of the reference range for mercury, is that right?

A That is correct.

Q Okay. And I believe this was the test that you referenced yesterday during your testimony, is that right?

A That is correct.

Q Now, this test was taken on January 8, 2001, is that right?

A Right.

Q And William's last vaccination containing thimerosal was on April 12, 2000, which was approximately eight months earlier, is that right?

A Right.

Q Since you've testified that mercury or that ethyl mercury from thimerosal only stays in the blood for a matter of weeks, is it your position that this test reflected a more recent mercury exposure?

A That would be my concern, and that was one of the reasons that I included in my reports the concern about ongoing environmental toxicities. It is known that in the Pacific Northwest there are concerns about environmental mercury exposures, so it leaves the door open for environmental exposures.

Q And what specific environmental exposures are you referring to? From what source?

A Coal-burning power plants would be one potential source; being located next to a mercury mine would be another potential source.

Q Okay. And those would not be ethyl mercury, is that right?

A Right.

Q Okay, so --

A I think that -- at least I intended to testify that when these tests say "mercury" they are not species specific.

Q Okay. And I guess I want to make clear that your opinion in this case is that is it the ethyl mercury that's causing the problem or is it the inorganic mercury that is causing the problem?

A My concern is the inorganic mercury that is in the brain and in other tissues of the body.

Q Okay. So this test being eight months after

the most recent thimerosal-containing vaccine in your opinion would not be a reliable measure of mercury from the vaccination. Is that a fair statement?

A Yeah. To the best of my knowledge, I think that is probably true.

Q Let's look at the next test, which is William Mead Exhibit 5, page 3, and this is a post-provocation urine test from January 22, 2001, is that right?

A Yes.

Q And this indicates that mercury was out of the reference range, correct?

A That's correct.

Q Okay. Was there a baseline test performed prior to this post-provocation testing?

A I don't recall.

Q In the medical records, it indicates that William's parents referred to this test and said that William's test demonstrated extremely high amount of mercury in his system for which he will require chelation.

My question is, would you in your practice rely on a single post-provocation urine test where no baseline was performed to determine that a child required chelation?

A I am not as experienced in chelation as John Green is, and I actually tend to do many more things first than more experienced environmental doctors. So, I probably would not have even gone to chelation quite that early in the course of William's care. But I would yield to Dr. Green's greater level of experience.

Q Does the fact that William excreted a large amount of mercury after his first post-provocation test indicate that he did not have any problems excreting mercury?

A Did the fact that he excreted it with provocation indicate that he does not have problems excreting it, is that the correct --

Q On the first provocation, the very first time that he had a post-provocation test done he excreted a large amount of mercury according to the records that we have, is that correct?

A That's correct.

Q Does the fact that after the very first test he excreted a large amount of mercury, does that indicate that he did not have problems excreting mercury?

A That tells me that with an agent designed to mobilize mercury he was able to excrete it. To my

knowledge, that doesn't really inform us about his own innate capabilities.

Q In Dr. Green's records, he makes a statement, and this is at William Mead Exhibit 5, page 89, and Dr. Green states that, "William excreted no mercury with challenge while he clearly has a mercury load to deal with."

Is that consistent with the post-provocation test result?

A Okay.

Q And this is from a record dated --

A February 12th.

Q Right. And the test that was done was January 22, 2001.

A Okay, and the January '01 was the one that did show a lot of mercury on provocation, the one we just discussed, is that correct?

Q Yes.

A Okay. You know, I'm not sure what he means by that. Could you flip the other lab back up for just a second?

Oh, I'm sorry. Now I understand -- well, I don't exactly understand what he meant, but one thing that makes this difficult to interpret as flashed is that John has made a note that this is a spot urine

and not a 24-hour urine, and I apologize for not seeing this a moment ago.

So, John is saying that because that particular specimen, and you can imagine how difficult it is to get urine from these kids, was not obtained in a 24-hour fashion. I think he's saying that he can't rely on it, sorry, as showing the kind of excretion that they were saying, because they corrected it for creatinine and volume, and I apologize to the Court because I had talked about the issue that when you have the creatinine out of the normal range, the markers are done in order to compensate for your creatinine.

So, if you are compensating for it, assuming it's a 24-hour urine, and it's indeed a spot urine, you can't apply those same standards.

Q So would spot urine samples generally not be reliable for showing mercury levels?

A They're limited. You know, we try to get as long a sample as we can, but understandably we are limited by what the parents are able to collect. Yes, they are limited utility.

Q Thank you. Let's look at the next test which is William Mead Exhibit 5, page 34, and is this a blood test?

A This is a -- erythrocyte means red blood cell, so this is a test of the blood done in June '01.

Q All right. And was this a test result that you discussed yesterday?

A I don't recall. Can you tell me if I did?

Q I believe it was.

A Okay, thanks.

Q And I was wondering if you could tell me what you view as the significance of this result.

A Actually, you've just covered up the part that I viewed as significant. I believe that I was using this to talk about the low essential elements and how we use these blood tests to monitor the safety of chelation so that we can use them for replenishing supplements.

I also may have pointed out the low zinc and the low selenium which we pay particular attention to when we're replenishing, and I may have made a comment that since the mercury was not in the very elevated range, that I would not see evidence of ongoing high mercury exposures.

Q Now, explain for me why in this test you don't find the mercury results particularly significant whereas for the first test we looked at, the January 8, '01, test, you did find the mercury

level that was elevated there significant?

A Can you put them side by side? They were both red blood cells done by the same laboratory, is that correct?

Q I believe so. That's Exhibit 5 at 5. And I asked a vague question and let me try to be a little bit more specific.

A Right.

Q You found the mercury levels when they are elevated significant, but you didn't really find the ones where they were normal as significant, and I'm wondering how those relate to your opinion in this case that thimerosal and vaccines cause William Mead to have autism.

A Okay, now I understand.

When we red blood cell essential elements test that also includes the toxic elements at the bottom, we are primarily using it for the top half of the test, the part that looks at the essential elements. The only thing that the toxic elements part at the bottom tells me is whether there is concern that there is a potential ongoing exposure.

Red blood cells typically only live about 120 days, and obviously we don't know for any particular cell, you know, what day of their life we

were measuring them on here, so we use a rough estimate. When we look at the toxic part of the red blood cell essential elements, we use a rough estimate that it's telling us something about potential exposures within the previous or surrounding three-month period max.

We specifically are not using the red blood cell test to try to estimate body burden, and this ties to the fact that we believe that blood tests that look at mercury are only valuable for acute toxicities, which is not what we're alleging here. We're not alleging acute toxicity from mercury poisoning, and here, even though the level initially was at the 97th percentile, we're not alleging that that is evidence of this child having a body burden of mercury that's above the 99th percentile. We're only using the bottom part of this test to look at the possibility of ongoing exposures.

Q And you during that explanation used the term "we" a number of times, and again is "we" referring to your colleagues at the Autism Research Institute?

A Yes. Thank you. I'm sorry.

Q Let's look at the next test, which is William Mead Exhibit 15, page 98, and this was after

William had been on and off chelation for almost a year at this point, and this is an unprovoked fecal metals test, correct?

A Actually, would you mind blowing up the bottom? Yes, that's correct.

Q And here mercury was in the reference range, correct?

A That is correct.

Q And the date of this was December 26, 2001?

A Yeah. I believe you.

Q Okay. I don't believe you discussed this test result yesterday, and I was wondering if it was at all significant to your opinions.

A I tried to make the point that there is a wide ebbing and flowing of various excretions of mercury both in the stool and in the urine, and that we expect to see that and that we expect to see it fluctuate. So the fact that this was in the normal range at this point in time does not deter me from my overlying opinion, no.

Q Okay. The next test is William Mead Exhibit 12, page 8, and this was, I believe, a hair test on May 8, 2002, is that correct?

A That's correct.

Q Okay. And just for the record, as you've

testified earlier, you don't find hair tests particularly reliable, is that right?

A That's correct. Again, the best technology available back in the early 2000s, but we know now not as valuable as we would like.

Q Right. And just to be clear, I think you said that it's not reliable based on the age; that for infants it might be reliable but not for older children, is that right?

A Exactly.

Q The next test that we found in the records was at William Mead Exhibit 15, page 97.

A Yes.

Q And I believe this is a test that you discussed yesterday.

A Yes.

Q This is an unprovoked urine test on July 10, 2002, is that correct?

A Correct.

Q And here the mercury was within the reference range, is that right?

A That's correct.

Q Okay. Was a baseline test performed prior to this test? Do you know?

A I'm sorry. Didn't we decide this one is

non-provoked, so wouldn't that be the baseline?

Q Absolutely. Thank you for correcting me.

A Okay.

Q So this was the unprovoked urine test, and then was there a provoked test performed after this? Do you know?

A I don't recall.

Q Okay. Because the next test that we were able to find in the records was on August 14, 2002, and this is at William Mead Exhibit 15, pages 87 to 88.

A Yes.

Q And this is a provoked urine test, is that right?

A Actually, I'm having trouble finding where it says -- yeah, information regarding pre- or post-provocation was not provided. I thought that I had discussed that.

Q You did.

A Oh, okay.

Q So am I correct that the test, the unprovoked test on 7-10-2002, July 10th of 2002, that's not a normal -- the fact that the post-provocation test was done over a month later, that's not the normal protocol, is that correct?

A Okay. First of all, do we know that this was provoked or unprovoked because I think I can't know that.

Q Okay. So you don't know one way or the other?

A Right.

Q Okay.

A Right.

Q But assuming that it were a provoked test, the fact that an unprovoked test was performed a month prior, that is not consistent with the recommendation in the consensus statement, is that right?

A Well, let me clarify that the recommendation in the consensus statement, as I recall, says it is suggested, and the issue at that point in time, and I can't remember when the norms were tightened up, but the concern at that point in time had to do with this issue of normalizing for creatinine.

So if I could call your attention to the very top of the chart, this one is comparing it as a ratio to grams of creatinine. So whereas when we wrote the consensus statement in like '99 or 2000, we wanted to make sure that that happened.

I think that the labs that we rely on had since -- well, I shouldn't say that because I don't

know when they started doing that, but my point is that we were trying to deal with the creatinine issue. This seems to deal with the creatinine issue.

Q Ma'am, when you say "this seems to deal with the creatinine issue" --

A Doing the test such that it's reported out as in relationship to the creatinine as opposed to, for example, if I had a urine test and the lab reported back to me that the mercury level was, you know, 860, it would mean nothing to me until I knew what units of measurement they were talking about, and what it was in relation to creatinine. They do that here.

Q Okay. Are there any other metals out of the reference range on this test?

A Cadmium, cesium, chromium, copper, tetalinium, lithium, manganese, which may have been related to supplementation, maliptium, molybdenum, and possibly nickel, and can you scroll back up to show the name of the laboratory? Yes.

So based on what I said earlier, you understand that we are doing ongoing reliability testing.

Q So this test, William Mead Exhibit 15, pages 87 and 88, at this time it's not a test that you might

rely on, is that correct?

A That would be fair.

Q The next test that we found was at William Mead Exhibit 15, page 77, and this is a November 7, 2002, provoked urine test, is that correct?

A Yes.

Can you tell me if this is one that I showed yesterday or not?

Q I don't believe that you did discuss this one yesterday.

A Okay.

Q Was a baseline test performed prior to this one?

A I don't know but I think that the reason that I may not have included that is because I didn't think that it would be informative based on the reasons I have already stated.

Q And remind me what those reasons are again.

A That I find that there are other laboratories that seem to have more specialized expertise in the toxicology area.

Q Okay, so it's the lab and not the test?

A Right.

Q The next one that we found was from September 8, 2003, and this is William Mead Exhibit 15

at 67. This is not in the same lab, correct?

A Yeah. And Doctor's Data, at least based on my evaluation, seems to have a very good set of toxicologists on board, so at least to the extent that I as a general pediatrician can judge the competence of the toxicologists, Dr. Quig has been able to answer every question I've ever asked him about toxicology. So I have relatively more faith in this lab's expertise.

Q This test, I believe, was one that you did not discuss during your presentation yesterday. Is there a reason why you did not find it significant?

A I think that it was probably that it was yet another urine toxic metals from the same lab that shows an elevated mercury, and I thought that we had sort of established that as a pattern.

Q And do you know whether a baseline test was performed prior to this test?

A No. Since you have this up here though, I will say that one of our concerns about thimerosal-containing vaccines has to do with the co-existence of aluminum which we regard as a potential synergistic toxicity. So, one of the things that I do when I see a high aluminum, again it's just an environmental measure, is to talk to the parents about whether they

use aluminum-containing cookware, whether the child is taking a lot of aluminum-containing antacids, or whether they are using a lot of aluminum foil.

Q And when you say "we", I believe that there is a synergistic relationship between aluminum and mercury, who are you referring to?

A I'm talking about those of us that work with ARI and Defeat Autism Now.

Q Okay. And has ARI done any studies to determine whether there is in fact such a synergistic relationship?

A I'm not sure if we had, but I would be surprised if we used our limited resources to do that because that is something that's been very well documented in numerous papers throughout the toxicology literature. So I think we would regard that as a question that's already been addressed scientifically.

Q The next test that we were able to find was at William Mead Exhibit 15, page 120.

A Yes.

Q And this is a post-provocation urine test from February 10, 2004, correct?

A Yes.

Q And I believe this is another one that you

discussed during your testimony yesterday, is that correct?

A Yes.

Q And do you know whether a baseline test was done prior to this testing?

A I don't recall, but again, because the results are being reported by a lab that I trust in a way that reports the results in ratio to creatinine, it essentially obviates the concern that we expressed in the paper that we wrote back in '99.

Q Is there no other reason to perform a baseline test other than to deal with the creatinine issue?

A You know, actually, I don't know the answer to that not being a toxicologist.

Q The next test that we found was, I believe, another one that you discussed. This is William Mead Exhibit 15 at page 118.

A Yes.

Q And this is a test dated December 6, 2004, is that right?

A Yes.

Q Okay. And is this a post-provocation test?

A It appears to be so.

Q Okay. And just if you know, was a baseline

test performed prior to this test?

A I do not know.

Q And the next test that we were able to find was at William Mead Exhibit 15, page 116, and this is a November 12, 2005, test, is that correct?

A Yes.

Q And here lead and mercury were high, is that correct?

A That's correct.

Q Okay. Is this a post-provocation test?

A Yes, it is.

Q And to your knowledge, was any baseline performed prior to this test?

A I do not know. Again, I would be surprised if John would continually do that because each of the tests would have been out-of-pocket costs for the family, and I don't understand why he would feel the need to do it repetitively since his results were being reported out normed to creatinine.

Q Would the test be more reliable if a baseline had been done?

A Would the test be more reliable? You would expect the baseline to either show that the child was not excreting or may be excreting. To my knowledge, the ones that we have seen have shown benefit with a

provocation challenge.

So, I actually don't think that for the purposes Dr. Green was using this test it would have enhanced the reliability significantly in the clinical situation to have to compare it to pre-provocation.

Q If the pre-provocation test showed that the child was excreting mercury, then that might indicate that there was no need for chelation, wouldn't it?

A Perhaps.

Q The next test that we were able to find was Petitioner's Exhibit 15 at 114, and again this was a post-provocation urine test, is that correct?

A Yes.

Q And here mercury was in the reference range, is that right?

A That's correct.

Q Okay. And then the next test that we were able to find, and this is actually the last one that we were able to find, was William Mead Exhibit 15 at page 112, and this is a test from February 22, 2007, and to your knowledge, was a baseline test performed prior to this test?

A I do not recall.

Q What kind of chelation results would you expect to see in a person without heavy metal

poisoning?

A I don't know that I know the answer to that question. Not being a toxicologist, I am concerned that we all do have body burdens of various substances. So one of the areas of research that perhaps would be informative would be to do such a study.

Q And I believe you testified yesterday, and alluded to again this morning, that there is really not a standard pattern that you can identify for these kinds of test results, is that correct?

A That is correct.

Q And I think you testified yesterday that sometimes you get lead when you administer DMPS, is that correct?

A Occasionally, more typically you see more lead with DMSA, but --

Q Because DMSA is actually more associated with excreting lead, isn't that correct?

A Right. Right.

Q Okay. And DMPS is actually what you would administer more specifically for mercury, is that right?

A That's correct.

Q And sometimes you see tests where you've

administered DMPS, and you would expect to see more mercury, but you get more lead, is that right?

A Right, and one of my questions about that has to do with the other things we're doing for the child at the time. So I think I mentioned yesterday that I tend not to use chelation very much at all, but work more on the body's own mechanisms that are naturally intended to do that.

So, I think the thing we have may have lost sight of here is that at the time that Dr. Green was trying to use various chelating agents to the best of his ability, he was also doing the much more fundamentally important work in my mind, which was providing the co-factors for the body's methylation and transsulfuration biochemistry.

By giving supplements, specifically things like magnesium, or pyridoxal biphosphate, or methyl cobalamin or folate, all of those things are working on potentiating, to use the DOJ's favorite word, methylation biochemistry and sulfation.

And so we can't possibly control for how much of the body excreting lead or mercury at any given time is going to be a direct correlation of what agent we picked because there is so many other factors that are going on with the body, and John Green was

doing so many other fundamentally very important things in these children that I think it's very misleading to try to make too much from a pattern that fluctuates over time with different chelating agents when so much else was also being done to try to heal him, if that makes sense.

Q And, Doctor, just for the record, potentiate was your word. That's not the DOJ word.

A No, it was a bad joke. Sorry.

Q Doctor, since there is no standard pattern and we've seen with both William Mead and Jordan King that their results fluctuated, is there any pattern that you can imagine that you would interpret as supportive of mercury toxicity?

A I have told that to parents on the basis of their test results, and again I'm really hesitant to try to layout some quantifiable pattern because the science is still evolving, and I don't have it clearly established to be able to make a generalization.

So, what I can say is that when I have taken a thorough history, examined a child, looked at the symptoms, suspected that mercury or lead or other toxicities might be an issue, and then I get labs that come back and don't seem to support that hypothesis, then what I do is move on to other possibilities or

move that down lower on my list and move other things up higher on my differential diagnosis. So, it very much depends on the other labs that are involved.

Q Doctor, as you know, there are approximately 5,000 of these cases in this program, and the evidence that's being introduced into this proceeding is going to be applied to those other cases.

Are you saying that there is no pattern that you can identify that you would not interpret as not being supportive of mercury toxicity?

A No. I was saying that -- I thought you were asking me to make a judgment based on patterns of excretion related to chelation. Was that correct?

Q That is correct, and I'm wanting to know if there is a pattern that you would find to be not supportive of mercury toxicity.

A Patterns in which the children were able to show that they had good oxidative stress markers at the time of thimerosal-containing vaccines, glutathione working well, methylation biochemistry working well, those kinds of things. I think there is very limited utility for me as a pediatrician to propose a toxicological benchmark for your upcoming decisions. So, I think that you will need to rely on the toxicologists to try to help identify that, and I

would have to take myself out of the running as being not the person to deal with that part of the case.

Q But, Doctor, these are clinical tests, aren't they?

A Yes, and by very definition clinical means that I'm using them in context with the history and the responses of the child.

Q And so you would still leave it to the toxicologists to testify as to the pattern that would be not supportive of mercury toxicity?

A I thought that your question to me was what pattern would I propose as a general pediatrician, based on my clinical experience, that the government and others should use to decide 5,000 cases, and that is what I'm saying I don't want to do on the stand in this kind of a hearing. I think that that requires much more input and thought.

Q You do understand that these are test cases, correct?

A I do.

Q In fact, I believe you were the one who was actually responsible for selecting the test cases, is that right?

A That is correct.

Q Did you not have an understanding when you

were doing that that this evidence that was introduced into these test cases would be applied to the other 5,000 cases in the program?

A I did understand that. This is my first time testifying. I honestly did not anticipate that I would be asked to propose the kind of pattern that you're asking me to on the stand. And if that's part of my responsibility, I would be -- I cannot do that on the spur of the moment.

Q Okay. Doctor, a number of the tests that we looked at and that you relied on were from the lab Doctor's Data, correct?

A That's correct.

Q And that was the lab that you said that based on your experience you had a particular amount of confidence in, is that right?

A That's correct.

Q And do you send samples in from your own practice to Doctor's Data?

A I do.

Q How important is it to you that the labs that you rely on us reliable scientific techniques to test samples?

A It is important to me.

Q And when you consider the reliability of a

lab, how important is it to you that the lab uses consistent reference values when reporting lab results?

A It doesn't matter to me if they use consistently the same reference ranges as long as on any given test they are telling me what the normal reference ranges are. I think that there are indications over time when by virtue of a change in the technique in the lab the reference ranges might change. That happens with lab core all the time, well, not all the time, but you know, not unusually. So what would be important to me would be that the reference ranges are defined.

Q How important is it to you that a lab avoids contamination of both samples and controls?

A I would think that would be important.

Q And how important is it to you that a lab uses sterile solutions such as saline?

A Sterile saline. It would be important.

Q And how important is it to you that a lab replaces the saline if it is known to be non-sterile?

A That would be important.

Q And how important is it to you that a lab uses uncontaminated assays?

A That would be important.

Q And how important would it be to you that a lab replaces unexpired reagents?

A That would be important.

SPECIAL MASTER VOWELL: Is it unexpired?

THE WITNESS: Oh, I'm sorry. I substituted expired in my mind. Sorry.

MR. JOHNSON: Sorry. I misspoke.

THE WITNESS: Thank you.

BY MR. JOHNSON:

Q If Doctor's Data has problems with all of these areas and others, would you still trust their lab results?

A I would want to know the details about that and the timing. I have only really been evaluating them in the last several years on the basis of what I can judge as a clinician.

Q And do you do any hair testing in your practice? I know you said you don't rely on them, but do you send out hair samples?

A No. The only time I send it out is if I had parents coming in for intakes who had saved baby hair samples, and in that case I would send them based on the work that was initially done by Holmes and replicated at MIT. Other than that, I don't think I've ever sent a hair sample that didn't fit into that

category.

Q And would you send the hair samples to Doctor's Data?

A You know, I honestly can't remember if the hair samples we sent went to Doctor's Data or another lab. But I would have no reason that I know of now not to send it to Doctor's Data.

Q Okay. I want to show you a report from the New York State Department of Health, which inspected Doctor's Data Lab.

A Okay.

Q This is a letter from --

SPECIAL MASTER CAMPBELL-SMITH: Pardon me. Are you planning to introduce this as a trial exhibit?

MR. JOHNSON: Actually, yes, Special Master, we will. We can go and do that now.

SPECIAL MASTER CAMPBELL-SMITH: Okay, if we could have copies, that would be great. And this would be Trial Exhibit 2, the first of which was the Dr. Kinsbourne matter.

(The document referred to was marked for identification as Respondent's Trial Exhibit No. 2.)

SPECIAL MASTER CAMPBELL-SMITH: Thank you.

(Pause.)

BY MR. JOHNSON:

Q Have you had a chance to review the letter?

A I'm halfway through the last paragraph.

Q Take your time.

(Pause.)

A Okay, I think I have the gist of it.

Q Okay. Doctor, this letter is in response to a request made by Doctor's Data to perform multi-hair analysis on patient specimens collected in New York State, and the Department of Health denied a similar request in 1986, and in this 1999 letter, it noted, "The Center's 1986 decision was based primarily on concerns about external specimen contamination and lack of good reference values. These concerns persist."

And then further down, "Well defined reference intervals are an essential component of properly validated procedures. Our attempt to ascertain the derivation of Doctor Data's reference intervals has been confounded by the inconsistency of reference intervals posted on the lab's webpage and those reported in publication reprints purportedly supporting the use of diagnostic hair analysis."

Have you seen this letter before?

A No.

Q Does this letter change your opinion in any way regarding the reliability of Doctor's Data lab results?

A Well, actually, I'll point out a couple of thoughts that I have.

First, the concerns seem to be primarily related to external specimen contamination, and to my recollection Doctor's Data does send out information about avoiding certain kinds of shampoos and external contamination, et cetera, et cetera, in the time period when I would have sent the baby hair, which is many years after this letter.

Secondly, the concerns to my read seem to be primarily with reference ranges where you would be dealing with an argument about what the upper ranges of normal are and not using it in a situation in which you were looking for lack of excretion.

So, I had read some stuff in the literature about hair analysis which led me to decide not to use it for looking for high levels. I don't know that this would impact using it on baby hair because with the baby hair study what you are looking for is that the child doesn't show mercury, and so the levels are very, very low.

So, if anything, I think that these alleged difficulties would actually cause me to have more what would be a false/positive in the sense of a false indication that my baby was able to excrete mercury as opposed to not being able to excrete mercury. So, I really don't see -- even though I'm distressed to see this -- how it impacts on the way that I would use my hair analyses.

Q Doctor, we're now going to show you a 2005, actually it's a 2006 report from the New York State Department of Health, and we'll go ahead and mark this as a trial exhibit as well.

SPECIAL MASTER CAMPBELL-SMITH: That would be Respondent's Trial Exhibit 3.

(The document referred to was marked for identification as Respondent's Trial Exhibit No. 3.)

BY MR. JOHNSON:

Q I would like to draw your attention to a particular finding in this report.

MR. POWERS: Excuse me. Dr. Mumper, do you have the paper copy?

THE WITNESS: I do not, but if they -- or I don't think I do, but if they blow it up for me. Did

you give it to me? I'm sorry.

BY JOHNSON:

Q This is on page 10 of the report.

A Okay.

Q Blow that up for you.

SPECIAL MASTER HASTINGS: Is that page 10 of the exhibit?

MR. JOHNSON: Yes, Special Master. Sorry.

BY MR. JOHNSON:

Q And it states, "There is no system to monitor the technical competency of the assays. This is noted for bacteriology proficiency testing where a score of 100 percent has not been received for five events. For two events, extra organisms were reported. Their remedial action was noted as unsterile saline being used, yet the saline was not cultured or replaced. Extra organisms were also noted on internal proficiency testing with no remedial action performed. On two events, expected organisms were not reported. There was no remedial action."

Doctor, based on this document, does this cause you to have any concerns about Doctor's Data?

A It does cause me to have concerns with the caveat that we're reading one paragraph out of a multi-page document, and with the other caveat that I

don't know upon lab review what the standards are for corrective measures and those kinds of things. But this is very concerning, and I had not seen this before.

Is this my copy to keep, by the way?

Q Sure, you can have that.

A Okay. Go ahead.

Q Okay. I would now like to talk a little bit about some of the facts specific to William Mead's case, and some of the support that you provided in your report in particular about William Mead.

The first thing I want to ask you is that when you reviewed the record, and we'll start with William Mead's case, was there a certain profile that you were looking for to determine that the child's autism was caused by thimerosal from vaccines?

A I've not yet identified such a profile other than to say that with the understanding we have of the chronic inflammation I tend to think of it more in kids that I identify patterns in which they might not be able to handle a thimerosal-containing vaccine.

Sometimes it's kids who seem to be sick at the time that they get their shots or have chronic illnesses. Sometimes it's kids where the parents seem to report bad reactions to the shots. But I really

don't have a standard kind of clinical profile.

Q Is there a test that you consider to be conclusive evidence of thimerosal-related autism?

A No.

Q Is there a key piece of evidence in William Mead's case that you rely on for your opinion?

A I think that the -- well, there are a number of things that I relied on, and again because I'm a clinician and because I don't have a good marker, it's difficult for me to isolate a key piece. But the way that I thought of this case as I read it was that this is a child that I would expect not to have well operating oxidative stress and methylation markers on the inferential, indirect, incomplete and having improved over time since then. But given the evidence that John Green had when he was trying to manage this case, it seemed to construct a story of a child who would not be able to mobilize the mercury due to oxidative stress issues.

And then when Dr. Green looked for mercury, he found it, and when he started working on the child's medical problems the child's autism got better. So it would seem to me that we then have to include thimerosal in a list of potential environmental factors because if this was purely

genetically-related autism it's difficult for me to explain why it would switch on at 15 to 18 months, and then, you know, seemingly get better after interventions.

I will be the first to acknowledge that not being the treating physician it's hard for me to know which intervention might have really helped him the most. My suspicion is that since we deal with the whole child and take a systems approach, that it's a combination of what we do that gets the kids healthier and gets them better.

So, I have to go back to for me I think the key is the parents' story, the child's history, putting it together with the absence of physical exam findings that would be suggestive of other causes of autism, and then the biologic plausibility of damage from the injection of something that we know contains ethyl mercury which we know breaks down to inorganic mercury, which we know persists in the brain for as yet undetermined years or decades.

And so as a clinician I have to go back to that kind of careful synthesis as opposed to identifying one isolated factor.

SPECIAL MASTER CAMPBELL-SMITH: Counsel, let me just ask. Dr. Mumper, you did state that this was

a child that you would expect would not have good oxidative stress markers. Is this the type of child, as you indicated earlier in your patterns, based on William's ear infections, his series of ear infections and upper respiratory infection?

THE WITNESS: That is correct.

SPECIAL MASTER CAMPBELL-SMITH: Okay. Thank you.

THE WITNESS: Thank you.

BY MR. JOHNSON:

Q What is your strongest evidence that William Mead's autism was caused by thimerosal from vaccines?

A Well, I apologize if I have misspoken, but I think I always try to be very careful not to say that it was the cause, but to say things like it was a substantial contributing factor, or I thought that it exacerbated his problems, or I thought that it was in my best medical judgment contributing.

So, my strongest piece of evidence would be the demonstration that he excreted a body burden of a substance that is known to after injection into infants bypass the normal protective mechanisms of the gut, go across the blood-brain barrier, be broken down such that to me a very scary fraction persists as inorganic mercury, and that the best available

evidence of what we understand about the impact of thimerosal, not on direct neurotoxicity, direct killing or acute toxicity at all, but our best available evidence about how it affects things like calcium channel signaling and redox ratios and neuronal communication with disruption of inhibitory neurotransmitters and exacerbation of excitatory neurotransmitters. I think that is my best available evidence.

Q So the post-provocation testing is your strongest evidence, is that correct? Is that what you just said? That's what it sounded to me what you just said.

A No, no.

Q Okay.

A We're back to this bigger picture of utilizing indirect evidence, i.e., post-provocation urines, the laboratory data that I showed yesterday about him having metabolic acidosis which applies association with oxidative stressers, the evidence that we have by history and exam of his difficulty utilizing nutrients, the clinical picture related to his reported failure to thrive, those things taken together point to a child where we know that he received a substance that, at least based on animal

models, converts to inorganic mercury in the brain, and we know from in vitro testing that thimerosal has adverse effects on the crucial enzymes in his methylation pathway on glutathione, also in the methylation pathway on calcium channel, signaling and on crucial neurotransmitters.

So, I am only using the post-provoked urines as one piece of the puzzle that helps develop this bigger picture. So, I guess my strongest piece of evidence I'll have to say is those things that I determine on the basis of reviewing a comprehensive record that I, to the best of my knowledge and understanding, believe put him in a situation of oxidative stress and poor redox status at a time that he received vaccines known to convert to inorganic mercury.

Q If there were no evidence of oxidative stress, would you be able to reach the same opinion in this case?

A No, because we are acknowledging right up front that the vast majority of children that did get these thimerosal-containing vaccines do well with them, and you know, I'm a pediatrician. I have given thousands of vaccines in my life.

So, the problem is that we didn't have the

markers back in 2001 that are as good as they are now for oxidative stress. For example, you know, I would have loved to have seen an early on fasting glutathione in John Green's records, but you know, we didn't really have that then. I would have loved to have seen urinary neopterin and biopterin, but we didn't have that then.

The methylation markers that Jill James has done work on for three or four years still aren't commercially available. They would be wonderful to have but we don't have that then.

So, I don't think that it's fair to penalize the kids that were born at a time, or develop symptoms at a time when we couldn't sort all this out, and we couldn't have great lab markers. I think that we can be informed by what we've learned about other kids in the meantime where we have been able to do the measurements, and in my clinic about 80 percent of the regressive autism cases that I see, when I look for those methylation abnormality markers they have them, and so I use that clinical experience to extrapolate and I only do this because I don't have the markers available to me that I've seen the story over and over where the kids have multiple ear infections, they have chronic diarrhea, and when I send methylation by

chemistry markers on those kids it tends to come back abnormal.

So, I just -- I feel very bad that we're limited by what was known then in trying to make decisions about kids that were born when they were born, and are coming before us.

Q Do you look for the biomarkers you were just discussing in your non-autistic patients?

A No, and we acknowledge that we have not yet had good comparisons between normal kids and kids with autism. The labs that we use we hope are norming them accordingly based on normal populations. But one of the things we hope to do, and we've recently committed some of our very small resources into doing controls with the studies that we are doing.

We are going to find out, I feel quite sure, that there is going to be some overlap, and so that's when the whole picture and clinical judgment comes in. There may well be normal children who have some abnormal markers, but it's a matter of how many things are wrong with any given child, and how well that child is able to compensate. So, we are trying to do those studies. We just do not have very much funding to do them.

Q You state in your report in William Mead's

case that low zinc levels compromise the ability to excrete metals. What is your support for that statement?

A I was taught by -- I can't recall who in a toxicology lecture, it was one of the people that comes to the think tanks that zinc is one of the things that complexes with metallothionein in order to take mercury out of the body. My memory is that it's four molecules of zinc that's necessary to take one unit of mercury out, but I would not want to hang my hat on that number.

Q So, your support for that statement was a talk that was given by someone at -- is that an Autism Research Institute think tank?

A Yes.

Q You would agree that low zinc levels can be caused by diet. In fact, I think you testified about that, is that right?

A Yes.

Q Could low zinc levels also be caused by chronic infections?

A Yes.

Q And William Mead in this case had persistent bronchitis and other infections as a child, is that correct?

A Right.

Q And could rapid growth also cause low zinc levels?

A Yes, I'm recognizing a number of these from one of my slide presentations.

Q You state in your report that low plasma amino acids documented at Massachusetts General Hospital, and you may have actually discussed these or shown these test results yesterday.

A Right.

Q You cite these as support for your opinion. Can you explain the significance of those test results?

A I'm going to go back to the paper because I worded that very carefully.

SPECIAL MASTER CAMPBELL-SMITH: Just for my own reference and the record, this is page 5 of Dr. Mumper's expert opinion in the Mead matter.

MR. JOHNSON: Thank you.

THE WITNESS: All I see that I said in that report was that one of the things I listed as laboratory evidence of impairments was low plasma amino acids documented at Mass. General Lab. I had tried to be very careful when I discussed these yesterday to acknowledge that it did not show any kind

of classic pattern of in-born error of metabolism.

In looking at the particular amino acids there, I had gotten -- there is a biochemistry chart that I brought with me that I use when I'm trying to look at laboratory values, and it hangs in my clinic, and what I do is look at the pathways that are affected in those particular cases and see if can tie it into what we do know about autism pathology.

So, in looking at these particular markers, there were several of them that -- the concept here is that if you've got one thing, substance A, and you're trying to make substance C, but you need something to drive that reaction, that something might be an enzyme, a nutrient, a co-factor.

So when you see low levels of a substance, that makes me want to look back in the pathway and see what would have had to happen in order to take the precursor to the amino acid that was at a low level, and you know, one possibility is always diet, that the child is not eating protein enough to make the amino acid, but another possibility is that that co-factor, which in some cases is the conversion of ATP, or in some cases is a cellular mineral like zinc or magnesium, if I can discern any patterns.

And here the cysteine and the cystathionine

are one of the things that is pretty consistently now implicated in the methylation biochemistry that we're concerned with, and I may be misrecalling the numbers but I thought that the cystathionine at the Mass. General Lab was actually reported as zero, although the reference ranges were a little confusing in that it was marked as less than three would be the reference range.

So, in terms of looking at those labs in a functional way as opposed to a way of looking for an in-born error of metabolism, those impairments I was able to tie directly to either the methylation biochemistry or the glutamate pathway. So that's why I tried to word this very carefully to say that it was a laboratory evidence of impairment, but not to try to overinterpret it anymore.

BY MR. JOHNSON:

Q And these tests were not typically used to determine mercury toxicity, is that right?

A That is correct.

Q You also state in your report that William had dramatically low digestive enzymes, is that right?

A Yes.

Q Can you explain to me how that is related to autism?

A It will be a little bit of a longer explanation, but yes.

One thing that we're concerned about is that there is an enzyme called DPP-4, which stands for diopeptidyl peptidase 4. There was evidence, I think, as early as the early eighties, that certain toxins, such as organophosphates and mercury, inhibit the function of that enzyme, of DPP-4.

DPP-4 is one of the things that the body usually uses to breakdown gluten and casein. So one of our possible mechanisms by which children with autism can benefit from a gluten-free, casein-free diet, as both of these children did, is that they no longer have to process a food that they lack the enzymatic ability to digest.

So, there is concern about mercury and other toxins effect on that enzyme, but my understanding is that that's somewhat beyond the scope of what we're presenting primarily, but nonetheless we're dealing with the full child.

The other way that that's related to autism is that there are lots of neurotransmitters in the gut. I think something like 70 or 75 percent of serotonin is actually in the intestine, and serotonin is one of the more well known neurotransmitters

because it's what Prozac and Zoloft and all those things are working on the SSRIs.

The test that was done at Mass. General, to the best of my knowledge interpreting it, showed very, very low digestive enzymes prior to a secretin infusion. After the secretin was infused the child had a dramatic increase in -- I believe it was trypsin, amylase and lipase. This has been reported in a small subset of children to be remarkable in restoring their former normal neurodevelopment, and the mechanisms of that are all worked out, but it's undeniably true that there are children that dramatically benefit from this.

The most remarkable example was a child that was going in, who happened to be autistic, went into Dr. Horvath's lab at University of Maryland sometime in the late nineties, and had a secretin infusion which was purely at that point designed to try to figure out how his gut disease -- you know, what was going on with his chronic diarrhea, and I can't recall the details of how non-verbal he was or if he only had a few words, but he essentially started talking in sentences, and what happened then was that there was a big rush in this country for everybody to use secretin, and many parents paid a lot of money and

were very disappointed that their child didn't have similar results.

So, I use this as an example of how there may be many contributing factors to autism, and for how -- then when we tested secretin in the alleged, you know, good trial, the effects washed out, and what happened was that there was a small population of kids that did great and improved dramatically. Then there were other kids who didn't improve. So that when you looked at the results all together it showed that there was no improvement.

So, in William, my understanding was that Dr. Green was using that very dramatic information from Mass. General to inform a rational treatment plan by providing digestive enzymes to the patient. So, that's the first part of the answer, which is that it may be affected by mercury when the normal pancreatic enzymes are low, and when we give secretin the child improves.

There is another more complicated answer which is that the use of digestive enzymes tends to not only improve the gut status of the children in terms of helping them absorb their nutrients, because one of the issues that concern me about William Mead when I saw that he had virtually undetectable levels

of the enzymes that would have made him break down proteins and fats and carbohydrates was that essentially he had been operating with his tank empty for quite awhile, and he wasn't able to turn those food stuffs into his body part, so by definition that was another piece of evidence that caused me to conclude that he was under chronic oxidative stress.

So, the second part of it is above and beyond treating the child so that they can absorb their food and utilize their amino acids and their fat correctly, there is also the issue of how if you lack digestive enzymes you're likely to be presenting with chronic GI symptoms, and I am one of those that's concerned, based on what parents have told me over and over, that when the child's gut is not good the child has more autistic symptoms, and all those mechanisms remain to be worked out, but there is a body of literature looking at how that happens.

Welch, for example, did a study in animals in which she actually gave them gut inflammation, and looked at their brains, and the gut inflammation did impact on neurologic function.

So, that is my best and shortest answer about the relevance of looking at digestive enzymes in children with autism.

SPECIAL MASTER CAMPBELL-SMITH: Before you go on for my own reference and I'll ask you to do this perhaps beforehand, the reference that you had to page 6 of Dr. Mumper's report.

MR. JOHNSON: Sure, Special Master.

BY MR. JOHNSON:

Q Doctor, you mentioned the secretin study. Isn't it true that in that study that that was a placebo double-blinded trial, correct?

A Right.

Q And isn't it true that in that study it was actually the children who received the placebo that showed more improvement?

A That was not my understanding. My understanding was that there were either one or two dramatic responders in the treated group whose responses were wiped out when the data was analyzed in a way that would show the overall effect on the whole population.

And the reason that I'm pretty sure about this is that we've used this as more impetus to try to develop biomarkers for subpopulations or symptom constellations for subpopulations so that when we do clinical research we're not putting apples and oranges and cantaloupes and bananas together in treating all

those differences with one single treatment.

I don't deny that the placebo response is real.

Q And the result of that study and the conclusion of that study was that secretin was not effective in treating the symptoms of autism, is that correct?

A That is correct as I've just explained.

Q Your discussion of the digestive enzymes and the issues with the gut, does that relate to -- I'm trying to find a page reference for the Special Masters -- does that relate to your reference to intestinal dysbiosis? And this is on page 6 of the William Mead report.

A Again, I don't have the pagination so can you tell me what the bolded title is?

Q It's "Clinical Evidence Compatible With Damage from Mercury".

A Yes. Not directly, no.

Q What do you mean by intestinal dysbiosis?

A In children who have received multiple antibiotics, especially if they were given in the very early period of time as with Jordan King where his mother got antibiotics during pregnancy, during delivery and then she also got them while she was

breast feeding, that interferes with the process called immune modulation.

The gut is actually a very important part of modulating the immune system. Normally what happens is that the baby is populated with things like lactobacillus and bifida bacteria from the mother's breast milk, and those are so-called good flora that do things for us like making vitamins and demethylating mercury, for example, methyl mercury here primarily, not ethyl.

And so if you have a situation in which because of early antibiotic use, and those of us at ARI are concerned even about one or two courses of antibiotics when given very early on in infancy, you can see a pattern where the good bacteria are wiped out and that interferes with the normal balance in the gut where the good bacteria and the good yeast are trying to fight out and live in symbiosis with organisms which might otherwise become pathogenic.

So, we look for intestinal dysbiosis to look for low levels of these good bacteria so that we can potentially use probiotics to elevate the good bacteria. One of the reasons that we like to do that is that when you do that in early infancy you actually have less incidence of asthma and allergies as the

child ages as shown in a large European study. So, we look for that and treat it in the context of treating the whole child.

Q Now, you're not a gastroenterologist, is that correct?

A That's correct.

Q And this concept of intestinal dysbiosis and the involvement of the gut, did that originate with Dr. Wakefield's research?

A I do not think so. I think that that concept had been around before Dr. Wakefield, but he certainly also agrees that that is an important part, so he's certainly talked about that, and I have to say I agree with him that it is an important factor.

Q And Dr. Wakefield's research involved the measles component of the MMR vaccine, is that correct?

A That's correct.

Q Okay. Do you believe that the MMR vaccine was a substantial contributing factor to William Mead's development of autism?

A You know, I don't really know. I am worried about it. I'm concerned about triple live virus vaccine being presented to a potentially oxidatively stressed child with prior antibody cues. I don't have a way of knowing with certainty to what extent MMR may

have contributed, but my understanding was that in this particular case my task was to look at the thimerosal-containing components, and that there were other cases that were going to examine MMR alone, or potentially even MMR and TCV in combination.

Q So you just didn't look at the MMR, is that what you're saying?

A No. I looked at it and I believe that I listed when he got it, which as I recall -- let me make sure -- he got it at the so-called usual time, 12 months. He also received Varivax on the same day.

Q And so you included it in your differential --

A Yes.

Q Okay. And you ruled it out?

A No.

Q Let's now move on to Jordan King and talk a little bit about your report in his case.

A Okay.

Q On page 4 of that report, and it's actually under the section that begins on page 3, "Clinical Evidence", you note a number of other potential exposures for Jordan, including pesticides, fungicides, toluene, and tuna.

A Yes.

Q What role do those exposures play in your causal analysis?

A At the time that I was writing this report I was developing a differential diagnosis in which I was trying to see any possible contributors. Our work at ARI has led us to be concerned about fungicides, some of which contain mercury and many of which were taken off the market because of their mercury content.

Pesticides are in our differential diagnosis of what can be harmful to some children with autism. A lot of that work has been looked at by Paul Shattuck and others.

The toluene that went on the deck, I wasn't sure at the time that I wrote this report whether or not the child was playing on the deck or playing in the yard at the time that it was applied, and so I wanted to raise that as a potential co-existing or exacerbating toxicity.

Since then I'm actually less concerned about it because I was able to interview Mrs. King and find out that she actually took her son to the park and the museum that day because she was very environmentally savvy mom who did not want him to be exposed.

Then the tuna is a potential source of methyl mercury, and especially in a child who has

chronic diarrhea where his normal gut mechanisms or he has low selenium so that his ability to excrete other sources of mercury might be impaired. I thought it was only fair to include things in my report that could be possible contributing factors to his autism.

Q You've addressed the toluene. Were you able to rule out the pesticides as a potential contributing cause?

A I really don't know how to rule out the pesticides or fungicides as a contributing cause based on the medical records that I have. No.

Q And you noted in your report that Jordan ate a lot of tuna which could also contribute to the total mercury load, is that correct?

A That is correct.

Q Is it part of your opinion that Jordan suffered from glutathione deficiency?

A I don't think that we have direct evidence of that, but based on evaluating many children similar to him and looking at John Green's clinical decisionmaking and also taking into consideration the fact that the parents report improvements with therapy that we design in order to make glutathione work better, specifically the methyl cobalamin injections which provide the precursors to make more glutathione,

and if I'm not confusing the cases, I believe Mrs. King testified that even now she can tell if a day goes by when he doesn't get his MB12 shots.

So given the fact that I'm trying to evaluate a child who was presenting in 2001-2002, all of those are consistent to me, although indirectly and inferentially, with glutathione deficiency, yes.

Q I want to direct your attention to -- it's actually mentioned in your report, but the actual record cite is Jordan King Exhibit 1 at 3, and we'll pull that up for you. This is a note that indicates that Jordan is doing amazingly well with B-12 and glutathione, is that correct?

A Yes, and it says "incredible difference, just wanted you to know."

Q And this appears to be perhaps one of the parents who called in. It looks like MyLinda King who called in and reported that, is that correct?

A That's correct.

Q Okay. And the date that this was called in, this is April 19th, it looks like 2001, is that right?

A I think so, yes.

Q Looking at the records, it appears that, and this is Jordan King Exhibit 8 at pages 21 to 22, let's pull the date up on this. So this is a few months

later, it says that, "Jordan demonstrates a high level of distractibility. He is able to maintain a sitting posture, demonstrates dynamic mobility but without purposeful attention to anyone passed."

Does that indicate that glutathione and B-12 were making him improve?

A You are drawing one aspect of a record that demonstrates he's not doing well, and asking me to make a judgment about one particular therapy given many months before. I would have to in that situation know more information about what Mrs. King was thinking and specifying when she said "doing amazingly well." She may have meant that he was attending to task more, or she may have meant that he was talking more, or she may have meant that's when he started showing more affection to her, or playing with his sister or any other number of other things.

Q In your practice, what do you typically look for to see whether therapy such as B-12 or glutathione are causing improvements? Is there any one particular thing?

A We have a 145 question questionnaire that we use. When we do methyl cobalamin injections, we do a five-week period of time in which the parent is asked not to start any different interventions or not to

start any new therapy. We ask the parents to fill out the questionnaire but we also ask the parents to keep all the therapists blinded, all the relatives blinded as to the intervention.

We bring the family back five weeks later, and the parents have filled out this behavioral questionnaire, so in that piece of it there is inherent reporting bias and the potential for placebo effect, which I acknowledged up front. But we also asked for notes from the therapists.

It's ideal if we can get like the number of words he was saying in speech therapy before the B-12 and then the number he was saying at the five-week point and compare that to his previous trajectory of progress.

So whereas we expect that children will improve over time, what we're looking for is a change in the trajectory of that improvement, and in my experience having done this now in at least 200 kids, if not more, our experience is that we find demonstrable behavioral, language, et cetera, improvements in a substantial subset, probably greater than 50 percent in my experience.

The other thing that we know that we're doing though, even in the absence of clinical

improvements in speech or repetitive behaviors or social interactions or the so-called autistic stereotypic behaviors is that if we have a child who has demonstrated to us that he does not have glutathione in adequate amounts or that he has low methionine and therefore has that crucial methylation biochemistry disrupted such that he's not making good cell membranes, he's not regulating his genes appropriately, he's not making normal neurotransmitters, I feel that there is justification in fixing that biochemistry and the improvements in the autism symptoms are nice for the families and a wonderful bonus, but not the only reason to do the MB-12, if that makes sense.

Q Okay, let me ask you this. In the William Mead case, you testified that if there was no evidence of oxidative stress, you would not be able to reach the same opinion.

In Jordan King's case, is oxidative stress essential to your opinion that his autism was substantially contributed to by thimerosal-containing vaccines?

A You know, I have a little bit different formulation on him because I think that he has more evidence, at least for the potential of

environmentally-mediated synergistic toxicities. I think he has a different pattern in that he got antibiotics very early on but not continuously. You know, once he was born, he never really got the antibiotics himself, so my issues with risk factors in him are related to gut issues also.

I do think that he had times at which he was under oxidative stress. I just don't have a marker retrospectively for quantifying that at the time of receipt of his thimerosal-containing vaccines or at the time he was trying to process those.

Q And again, I apologize. I'm trying to find a page cite. Maybe you can help me. This is where you comment on Jordan's amino acid analysis and indicate that it demonstrated impaired xenobiotic detoxification.

A By the language, I am assuming that that was one of the functional labs that had a chart and again this is looking at function and not in-born errors of metabolism. They have looked at the biochemistry and made assessments of clinical clues on the basis of the analytes that would be suggestive of xenobiotic toxicity, and so that is relevant in that we include that in our differential diagnosis.

Q And when you were reviewing the records, did

you see records from a Dr. Anadiotis? That's A-N-A-D-I-O-T-I-S.

A I think so because I think that we talked about it yesterday.

Q Okay. And if we look at Jordan King Exhibit 12, page 21.

A Yes.

Q Dr. Anadiotis noted that Jordan never had true metabolic testing and that he was struck by the differences in the laboratory values assumed to be abnormal by the treatment centers versus those reference values that I know he knew are used in academic institutions across the country, is that correct?

A That's correct.

Q And as a result of that, Dr. Anadiotis recommend that Jordan receive standard immunoacid testing?

A Yes, a standard plasma immunoacid and a urine organic acid study, and I did not see that in his record, so I do not know if that was done.

Q Was that not testing that was done at Oregon Health Sciences on September 27, 2001? I can show those to you.

A Oh, I'm sorry. Maybe so.

Do you have the previous page that would show the analytes.

Q We're showing that up now.

A Okay.

Q Is it your understanding that this is the testing that was recommended by Dr. Anadiotis?

A Yes.

Q Okay. And do you agree that the conclusion of that test was that there were no diagnostic findings?

A Yes. Can I see the first page back again though? I'm sorry.

Again, the standard utilization for that test is to look for in-born errors of metabolism. The thing that might potentially be informative in this case again has to do with methylation biochemistry in that there was a low cysteine. Without it up now, I think it was like 13 when the normal was 22 to something. Even on the standard university test was showing up and that's an area that we're particularly concerned with with our methylation biochemistry cycles.

So again, a functional isolated inferential suggestion that there may be problems in the pathway, and that's all I can really get from that, and I

totally acknowledge that the university test is not showing in-born errors of metabolism.

Q Doctor, would it be fair to say that your opinions in this case that are primarily or in large part based on your belief that certain treatments that were provided to both William Mead and Jordan King were effective?

A In some part, yes.

Q Okay. And that's also based on your own clinical observations from your practice, is that correct?

A That's correct.

Q But you have not published any control studies based on your patient population except for, or you haven't published any control studies, have you?

A No.

Q Okay. You said that you were slow to come around to chelation but you do actually chelate children in your clinic, is that correct?

A I do now, yes.

Q Okay. What happens biochemically when you chelate a child?

A It depends somewhat on the agent, but the basic idea is that you're trying to use an agent to

grab on like a claw is where the word "chelate" goes to, and escort the offending agent, whether it's lead or mercury, out of the body, frequently largely in the stools and urine.

Q And I believe that you testified that it's your understanding that chelation does not remove mercury from the brain, is that correct?

A That's correct.

Q Do you chelate your patients with the oversight of an institutional review board?

A No.

Q Would you agree that the Defeat Autism Now consensus statement says with relation to chelation therapies that no well controlled outcome studies have yet been performed?

A Yes.

Q Is that still true today?

A Yes, that is true. We were hoping to get that done through NIH, and I was working with Sue Swedo but the project got stalled. That was about a year ago.

Q Is DMSA approved by the FDA for treating mercury toxicity?

A No, but it is approved for lead toxicity, and I typically start with the FDA-approved agents and

typically use DMSA first. Most of my patients have at least history consistent with and some evidence of lead when I do that.

Q Do you also use the gluten-free, casein-free diet in your practice?

A Yes, I do.

Q How does the gluten-free, casein-free diet treat thimerosal-related autism?

A I'm not at all sure that it does.

Q So if a child showed improvements on the diet, that wouldn't really be supportive, in your opinion, of mercury toxicity?

A In terms of directly, no.

Q Do autistic children who haven't received biomedical intervention ever improve?

A Yes.

Q Dr. Mumper, we asked you earlier whether you were the one that was responsible for selecting Jordan King's and William Mead's cases as test cases for this proceeding, is that correct?

A Yes.

Q Why did you choose those two cases as test cases?

A Well, it was a complex set of decisions. One was that they were kids that we had some evidence

of mercury excretion on them. Secondly, we wanted to put forward some instructive cases and I conceived of these cases differently. I didn't want to bring forward, you know, the first three test cases that all essentially had the same history.

So these cases represented to me clinical patterns that I see in my practice, and William Mead represented to me the kids that have a lot of antibiotics, ear infections, respiratory infections, plus or minus asthma and allergies, and Jordan King represented to me a child where the potential vulnerability probably happened earlier on with his mother's antibiotic use during pregnancy, and also raised the issue of synergistic toxicities.

In both case, I thought that there was very good evidence that they weren't classic autism cases from either a chromosomal standpoint or the children with autism who seem to be abnormal from birth. So their pattern of initially being developmentally normal, being exposed to an agent that could breakdown to inorganic mercury in their brain, and then having a subsequent regression with loss of milestones and then emergence of autistic behaviors seemed to provide two patterns that will need to be tested in the system.

It would be nice if we could take kids who

had more laboratory data, but one of the things that I ethically felt bound by was this idea that we should test some cases for the kids that weren't able to get the million dollar workup, and where we would be forced to rely on things like clinical histories and the reports of the parents.

So of the six or seven cases that I was given to review, those are the reasons that I can recall that I ended up choosing these two.

Q Can you now tell us without talking about specific cases, but some specific reasons that you decided not to choose the other cases that you reviewed?

A In many of them, I had such little informative laboratory data that I didn't think that I would be able to make a strong enough case because I do want this process to be driven by the science, and I was concerned that if we didn't have at least some type of biomarkers to present, that there was a possibility that the parents' stories might continue to receive less than the respect that I think that they deserve.

Q And when you say the lab testing, what particular lab testing was not present in those cases that caused you to think that you would not be able to

form an opinion?

A Well, I honestly don't remember because I was reviewing these in September, October, and since then I've seen a lot of patients and given a lot of lectures, but what I would have been looking for would be something that could tie it to some type of footprint for mercury or some type of footprint for methylation and transsulfuration abnormalities, and so it was a relative value strength type of judgment. And I'm sorry, and clear documentation of regression was also an important criteria for me.

Q Doctor, you are a member of the American Association of Pediatrics, is that correct?

A The American Academy of Pediatrics?

Q Yes.

A Correct.

Q I'm sorry. Have you ever served on any committees for the American Academy of Pediatrics?

A Yes.

Q Okay. What committees?

A The School Health Committee back many years ago, and currently we're actually being sought out by the American Academy of Pediatrics for our opinions on these issues. I was invited to meet in March with the current president, the upcoming president, the head of

mental health for the AAP, the executive director and a couple of other people that I'm forgetting, and we made plans for them to sit down with 15 or so of the so-called senior clinicians and researchers in DAN, Defeat Autism Now, who will -- we hope to have a brain-storming session and interchange where we try to teach them some of what we think we know, and they represent the importance of, you know, vaccines for public health reasons, and we come to some kind of plan for how to make vaccines safer.

Q Is that a formal committee?

A No, not yet, but it will be a formal brain-storming session think tank, potentially an ad hoc committee. I'm not sure where the plans will lead.

Q So the one committee on school health back many years ago, that's the only committee you've actually served on with the American Academy of Pediatrics?

A To the best of my recollection yes.

Q Have you ever served on any NIH committees?

A No.

Q Are you a member of any NIH councils?

A No.

Q Have you ever received a research grant from NIH?

A No, nor have I applied.

Q Have you ever served on an editorial board for a scientific journal?

A Yes, but only once.

Q And which journal was that?

A American College of Physicians and Surgeons.

Q And you were actually on the editorial board of that --

A Oh, oh, I'm sorry. I may have misunderstood the question. I did misunderstand the question.

I was not on the editorial board. I was asked to be a reviewer.

Q And how many articles did you review for that journal?

A Only two or three. I'm primarily a clinician, so it's really without -- you know, outside the scope of a typical clinician to even do clinical research, write papers, you know, review them, et cetera.

Q When was the last time that you reviewed an article for the Journal of American College of Physicians and Surgeons?

A Between two and three years ago, I would guess, but I'm fuzzy on the date.

Q In the past few years, it appears that most

of your professional involvement has been with the Autism Research Institute and Defeat Autism Now. Is that a fair statement?

A That is a fair statement.

Q And I believe you have a position with Autism Research Institute?

A Yes, I am their medical director.

Q Is that a paid position?

A I get a stipend for organizing the conferences twice a year. We plan them over a six-month period. I'm gone away from my practice for six, no, I'm sorry, eight days, and the stipend is either 16 or 18 thousand dollars for that meeting.

Q You mentioned that when you were a member of the faculty at the University of Virginia that you received a student -- oh, an award --

A Resident.

A -- voted on by the students. Did you receive any awards that were decided by the faculty?

A No.

Q It appears from your CV that you left UVA or stopped teaching at UVA about the same time that you got involved with the Autism Research Institute, is that correct?

A I need to clarify what my situation was

there. I was not actually teaching at the UVA campus. I was teaching in a residency program for family physicians that was affiliated with the University of Virginia, so I was actually leaving Central Hospital and the residency program, and not leaving the UVA campus.

Q When was the last time or how long ago was it when you last taught a student from UVA?

A I think about three years ago I had a student in the nurse practitioner school at UVA for a year.

Q Are you sure it wasn't more like 2000 that you last taught a student at UVA?

A Are you talking about a medical student from UVA?

Q Yes, a medical student.

A Okay. Well, around 2000, I would have been teaching medical students through the residency, and I'm trying to remember if any of the medical students that I had post-2000 were from UVA. I don't think that they were. I think they were all from other places.

Q And isn't it true that in 2005 the university actually decided to terminate your position because you hadn't taught any students for a number of

years?

A That's correct.

Q You mentioned during your direct examination that in your practice you have about --

A Oh, I'm sorry. Can I modify my last answer?

The letter that I received was that they weren't going to renew my position teaching residents because I had not taught them for many years. I actually did get my appointment renewed for several years after I left the residency, so I'm concerned about the connotation of the word "termination" versus not renewing my clinical appointment.

Q Doctor, you testified during your direct examination that you had medical records for about 2,000 patients in your clinic, is that correct?

A Two thousand total patients. That includes general pediatric patients.

Q How many of those patients do you follow on an ongoing basis?

A I really don't have a good way of estimating that. I'll tell you that I see patients typically three and a half to four days a week, and that it's usually about 35 patients a week.

Q How many of the 2,000 patient files that you have in your office were consultations from other

physicians?

A I would suspect about 100 because the vast majority of those 2,000 files, you understand, are primary care pediatric patients that, you know, the parents choose me out of the phone book or by word of mouth or whatever, but I think probably about 100.

Q Okay, and I believe you testified that about four to five hundred of your patients are your autism patients, is that correct?

A I think that's correct, but it's difficult for me to nail down the exact number because of the way my records are set up.

Q And the 100 files, or around 100 files that you think are consultation files, are those autism patients that have been referred to you?

A Some of them are. I actually also get consultations from other doctors for things like chronic diarrhea, chronic failure to thrive, food allergies, situations in which the referring doctor might perceive that the parents are concerned about chronic illnesses that the referring pediatrician doesn't have either the time or interest to be able to address in a busy pediatric practice.

Q So, four to five hundred, I guess, is your best estimate for your autism patients?

A Correct.

Q For how many of those four to five hundred patients have you concluded that a thimerosal-containing vaccine caused or contributed to their ASD diagnosis?

A I have no way of knowing that.

Q I mean, Doctor, they are your patients, and I assume that you've reviewed their records, right?

A Right.

Q And you've performed an examination.

A Right.

Q And you've taken histories from their parents, correct?

A Right.

Q So you actually have more information about those patients than you had when you prepared your reports in these cases, so it seems to me you would have to be able to ballpark the number of those patients that you believe their autism was contributed to by thimerosal-containing vaccines.

A Okay. So I do know that based on the review of my last 156 patients, only about 50 percent of them were clearly regressive. So, one could postulate that the number from which I would draw the thimerosal-containing vaccine contributed patients would be half

of four to five hundred, so 200 to 250. So, I would say somewhere less than 200 patients, somewhat more than one.

Q So between one and 200 of the four or five hundred you would estimate their autism or ASD was contributed to by thimerosal-containing vaccines?

A It's very difficult to work out the subset. Yeah, obviously, this has not been well studied, and again I really resist the idea of trying to give a number to the clinical work that I do.

Q You believe there is an epidemic of autism, correct?

A I do believe that there is an epidemic of neurodevelopmental disorders. The best numbers are that one in six children in this country now has a neurodevelopmental disorder. That comes right from the American Academy of Pediatrics, and the CDC reports one in 150 children now with autism.

Q And in fact you've stated in the past that in Virginia where you practice there was an eleven-fold increase in autism cases since 1988, is that accurate?

A That's based on DOE data, Department of Education data, and that obviously is subject to the idea that there is potentially some ascertainment bias

and all those things that have been looked at. But yes, those were the numbers that I was given.

Q And you mentioned the notion of ascertainment bias. What percentage of the increase would you attribute to ascertainment bias?

A I don't know, but I think that it is real, so somewhere between 15 to 40 percent perhaps. It's a matter of some subject. I don't believe that there is diagnostic substitution. I do think that the term "ASD" may be used more broadly now, but you recall that the reason that I went into this work is that I perceived there was a qualitative change in children, and so before I ever knew the DOE numbers, I was seeing something in my practice, and that was, frankly, before I had even thought about thimerosal or toxin-induced autism, or you know, any of those issues.

Q And that leads to my next question. What percentage of the eleven-fold increase that you saw since 1988 did you attribute to thimerosal-containing vaccines?

A I don't know. I don't know.

Q Do you have any guess at all?

A You know, I really don't. I don't want to be tied down to a number for something that's not been

well studied.

Q The amount of thimerosal that children receive through vaccines increased from the 1980s to the 1990s, is that correct?

A That is correct.

Q Okay. When did you the schedule change?

A To the best of my recollection, Hib vaccine was added around 1988, and actually now that I think about it, you had asked me if I had participated in any controlled studies, and I did participate in the Hib vaccine trial in my clinical practice back then.

In the early 1990s, Hepatitis B vaccine was introduced at birth, and then prevnar somewhat later. So there were changes over a period of five to seven years.

Q What amount of thimerosal did a child receive in 1985 if he or she received the full schedule of vaccines?

A I would think that at that point it would have been five DPTs, which would have been 25 micrograms, so that would be half of 125, which is 62.5 if I did my math right.

THE WITNESS: Tom, did you check my math?

MR. POWERS: I did not.

THE WITNESS: Thanks a lot.

MR. JOHNSON: It sounds right.

BY MR. JOHNSON:

Q When the schedule changed, and I believe that was around 1994, is that correct?

A Well, there were a series of changes. Hib was '88-89. Hep B was '90-91, somewhere in there, and prevnar in the '94 range, if I'm remembering right.

Q So by 1994, if a child received the full schedule of vaccines, what amount of thimerosal would that child receive?

A Counting or not counting the preschool boosters?

Q Not counting.

A I think it would have been 37.5 micrograms after the initial infant series, including the Hib and the DPT boosters, but not including the four-year-old DPT boosters.

Q Was there a corresponding increase in the number of autism cases after 1984 - 1994?

A My memory of the charts is that the increase started in the -- the dramatic increase started in the late eighties and then continued throughout the nineties and early 2000s.

Q And thimerosal was taken out of vaccines in 2001, correct?

A Thimerosal, I take issue with that statement. In 1999, the decision was made to phase out the use of thimerosal. Between '99 and 2001, efforts were made to manufacture vaccines without thimerosal. It was never taken off the shelves. So in looking at my patients who present to my clinic and looking at the lot numbers of their vaccines and trying to trace back whether or not it was thimerosal-containing or not, we had at least one patient in 2003 that received thimerosal-containing vaccines.

The other thing that happened that involves a continuing exposure to thimerosal is about the same time that we were getting the thimerosal out of the infant-containing vaccines, the recommendation was made to give thimerosal-containing flu vaccine to pregnant women, and in my community I order thimerosal-containing flu vaccine, and I typically run out by the end of October because 93 or so percent of the flu vaccine in this country still contains thimerosal.

So, the question becomes how do you factor in the potential vulnerability for thimerosal given in pregnancy at a time when some would argue the fetus might even be more vulnerable, and the fact that the recommendation was made that children receive

thimerosal-containing -- or flu vaccines, the vast majority of which is thimerosal-containing at six months, 12 months, and then every year thereafter.

So, one of the calculations that has been done is that even by taking away the thimerosal from the infant series if you have a situation in which the pregnant woman gets flu vaccine and the child gets flu vaccine, and it continues throughout early childhood, that your thimerosal load actually in the current system can be as much as 50 percent or so, the thimerosal that the kids got in the '90s. It's just time shifted and different distribution.

Q Doctor, you would agree that the number of autism cases has continued to increase since 2001?

A Yes.

Q And is it your testimony here today that that is because of the flu vaccine and maternal vaccinations?

A No. I think that there are many, many factors that as yet we need to look at.

Q Doctor, you're familiar with the Institute of Medicine, correct?

A Yes.

Q And you're aware that the IOM looked at the alleged link between thimerosal-containing vaccines

and autism?

A I am. I was there.

Q It first conducted an investigation in 2001, correct?

A Yes, I was not there for that one.

Q And the IOM looked at the issue again in 2004, and that's the one you were involved in?

A That's the one I attended.

Q You actually submitted a letter to the IOM and --

A I did, an impassioned letter.

Q And in 2004, the IOM concluded that the evidence favored rejection of a causal relationship between thimerosal-containing vaccines and autism, right?

A That's correct.

Q And you already mentioned that you're a member of the American Academy of Pediatrics, correct?

A That's correct.

Q And you're aware that the AAP has taken a position with respect to thimerosal-containing vaccines and autism?

A Yes.

Q And would you agree that the AAP's position is that no scientific data linked thimerosal use as a

preservative in vaccines with any pediatric neurological disorder?

A Yes.

Q Doctor, you're familiar with the World Health Organization?

A Yes.

Q And you're aware that the WHO has issued a position statement on the alleged link between TCVs and autism?

A Yes.

Q And am I correct that the WHO has recently stated, "In the latest review by the committee at its meeting of 6 to 7 June, 2006, the conclusion previously reached was reaffirmed that there is no evidence of toxicity in infants, children or adults exposed to thimerosal in vaccines." Is that correct?

A I was not aware that they had met in '06, but I certainly take your word for that.

Q Okay. And you are familiar with the CDC?

A Yes.

Q And you are aware that the CDC has taken a position on this issue?

A Yes.

Q And you would agree that the CDC supports the IOM's conclusion?

A That's correct.

Q Are you aware or familiar with the Public Health Agency of Canada?

A No.

Q Okay. Are you aware that the Canadian National Advisory Committee on Immunization has taken a position on the issue of thimerosal-containing vaccines and autism?

A I wouldn't be surprised if they have.

Q Okay. Would you be surprised to know that they concluded that the weight of the evidence to date clearly refutes an association between thimerosal and neurodevelopmental disorders?

A I am surprised that they used the word "clearly refutes".

Q But you have no reason to dispute that that was their conclusion?

A No.

Q Are you familiar with the European Agency for the Evaluation of Medicinal Products?

A Not very, but I know of them.

Q Okay. And were you aware that that agency has taken a position on this issue?

A No, but I would not be surprised.

Q All right. Would it surprise you to know

that they concluded that the latest epidemiological studies show no association between the vaccinations of TCVs and specific neurodevelopmental disorders?

A Not at all because I don't think that we will ever be able to show an association if we rely upon epidemiology. It is our contention that this affects some as yet undetermined subset of children, and that they will not show up in epidemiology as the studies have been done to this point.

Q And you would acknowledge that there have been numerous epidemiological studies in the United States, Canada, and Europe that have looked at this issue?

A I will, and I will have to tell you that even as a pediatrician I perceived flaws in a number of those epidemiologic studies. I do not think that they have addressed the relevant question here.

Q And you're not an epidemiologist, correct?

A Obviously not.

Q Doctor, as recently as this year the American Academy of Pediatrics and the American Academy of Family Physicians reaffirmed their position on this issue in response to a television show that aired, were you aware of that?

A I heard of it. I actually don't think I've

read the document.

Q All right. And the American Academy of Family Physicians issued a statement saying, "Scientific data overwhelmingly show that there is no evidence between vaccines and autism," is that right?

A That's correct.

Q And the American Academy of Pediatrics issued a statement indicating that "No scientific link exists between vaccines and autism," is that correct?

A Yes, but please blow that up again.

The AAP said, "No mercury is used as a preservative in routinely offered childhood vaccines." That has led many of my patients to make the assumption that since flu vaccine is now included on the schedule of recommended vaccines, that their children's flu vaccine does not contain thimerosal.

Now, whatever the science shakes down on this in the years to come, whether this hypothesis is refuted or affirmed, I am very concerned that the American Academy of Pediatrics would make that statement because families are taking their kids in for flu shots thinking that they are avoiding thimerosal, and it is often not the case, and I have expressed that concern to the president of the AAP -- I presume it was Rene Jenkins that wrote that letter.

Could you get it back up? I'm sorry, please -- that I would like for them to be very careful, yes, about their language.

Q Doctor, do you believe that your opinion that thimerosal can contribute to autism is generally accepted in the medical community?

A No, it is not generally accepted.

Q And would you agree that for most members of the medical community the case is closed on the alleged link between vaccines and autism?

A Sadly, I think that is the case.

MR. JOHNSON: Thank you. I have no further questions.

SPECIAL MASTER CAMPBELL-SMITH: Thank you, counsel.

It is now 1:15. I anticipate Petitioner's counsel has some redirect.

MR. POWERS: Yes, ma'am.

SPECIAL MASTER CAMPBELL-SMITH: Would you like to take a lunch break?

SPECIAL MASTER HASTINGS: Do you have any idea how long you're talking about?

SPECIAL MASTER CAMPBELL-SMITH: I was going to ask that.

MR. POWERS: I think it will be long enough

that we ought to take a lunch break. It's hard to put a time number on it, but it won't be the 15-20-minute redirect. So having a chance for a lunch break, and particularly for the witness, if there is going to be any re-cross. I think the witness, in particular, needs a full lunch break.

SPECIAL MASTER CAMPBELL-SMITH: Okay. I think an hour? An hour?

THE WITNESS: That would be great. Thank you.

THE WITNESS: I'm sorry. You weren't asking me.

SPECIAL MASTER CAMPBELL-SMITH: The witness agrees that she needs an hour. Let's return at 2:15.

SPECIAL MASTER HASTINGS: Before we break though, I just wanted to say one thing. I see Ms. King is on her way out the door. I understand that you need to fly out this afternoon, so I just wanted to thank you very much, Ms. King, for being here with us this week. We really appreciate you coming here. Thank you, again.

MS. KING: Thank you.

SPECIAL MASTER HASTINGS: We are adjourned for the afternoon, I guess.

SPECIAL MASTER CAMPBELL-SMITH: For lunch.

SPECIAL MASTER HASTINGS: For lunch.

(Laughter.)

(Whereupon, at 1:17 p.m., the hearing in the above-entitled matter was recessed, to reconvene at 2:15 p.m. this same day, Friday, June 16, 2008.)

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A F T E R N O O N S E S S I O N

(2:20 p.m.)

SPECIAL MASTER CAMPBELL-SMITH: Mr. Powers, are you ready to begin redirect?

MR. POWERS: Yes, I am, Special Master.

SPECIAL MASTER CAMPBELL-SMITH: Thank you.

Dr. Mumper, you remain under oath.

THE WITNESS: Thank you.

Whereupon,

ELIZABETH MUMPER

having been previously duly sworn, was recalled as a witness herein and was examined and testified further as follows:

REDIRECT EXAMINATION

BY MR. POWERS:

Q Good afternoon, Dr. Mumper.

A Good afternoon.

Q I see you're getting situated there. Are you all set?

A I think so.

Q Okay. Well, you recall yesterday afternoon there were a series of questions that Respondent's counsel asked you. Do remember starting that cross-examination yesterday?

A Yes.

Q You recall a line of questions about some of the research that you were conducting. Do you remember those questions?

A Yes.

Q And questions about hyperbaric therapy and lab reliability testing?

A Right.

Q Why is it that you were conducting those tests in the first place, or conducting that research in the first place?

A Our perception is that some of the research efforts that have been going on in the field of autism have not been directed as much as we would like toward potential treatments and therapies and assessment methods that would actually lead fairly soon to taking better care of these children.

Classically, the resources have been directed to a lot of work in classic genetics, and we wanted to look at the treatment strategies that we had developed with our collective clinical wisdom and make sure that they were safe, ultimately evaluate efficacy in a more rigorous fashion, and to refine our use of laboratory assessments.

It came to our attention that a number of parents were renting hyperbaric chambers and we wanted

to make sure that they were not posing a risk to the children. So, we wanted to look at the methylation biochemistry and the oxidative stress markers.

So, the initial study, which was just a pilot study, was intended not to be so much a rigorous assessment of efficacy as it was the first step, which would be to prove safety, so that's why we chose to look at the methylation biochemistries, and since we were doing the study we also looked at therapeutic response, but the primary issue there was safety.

With our lab split sample study, our primary concern is that we are in a situation where we have found value in using so-called functional laboratory assessments. Many traditional laboratory assessments are targeted more toward the detection of disease as opposed to the detection of suboptimal function in the period of time leading to frank expression of disease.

So, we wanted, as best we knew, as best we could, to know that those functional assessments we were utilizing are replicable or to sort out which laboratories would not have reliability on split sample testing. So, ARI ended up budgeting over $30,000 of a pretty meager budget to essentially do the lab split sample study, and that will ultimately become known. It's certainly possible that some of

the labs we have relied on are stronger in some areas than others, and that's why we need to sort those issues out.

Q Excuse me. Dr. Mumper, would it be your intent then with hyperbaric study and the lab reliability study, if there are findings that you find significant would you then be integrating those into the Defeat Autism Now recommendations and protocols?

A Yes. We shared some preliminary data on split samples for allergy testing at the think tank that we held in April, and our plan is to also review at the think tank the split sample reliability for the other types of tests that we anticipate having statistically analyzed by then.

Q So these safety and efficacy studies aren't for your sort of proprietary use, but they are actually designed to be pushed out into the treatment community, is that correct?

A Oh, exactly. That's entirely the purpose, yes.

Q Are there any other research projects you're doing with that same general goal, that is, looking at the safety and the efficacy of various treatments that you employ?

A Well, I've actually just hired a clinical

research director since I am primarily a clinician and a medical educator. I want to continue to participate in clinical research studies, but I wanted to have someone who could help me design them and carry out the protocols, so I hired someone that has had training in that arena so that when we do clinical trials and research at the Rimland Center that will not be under my -- limited by my relative lack of expertise, but we can actually continue to do higher quality and more complicated studies.

One of the things that I'm very eager to do is what's called single subject, multiple baseline studies, and the reason that I'm so interested in this is that our anecdotal impression is that what we can do to recover Johnnie might be totally different from what we can do to recover Suzie. And so we need to figure out what is working for specific kids, and what is it about those kids that makes that strategy of treatment more effective.

So, when you group a bunch of kids together and do the classic placebo controlled double-blind study, that works great for evaluating a drug in patients that have the same symptom. It doesn't work as well for a situation in which you've got complex multi-system involvement, and so a way of doing that

is to adapt some research from the behavioral world where they look at a single subject and document behaviors over time, and then do interventions and look at the change in the subject.

Ted Carr is one of the people who has published a fair amount using this type of research protocol, and he's actually contacted me. We have a phone conference call next week because he would like to do some of that type of research in our clinic, utilizing not just behavioral measures but also wedding it to biomarkers biomedically.

So, I hope that my colleagues at ARI and I can lead the way in looking at well-respected research models that we can apply to individuals as opposed to always thinking about research having to be done in broader groups.

So another thing that we plan to do, I mentioned that we've met with the American Academy of Pediatrics. My job that I'm a little behind on due to this trial was to provide the AAP with our sort of wish list for research projects. We want to go after what we think of as the low hanging fruit. The studies that we can do that can be completed relatively quickly so that the results can be utilized to help children.

So, we are going to try to encourage research on treatment protocols and especially on identifying biomarkers that help us subtype each of these types of autism, because our perspective is that the evolving model that we have regards a combination of genetic and environmental components, and perhaps lots of autisms, with an "s", this is language that's actually been adopted by the MIND Institute, and what it implies is that there may be a number of different ways that children are affected in ways that they ultimately exhibit autistic-type behaviors. So there may be a number of pathways that are affected to give us this constellation of behavioral symptoms.

Q And, Dr. Mumper, is it typical or atypical that a general practice pediatrician in a small town like Lynchburg would bother to hire a clinical research director?

A I think that would be very atypical because it's a severely financially unwise thing to do.

Q If it's a financially unwise thing to do, then why are you doing it?

A Because I think it's the right thing to do. I've thought about that question a lot, and I think one thing that influenced me on this is that when I was a child my house had a community swimming pool

just behind our back yard, and at that time, which was in the sixties, they would not allow people of color to come and swim in the swimming pool.

So my father and my family took the stand that we would then not participate, and so for all the summers of my childhood I would play in the back yard hearing the splashing from the swimming pool, and it in a way that was reenforcing over and over told me that my parents thought it was important that I stood up for what I thought was right.

So, there have been significant personal and professional disadvantages to speaking up in such a lone voice when there are clearly so many agencies that don't have the same concerns as I do, but I do think it's important to do what you think is right even if there are some sacrifices involved.

Q What do you do to keep up with the science? I mean, do you follow the literature? How do you keep engaged with the science that's happening out there?

A Yeah.

Q Because you were asked about some of this, about what articles you read and what articles you rely on, and how you form your opinions. Can you describe to the Special Masters what you do to keep apace of that?

A It's quite a challenge because I think I mentioned I typically go to my office at 7:30 and leave about six, and most of that time is devoted to clinical practice. But in my job at ARI, one of my roles is to decide what literature we teach to the clinicians that we're teaching in the clinician seminars. So in order to make that distinction, I feel that it's necessary to try to keep up with the literature.

So, I have a couple of list serves that send me articles based about autism or related subjects, and I, you know, certainly can't read all of those articles, but I try to read as much as I possibly can.

Another impetus I have is that I'm the director for the clinical part of the think tank for the Autism Research Institute, so the scientists and clinicians have to submit their abstracts to me, and I'm the one that makes the judgment about what science they can present and what doesn't meet, you know, our expectations for presentation, so that helps me keep abreast of the science.

The other thing is that I get invited to speak in this country and overseas about these topics, and I like to be able to answer the questions when they're asked, and so I try to have a good grasp of

the scientific literature in addition to my clinical experience so that I can field those questions from the audience.

Q Now, in this sort of monitoring and ongoing review of the literature, is it your experience that in the field of autism there is significant new science that comes out in an evolving way?

A I have seen it explode exponentially, and I think that can be actually objectively validated by looking at the number of autism articles, and there is a big curve up over the last decade or so.

The thing I also really like is that since I am the medical director for ARI, I frequently will get prepublication confidential drafts of upcoming science with requests from the authors to make suggestions, I mean, obviously from my perspective as a clinician, on the way that they have written the papers or any aspects of the paper that I would critique or make suggestions on.

Q Now, I want to focus a little bit on some of the questions that you were asked about the content of your reports, and again focusing first on yesterday afternoon.

A Okay.

Q Do you recall questions about the article

that was published in Medical Hypotheses?

A Yes, I do.

Q And do you recall questions that suggested you were relying on that article to bolster an opinion that mercury as contained in vaccines was neurotoxic or that it was somehow being cited in support of your ultimate opinion and causation? Do you remember that line of questions?

A Yeah, and I actually don't think I appreciated at the time perhaps the intent of the question. I was thinking that the intent of the question was to point out that it wasn't in a referee peer-reviewed journal and that it had been submitted by people who were not scientists.

But the way that I cited the article, and I think that I tried to make it very clear in my report was that it was to point to it as an example of mercury having myriad toxicities, and that it raised the issue of biochemical individuality in the patients because it specifically talks about things like route of excretion and individual variability and those types of issues.

So, I certainly did not ever in any aspect of my testimony here before this Court mean to imply that I was equating autism with direct mercury

toxicity. I would certainly agree that that is not the case. I would be embarrassed to put that hypothesis forward.

Our concerns is a much more complicated one having to do with a much more chronic condition resulting after low-dose exposures. Having said that, if you look at the actual sheet that's printed about thimerosal, there are side effects listed that are consistent with what some of these children have. They list anorexia, for example, as a known effect of thimerosal. They list nausea and vomiting. They list fetal loss, and so I think that one thing that has been lost in this arena is the idea that we expect all drugs to have side effects, and we would expect that in vaccines there are going to be some children that have problems with them, hence the reason for this Court.

Q But certainly not cited for the proposition that autism, as you said, is the functional equivalent of acute mercury toxicity?

A No, that is not my thesis at all.

Q Now, another article that you had in your report that was discussed yesterday the first author is Stajich, and I never know if I'm pronouncing that right.

A I can't do it either.

Q Okay. So we will trade pronunciation skills and math skills, but Scott, if you could put just the --

MR. MATANOSKI: Actually we don't believe that we discussed that article. That's what I was informed.

MR. JOHNSON: I didn't ask any questions about that article.

BY MR. POWERS:

Q Well, put it this way, the article was cited in your report.

A Right.

Q And you were asked questions about why it made sense to rely on articles in your report.

A Right.

Q And why did you rely on the Stajich article in your report?

A Because it showed an outlier. They looked at a small number of children, I believe it was about 20, and in that one of the children had a level of 23.6 micrograms per liter, I think, and my point in including it in my report was that, even in those 20 kids, one of them was high enough to meet the criteria for acute mercury toxicity.

That does not mean that I think that the child had acute mercury toxicity. It was just to say that there is a wide variation in how individual children response.

So if in that group of 20 we see one child who is that high, what would happen if we looked at bigger populations and consider the possibility that there will be some children that are outliers and for whatever reason develop higher blood levels, and therefore potentially higher brain levels.

I also want to make it clear that I'm not concerned about what's in the blood. I'm concerned about what ultimately goes to the brain and other target tissues.

Q Now you were asked questions about the Berman article. Do you recall those questions?

A Well, I think the way, if I'm remembering right, that that happened was that they asked me about an article that was reported to have refuted the Hornig article.

Q That's correct.

A Is that the one that's I'm remembering?

Q That's correct.

A So, I had not read that article, but I did read it early this morning.

Q And having reviewed that article and then thinking back to the questions where that article was being cited to refute Mattie Horning's article that you had cited in your report --

A Right.

Q -- what do you think is significant in your review relevant to that line of questioning that you heard yesterday?

A Well, I actually found it very interesting to read the article because that particular article was looking at acute toxicity, and in fact they were not doing any kind of measures of neuroinflammatory markers. They didn't speciate the mercury. They were looking at mice, which is a good model and it's what Mattie used, but they were not using probably the best model, which would be primates, and they also didn't say anything about inorganic mercury in the brain.

What they did look at was, you know, behaviors of the animals, and so whereas I acknowledge that it didn't find the same thing that Dr. Hornig did, we don't expect that in science, and it really doesn't impact in any way on making my opinions any less strong.

Q Now, you mentioned the issue of mice or rodents generally is not a good surrogate for human

exposure.

A Right.

Q You were also asked questions specifically about Dr. Burbacher and Clarkson's 2005 infant monkey study. Do you remember that line of questions?

A I do.

Q Okay.

MR. POWERS: And Scott, if we could put the cover page of that article up, I would appreciate it, and just zoom in on the title. I just want to make it for the record so that everybody knows what we're talking about here.

BY MR. POWERS:

Q This the op cite at Petitioner's Exhibit 26, and we're looking at the cover page. Do you see that on the screen there, Dr. Mumper?

A Yes.

Q Your recollection of your cross-examination yesterday, this is the article that would have been referred to when they were talking about the infant monkey study, correct?

A Yes.

MR. POWERS: Okay. We can pull that down now, Scott. I just wanted to make sure that we're all talking about the same document.

BY MR. POWERS:

Q Now, that study, Dr. Mumper, involved primates, correct?

A Right.

Q What's the significance of primates in that study?

A Well, primates are an excellent model to look at what might happen to humans, and can be used experimentally, and primates are much, obviously closely related to us than mice and rodents.

I had the opportunity to hear Dr. Burbacher present this, perhaps for the first time it was presented publicly, I'm not sure, but it was at NIEHS in August of 2005, and we discussed at that symposium how it was very interesting the way that this was presented in the press because it was actually spun in the lay press as good news because ethyl mercury was shown to clear the blood very quickly, and what all of us who are clinicians and scientists took away from the paper was a much more dire kind of take home message, which was that it cleared from the blood but ethyl mercury went to the brain, and ultimately led to inorganic mercury that was in the glial cells.

Q And now the 2005 paper, did it say that the inorganic mercury actually ended up in the glial cells

or just that it was inorganic mercury that entered the brain?

A That perhaps it entered the brain. I'm sorry.

Q Now, the Burbacher paper that was the subject of questioning yesterday, you remember a bunch of questions about blood levels?

A Yes.

Q And there was a line of questions that involved the relative blood clearance levels in this 2005 paper --

A Right.

Q -- between methyl and ethyl, correct?

A Right.

Q Is the distinction between blood clearance rates between ethyl and methyl, is that why you thought this paper was important and included it in your report?

A No. No.

Q Why did you include it in your report?

A Because of the issue of the inorganic mercury being in the brain, and the fact that with ethyl mercury there was a significantly higher fraction from ethyl that got converted to inorganic mercury.

And the other thing that I remember getting chills up and down my spine about was listening to Tom present how when they looked at the half-life of the inorganic mercury, that they were projecting that it would be in terms of decades, and that because of the way the mercury went into the brain and was converted to inorganic, they thought that there was clear evidence for the possibility of accumulation of inorganic mercury over time as repetitive thimerosal-containing vaccines were administered.

They also made a point in their paper which I thought highly of because it agreed with my opinion that they found it very hard to understand how the Institute of Medicine could have concluded, in 2004, that no further science should be done on thimerosal since it was clear that we did not have good models for projecting about the pharmacokinetics of thimerosal.

So, one of the points in the paper that was discussed yesterday was that there are limitations to applying methyl mercury kinetics to thimerosal kinetics, but the concerning thing about that, when I heard him present this, is that it made me even more worried about ethyl mercury because of the very long half-life of the inorganic mercury and the relatively

higher fraction of ethyl mercury that was converted to inorganic versus methyl mercury, if that makes sense.

Q Now, is it also fair to say that the significance of the 2005 Burbacher/Clarkson study is related to other work that involves mercury speciation and neuroinflammation which is obviously a key part of your opinion in these cases?

A Yeah, and I'm actually distressed to find out over the years how long ago this information was known because there has been a lot published about mercury in the last several decades, and I think it was the mid-nineties that Clarkson did his work on methyl mercury. That was in adult monkeys, I believe.

Q And are you talking Dr. Clarkson or Dr. Burbacher and Charleston and Vahter?

A Oh, I'm sorry. Not Clarkson. Yes, you're right, Charleston, not Clarkson. I apologize.

Q And I raise that because other witnesses --

A Right.

Q We've got so many monkey studies going, we've got to keep them straight, and some of the authors are shared.

A Right.

Q If I could just use the shorthand, we're talking about the adult monkey studies in the mid-

nineties?

A Yes.

Q Okay.

A Yes.

Q And you cite Dr. Charleston's work in your paper?

A That's correct.

Q In your report.

A And I realize that that paper was about methyl mercury and not ethyl mercury, but the reason that I thought it was important is because it demonstrates this idea of conversion to inorganic mercury and then the fact that there was neuroinflammation in the adult monkey brains and that we are so concerned about the neuroinflammation that we believe to be happening in these kids.

Does that make sense?

Q Yes. So would it be fair to say that the adult monkey studies establish that methyl mercury --

A Right.

Q -- gets inorganic mercury into the brain?

A Exactly.

Q And that inorganic mercury in the brain goes to glial cells?

A Yes.

Q And that having glial cells contain a lot of mercury is evidence of neuroinflammation?

A Right.

Q And then you then transition into the 2005 Burbacher/Clarkson paper where they talk about, just as was the case with methyl, you have ethyl that dumps inorganic mercury into the brain, is that correct?

A That is correct.

Q We talked about the 2005 Burbacher paper as if it was the whole study, but is it one paper or does it represent the entire study as far as you know?

A Yeah, I think that Tom has said that they have a whole series of ongoing studies on this. And when you read the paper very carefully, you will see that in the 2005 study that I heard him present at NIEHS they had just used half the brains.

So, my understanding is that there are further studies being conducted about the other half of those brains that I hope will be informative as to whether or not that replicates perhaps the adult findings with regard to neuroinflammation. We have to wait and see. That's part of the evolving science.

Q Okay. Now, you also recall a line of questionings about a deposition that you gave in the Blackwell case in Maryland. Do you remember those? I

think those questions might have been today rather than yesterday.

A Yes, that was today.

Q Okay. I'm going to direct your attention if we can pull it up to a page from that deposition transcript, and this is on the deposition it's page 158.

MR. POWERS: And, Scott, if you could under the question "By Ms. Elliott", if you could highlight the rest of the page there, please.

BY MR. POWERS:

Q Now, Dr. Mumper, during that deposition, which was in January 2007, you were asked what it meant to have a -- it says, "What does it mean to be a neurotoxin, in your mind?"

A And I said, "It means that the substance has either direct or indirect effects on some aspect of the nervous system, either directly on cells that are neurons, astrocytes, microglia, or whatever, or indirectly with regard to enzymes that it affects that in fact then affect the nervous tissue or in other ways interferes with functioning of neurologic capacity."

Q Okay. I'm going to interrupt you there. So would it be fair to say that neurotoxin, in your mind,

doesn't necessarily mean cell death? Is that a fair summary?

A That's exactly correct.

Q Because what you're talking about is functional toxicity explicitly.

A Exactly.

Q Correct?

A Yes.

Q And when you talk about neurotoxicity in your opinion in this case, both your testimonial opinion and in your report, are you also talking about functional toxicity primarily?

A I am talking functional primarily, yes.

Q The statement that astrocytes, microglia are involved in the neurotoxic process involving thimerosal-containing vaccines, what is that informed by? What was your basis then for saying that those particular cell types were involved?

A At that time I had already had the opportunity to speak with both Tom Burbacher and Diana Vargas about their works, so I was specifically thinking of their papers related to, for Dr. Burbacher, the monkey study, and for Dr. Vargas, the neuroinflammation neuroglial activation paper that was published at Hopkins.

Q And having been reviewing this information back in January of 2007, you then had that information to rely on when you considered Dr. Kinsbourne's report that you reviewed about a month ago, correct?

A Yes, and in addition, I would say that on a number of occasions since I first heard the Vargas paper and the Burbacher paper I've had the opportunity to teach that in my clinician training because we really have regarded those as very seminal papers in our concept of what we need to teach clinicians about taking care of these children.

Q There is a further question there that asks, and Scott, you will need to bump down to the next page in a moment. The question is at the very bottom and it says, "Are you offering an opinion that thimerosal is toxic to the immune system as well?"

So you see the question there and let's look at the answer. What is your answer to that question?

A "I do believe that to be true, so I'm giving that as an opinion, yes."

Q And the question was about the immune system being implicated here. Would your answer include the brain's innate immune system also?

A Yes, it certainly would. One of the things that we teach a lot about in our Defeat Autism Now

physician trainings is the importance of modulation of the immune system, both cell-mediated immunity and antibody immunity, but also the primitive innate sort of first response system.

So, we're really not talking in the neuroinflammatory model, we're specifically not talking about evidence of an adaptive response. We're talking about evidence of this early innate, more privative type of immune response, and that's best probably characterized by the microglia who act as macrophages to go in initially and try to mop up the toxins much like peripheral macrophages would.

Q Okay, and we can be done with that page.

So, Dr. Mumper, I now want to move on and talk about some of the questions that came later during the day today. Do you remember questions about how you diagnose autism? Do you remember that line of questioning?

A Yes.

Q And do you remember questions particularly about regressive autism?

A Yes.

Q In diagnosing autism spectrum disorders, are there domains of symptoms that you look at in making a diagnosis?

A Yes. We look in areas such as communication and stereotypic behaviors as well as social reciprocity.

Q And those would be the three primary domains or categories?

A That's correct.

Q Within the communication category, what would be included in there?

A There is speech and then there is language, and language has a broader implication about being able to communicate. Language doesn't necessary have to be speech. It could be gestures. It could be sign language. It could be using a picture system, those types of communication would also be involved.

Q Because, as I recall, and correct me if I'm wrong, the question seemed to focus on word counts --

A Yes.

Q -- at particular ages. Do you remember that line of questioning?

A Yes, and I was very resistant to giving typical word counts, if you will recall, because as I tried to make clear by talking about things like babbling and jargoning and gesturing, there are ways that children can communicate pragmatically without using words. For example, if you have a child who is

able to point to an object or tug on the mother and point to the juice in the refrigerator, that child is essentially using gesture to communicate pragmatically.

So, I think it is very realistic to make the argument that if you have a child who progresses to a stage where they are using pragmatic language in that way to get their needs met, and then they lose that ability, that's also losing language in the sense of pragmatic language even if it's not implying that they lose actual words.

So, any mother will tell you that their babies can communicate with them in ways that do not involve words, and I think it would be a significant disservice to the children whose cases will come before this Court if we are led down a path that falsely uses standardized word counts as the only way of assessing whether or not children are losing their ability to communicate.

Q And that's in just one domain. There is, again, a lot of focus on word count in just that one domain.

Following up on the questions about regressive autism and in these cases, are there social interaction skills that you observed in these two boys

that they had and then lost?

A And to say that I observed it in the boys, it would be by virtue of video tape as I have -- you know, as we've established, have not met the children. But I did see very age-appropriate expressions of social interaction in both of the boys early on, and the most striking thing was that they both early on exhibited a lot of looking directly at the camera, and a lot of being responsive with their faces lighting up with different events going on around them.

In both of them, I appreciated a qualitative change in their demeanor. Again, at the time I kept myself blinded as to the supposed onset of the regression, but you could see the qualitative change in their faces as they looked past people instead of engaging with them, or they withdrew from social interaction whereas previously they had sought it out.

So, I think there is clear evidence of impairments in those domains and a loss of previous skills.

Q And you just used the word "qualitative assessment" when you were describing some of those skills. Flipping back again into the communications domain.

A Yes.

Q Would it be as fair within that domain to say that that is a qualitative assessment rather than a raw quantitative assessment based on word count?

A Exactly, because I'm very interested in how the kids used the words, and sometimes if you have very rote, repetitive use of words, which we call echolalia, that is actually a very bad sign, and one of the signs that's recognized for autistic behaviors.

So, we're not just looking at a list of words. A good example would be a child who could say "cat", "dog" and "pony" would not be exhibiting as high a level of function qualitatively as one who could, as Jordan did, point to the cat on the wallpaper border and say "meow", or point to the dog and say "ruff". That's an example of the kinds of qualitative aspects of language and communication that I really hope that we evaluate as we go forward.

Q And in these cases in particular then, would it be your testimony that in the communication realm both of these boys acquired communication skills and then lost them at a later point in life?

A That is my testimony.

Q Would it also be your testimony, to a reasonable degree of medical certainty, that they acquired behavioral skills and lost them later in

life?

A That is correct.

Q Would it be your further testimony, to a reasonable degree of medical certainty, that they had social reciprocity skills that they lost later in life?

A It would be my testimony, yes.

Q Would it also be your testimony that combining all of those three discrete opinions that they were clear cases of regressive autism?

A That is my best medical opinion, yes.

Q Now, I want to talk also about progress over time now. If a child is one year old -- this is a little bit of a hypothetical. Say a child is one old.

A Okay.

Q And maybe is at the low end of the normal number of words, so that even if we're looking at a quantitative analysis there are three ways that child could go in the future. They could ether catch up to the norm.

A Yes.

Q Actually, there are four ways. Make progress but below the norm.

A Right.

Q Plateau or lose what they have, correct?

A Right, or did we talk about even doing better than the normal? That's number five.

Q You know, being gifted would be wonderful, so let's include that as the fifth.

A Okay.

Q So beginning at that starting off point, there are five possible outcomes.

A Right.

Q And how many of those outcomes would you describe that child's progress as symptoms of regression?

A Now I've totally lost you on the last part of the question.

Q Yes. Put it this way. Only one of those outcomes would represent regressive autism, correct, that's that drop off?

A Oh, yes. I'm sorry. Yes.

Q Okay.

A I am talking about dropping off the -- actually, I'm talking about actually losing words that they had, so that is a clear dropping off, yes.

Q Right. There were questions about single-dose versus multi-dose vials this afternoon. Do you remember those questions?

A Yes.

Q And the question was posed to you as a fact that single-dose vials do not contain thimerosal. Do you recall that statement?

A I do recall that implication.

Q Do you believe that statement to be true?

A No.

Q Why don't you think it's true?

A Because there were no requirements that all single-dose vials be thimerosal free. One of the advantages of having single-dose vials is that you typically are able to get by with less preservatives, but to my knowledge, there was no mandate in that regard.

Q In fact, the only mandate is that multi-dose vials must have a preservative, correct?

A That's correct.

Q And single-dose vials could have a preservative and it was not prohibited, correct?

A To the best of my knowledge, that is correct.

Q There was also a series of questions about other environmental exposures that might contribute to the emergence of autism, particularly regressive autism. Given other exposures that may be there, and let's not assume whether they are or not, but if they

are there what do you believe, if any, about the potential contribution of thimerosal-containing vaccines in the presence of those other possible exposures, in general?

A In general, I am very concerned about the concept of synergistic toxicities. This is the concept where any given substance has the potential to be more toxic if it's given in combination with other substances known to be toxic.

So, the classic example is when you look at the LD-50, for example, the lethal dose that would kill 50 percent of whatever you're studying for a single toxin and then another toxin, when they get those two things together, it just doesn't double their risk of dying. It frequently increases it by many orders of magnitude.

So, one of my concerns, since we've polluted our planet so much, is that children who might otherwise be exposed to lead toxicity or perhaps coal-burning power plants, or live next door to a agricultural farm that uses pesticides, and also gets thimerosal-containing vaccines, that that child would be subject to the possibility of synergistic toxicities. That's one of the reasons that I find it so difficult for any given child to come up with a

dose of ethyl mercury given by TCVs that is going to be safe for every kid because the synergistic toxicities are going to vary, depending upon whether the child is living in a lead-infested ghetto in the inner city, or whether it's the child of a farmer who is using organic pesticides, those types of issues.

Q And generally with those kind of exposures information is not available, including how much got into --

A Right.

Q -- a person's body, correct?

A Right.

Q When it got in there. Exactly what the chemical formulation was you often don't know that.

A Right.

Q You don't know what dose entered the body.

A Right.

Q You don't know exactly where in the body the dose might have gone.

A Right.

Q Now, with thimerosal-containing vaccines, there is better information on the exposure, isn't there?

A Yes.

Q And where does that information come from?

A From the label on the vial or on the box.
Q So you know what the chemical of interest is.
A Right.
Q You know how much was there.
A Right.
Q You know the dose.
A Yeah.
Q You know when it was administered.
A Yeah.
Q You know the timing of the symptoms after it was administered, correct, just in terms of somebody who then develops symptoms.
A Yes.
Q If they got the shot in a day and the symptoms later, you can tell if there whether there's a gap.
A Yes. Yes.
Q And based on the peer-reviewed scientific literature that's been discussed, you know something about the specific pharmacokinetics of the compound?
A That's correct.
Q Knowing all of that as opposed to other exposures, does that make it more likely, in your opinion, that thimerosal-containing vaccines can be

part of a differential diagnosis in the etiology of regressive autism?

A Yes. That makes it quite clear that it should be in my differential diagnosis.

Q And that is also in review of Dr. Aposhian and Dr. Deth and Dr. Kinsbourne's opinions, correct?

A Yes. I very much rely on them to inform me about matters of toxicology, neuroinflammation. I have to be able to rely on colleagues that I trust to educate me in those areas.

Q And if we think of the mechanism of injury, there were some questions about exposures and exposure levels. The exposure that one gets through the administration intra of a TCV --

A Right.

Q -- in terms of the mechanism of injury is that ultimately the exposure that's of interest to you and that informs your opinion?

A Tell me that again, Tom.

Q Yes. The initial exposure and how much is --

A Oh, no, no. I'm not concerned about the initial exposure or the initial blood levels. I'm concerned about what happens potentially a great deal of time later as this process that we've discussed

where ethyl mercury has entered the brain, converts to inorganic mercury, and then causes potentially to varying degrees disruption of cellular biochemistry, neurotransmitter function, interference with crucial neuronal signaling, those types of functional jobs that those cells are tasked to do.

Q So just to explore the details then of your opinion in these cases, what you know in these cases is that both boys were exposed to thimerosal-containing vaccines, correct?

A Right.

Q And based on the review of the literature and reliance on other experts, you have reached a conclusion about where that thimerosal breakdown product ends up, correct?

A Correct.

Q And it triggers a process in the developing brain, correct?

A Correct.

Q What is that process that you believe, to a reasonable degree of scientific certainty, is involved here?

A Conversion to inorganic mercury and neuroinflammation.

Q And the neuroinflammation that's triggered,

is that consistent with the model describe by Dr. Deth again that you relied on in reaching your expert conclusions here?

A Completely, in my opinion, yes.

Q Is it your belief, to a reasonable degree of scientific certainty or probability, excuse me, that Dr. Kinsbourne's model of the overactivated brain resulting from neuroinflammation, do you believe that's what happened in Jordan King's case and in William Mead's case?

A I do believe that that's what happened to these children. I also believe that that Dr. Kinsbourne's model is consistent with what I see in my patients, and I think, in particular, one of the most egregious examples of that was the little boy I presented earlier today who had seizures for 18 years before he died several weeks ago.

His clinical presentation is precisely what I would expect to see in a child who had increased excitation and decreased inhibition. Throughout the time I took care of him, despite my best efforts, he was constantly in a state of anxiety. He had a lot of stimming behaviors, and as we mentioned, he had such severe seizures that we actually had to implant a vagus nerve stimulator in him to decrease the number

of seizures by 100 or so per day.

And so as I've gone on this journey everything that I've learned along the way has been evaluated in terms of my clinical experience, and I embrace those mechanisms, those scientific findings that I can in some logical way always tie back to what I'm seeing as a clinician because that is primarily what I am.

Q And if you saw evidence that you thought significant that would change your opinion in either Jordan King's case or William Mead's case, would you in fact change your opinion?

A I would have to do that because I think I have vowed to tell the truth and the whole truth, and I want to maintain my credibility because I'm going to be held accountable for my opinions.

I may turn out to be wrong. One of the things that believe me I've thought about is why am I up here when the WHO and the CDC and the AAP, and all those other organizations that were listed obviously disagree with my conclusions. So I may well be wrong, and would take it on the chin if that's the case, but so far as the science accumulates it seems like I am more right, and time will tell.

Q You were also asked questions about whether

you could identify one key piece of evidence or one most important piece of evidence, you were asked essentially the same question but in several different ways.

What do you think is the key evidence that you rely on in support of your opinion in each of these cases? I mean, is there one lab result or is it something else?

A You know, I really can't identify one lab result. I wish it were that easy, believe me. It would make my job day to day -- you know, I could get home a lot earlier, but this is a tedious process of using a combination of clinical history, many labs together.

I perhaps should take a chance for those of you who are not clinicians to explain that we teach -- we, ARI, teach that the child is the best lab. We use objective measures because we're trying to put a story together, but we have to look at labs in context.

I am trying to think of a simple example, but in addition to being differential diagnoses for disease states or symptoms, there are also differential diagnoses for laboratory values.

So when you see an analyte that's high, you should intellectually think about what potential

things that could be causing that analyte to be elevated. And if the same child has another analyte over here that also has a differential diagnosis, you should think about what things could cause that analyte to be elevated and on and on.

So, as we look at our labs what we're trying to do is to look at the thing that seems likely as the differential cause of that elevation in relationship to the other analytes so that if you just have one thing that might suggest metabolic acidosis or in this case mercury toxicity, you know, that's on your list of differential.

But if you find other things that are consistent with that, then you move that up to the top of the list, and it's a dynamic process. It also is informed by the state of the child at the time, and I don't know how to make that really simple. I really wish I did.

Q And it sounds like certainly you can't do a ranking like your top 10 things that you would be looking for, is that correct? Just in general, if you're looking at all of the things you would consider in a differential, you don't have some neat little checklist?

A No. I have a checklist of, you know, labs

that we find to be informative in certain patients, and I don't order every lab in every patient because part of what happens in the history is to help you rule out the need for some labs based on the clinical story. But I don't have a top 10 list.

Q And using the Respondent's words that they used on cross-examination, key evidence, is it key evidence in both cases here that these boys, in your opinion, suffered a clear autistic regression?

A Yes.

Q Is that key evidence?

A Yes.

Q Is it key evidence in these cases that both of these boys received the full on-schedule TCV exposure?

A Yes, with the caveat that I think that there may be some children who by virtue of their state at the time of shots would not necessarily have to get the full complement in order to have a problem.

Q Understood, but I really want to focus on these two particular cases --

A Okay.

Q -- and not in the more global. So the question again would be, is it significant evidence to you that both Jordan and William received the full

complement of TCVs on the pediatric schedule?

A Yes, that is key.

Q Is it significant evidence to you that you have ruled out through the medical records and the videos and talking to the parents any family history of autism?

A Yes.

Q Is it significant to you that neither one of the siblings of Jordan or William exhibits any developmental problems, delays or symptoms?

A Yes.

Q Is it key evidence to you that these boys have been found to have no genetic aberrations that are typically associated with autism, autistic symptoms?

A Yes.

Q Is it also key evidence that there is no sign that either of these boys received exposures to other known causes of autism, such as terbutaline, valproic acid and other agents?

A Yes.

Q So all of that is key evidence to you?

A Right, and a lot of what you've just described is what I mean when I say evaluating the kids individually and taking the history.

Q And the history being an entire history that we've just described?

A Yes. Yes.

MR. POWERS: I have no further questions.

SPECIAL MASTER CAMPBELL-SMITH: Thank you. Re-cross?

RE-CROSS-EXAMINATION

BY MR. JOHNSON:

Q Doctor, you were asked some questions about why you do the research that you do, and I believe why you employ a research director, and you mentioned that you did so because you believe it is the right thing to do, is that correct?

A Yes, that is correct.

Q And remind me again, what is the name of your clinic?

A The Rimland Center.

Q Okay, and did you at one time have a clinic called Advocates For Children?

A Yes, I actually still have that. The Rimland Center now has three different arms to it. Advocates For Children is my typical pediatric practice where I see ear aches and sore throats and well babies.

Advocates For Families is the part of the

practice where I see the children with autism or a few kids that have ADHD or other behavioral problems that are more of a consulting basis, and those tend to be the patients that have traveled from far away.

Then the Rimland Center is kind of over those two, and that is the center that was established to be a mentoring center.

So, one of the things that we recognized at ARI was that we wanted to provide some on-site training for physicians, and since I had the background and medical education for 11 years previously, designing curricula at the residency and teaching other doctors, and because I really loved to do that, I decided to start the Rimland Center so that we could invite clinicians.

And I announced my intention to do it last spring. I bought a building in June. I renovated it from July 27th to September 17th, and we opened on September 17th.

Since then we have had a doctor from Italy bring four to six patients over several times, and spend several weeks mentoring with me. We've had a clinician from Australia come, and we've had several people from around the country, and I'm excited about the fact that I'm getting requests by classically

trained developmental pediatricians to come and see what we do, you know, perhaps out of curiosity, but the tendency, or the feedback that we've gotten so far is that when you first look at what we're doing it might --

Q Doctor, I'm sorry to interrupt.

A Sorry.

Q The question really was just do you have a clinic called Advocates For Children.

A Yes, I do.

Q Okay. Do you feel that you're an advocate for autistic children?

A Yes.

Q Because you administered thimerosal-containing vaccines for a number of years as a pediatrician, do you feel complicit in the epidemic of autism?

A Yes, I do.

Q And do you feel that you have a debt to repay?

A Yes, I do.

Q Doctor, you talked a little bit about your role as deciding what literature to teach and that you do a thorough review of the literature in order to determine what literature to teach to the people in

your clinic, is that correct?

A As thorough as I can given the limitations I explained.

Q Okay. And you had not seen the Jill James CPOX article that I referenced on my cross-examination, is that correct?

A Yes. Remind me when that came out.

Q I believe it was within the last year.

A I was thinking it was even newer than that, but no, I had not seen that.

Q And you had not seen the Berman article that tried to replicate the Hornig study, correct?

A That's correct.

Q Okay. And on the topic of the Berman article, the purpose of that study was not to look for neuroinflammation, is that correct?

A That's absolutely correct.

Q Okay. The purpose of that article was to try to replicate the Hornig study, correct?

A That's correct.

Q And the Berman study was not able to replicate the Hornig study, is that your understanding of the article?

A That is correct.

Q You were shown some testimony from the

Blackwell case in which you testified that you were offering the opinion that thimerosal is toxic to the immune system, and then you -- it wasn't in your testimony but you qualified here today that that would include the immune system and the brain. Do you remember that testimony?

A That's correct.

Q You are not an immunologist, correct?

A That's correct.

Q And you're not a neuroimmunologist, correct?

A That's even more correct.

Q And you're not a neurologist?

A That also is true.

Q And you're not a neuropathologist?

A That also is true.

Q You were asked some question about the issue of whether single-dose vials did or did not contain the thimerosal. Do you remember that testimony?

A Yes.

Q Are you aware that certain manufacturers included thimerosal in single-dose vials and certain manufacturers may not have?

A I delegate that task in my office actually to my nurses. They're instructed to order thimerosal-free vaccines. So I'm actually a very poor source of

information about which manufacturers have thimerosal in versus out versus trace amounts.

Q So that's an issue you just didn't look into for the purposes of forming your opinions in this case, is that accurate?

A I will say that when I saw the shot records, I did in my mind make perhaps an unwarranted assumption that it had come from multi-dose vials.

Q And in any event, the amount of thimerosal really isn't particularly important to you, is that correct? You believe that a single thimerosal-containing vaccine could contribute to autism, is that correct?

A I stated that as a hypothetical to illustrate the issue of individual variability, but I would not characterize it as saying that the amount is not important to me. For me, the less the better.

Q But even with less you would still in certain cases be willing to render the opinion that a single thimerosal-containing vaccine contributed to a child's autism?

A I don't know that I can support the idea that the ones who supposedly only have trace amounts, I just don't know that science. I just don't know scientifically where we can draw that line.

I partially base that opinion on children who did not -- who came to my clinic who had not received thimerosal in their initial series of vaccines' got flu vaccine, and then the parents reported an autistic regression that was temporally seemingly related to that, but that is way far from being well studied.

Q You were asked some questions about synergistic toxicities. Do you remember those questions?

A Yes.

Q And in your report, I believe, that the article that you cite is the Schubert article, and this is Petitioner's Master List No. 520. Does that article look familiar?

A Yes.

Q And am I correct that this article looked at the combined effects of certain metals and actually determined that in some cases there was a protective effect when certain metals were combined, is that correct?

A Absolutely.

Q And this paper did not look at the issue of neuroinflammation as the result of the combined toxic effects of these substances, correct?

A That's correct.

Another paper that I did not list was published by Boyd Haley that did look specifically at thimerosal and its combinations with testosterone, neomycin and aluminum, and perhaps I should have cited that as being more informative in hindsight.

Q But you did not cite it, correct?

A But I did not, right.

Q And then you were asked a number of questions about neuroinflammation and the adult money studies that Charleston did, and you were asked a series of questions about how those informed your opinion.

A Right.

Q Now, again, your belief that neuroinflammation is caused by inorganic mercury, that essentially came to you through Dr. Kinsbourne's report, is that correct?

A The first part of that was that the adult monkey study showed that, so I didn't see Dr. Kinsbourne's report until -- I don't know -- maybe two months ago or one month ago, and the time that I had the opportunity to talk to Dr. Burbacher was August 2005, I think. So no, it did not come to me after reading Dr. Kinsbourne's report.

Q However, the precise model that Dr. Kinsbourne is describing came to you through his report. That was the first time you had seen that model, is that correct?

A That's probably correct. We've talked a lot at ARI about the issues that he raises with regard to glutamate toxicity, increased excitation and inhibition, and the result of reactive oxygen species as detailed by both him and Dr. Deth. But the way that he put it together for his report, and so in the sense that you're calling it a model, I agree with that model because it's consistent with what I believe to be true in my study of those same issues.

Q We can call it a hypothesis and so that was the first time you had seen that hypothesis?

A As articulated by Dr. Kinsbourne, yes.

Q Right. And you were shown some testimony from the Blackwell case in which you were describing some effects that mercury might have in the brain, and you mentioned neurons and microglia and astrocytes. You didn't describe the hypothesis that Dr. Kinsbourne has put forth when you testified in Blackwell, is that correct?

A That is correct.

Q Do you know what happened in the Blackwell

case? Do you know how that case has been resolved?

A I know that what happened was is that the expert witnesses participated in something that I think was called a Frye hearing or maybe a Daubert hearing, and after that the Judge ruled that the case would not go forward; that he would not accept the expert testimony as we had put forth.

Q We being the plaintiffs?

A Yes.

MR. JOHNSON: Thank you. That's all I have.

MR. POWERS: We have nothing further. Rather than doing re-redirect, I don't think I have anything else for Dr. Mumper.

SPECIAL MASTER CAMPBELL-SMITH: Thank you. Any questions?

I think this is it, and thank you very much, Dr. Mumper.

THE WITNESS: Thank you.

SPECIAL MASTER CAMPBELL-SMITH: It appears that, Mr. Powers, Petitioner's case-in-chief, you're ready to rest, at least with your witness testimony here?

MR. POWERS: That's correct, Special Master. We're prepared to rest our case-in-chief with our witness testimony, understanding, obviously, there is

plenty of work to still do on the case moving forward potentially as far as July. However, we do rest our case-in-chief at this point.

MR. MATANOSKI: I just want to have clarification of that. The moving forward to July is to take testimony of Dr. Clarkson and Dr. Magos.

MR. POWERS: And a potential third test case.

MR. MATANOSKI: Oh, I understand. Fact-specific, your case-in-chief in fact-specific test case is --

MR. POWERS: And rebuttal.

MR. MATANOSKI: And rebuttal. I just want to clarify that you weren't talking about continuing your case-in-chief in July.

MR. POWERS: No, we are not --

MR. MATANOSKI: On general causation.

MR. POWERS: We are not continuing our case-in-chief on general causation into July. So I will just be very clear. In terms of moving forward, we mean that we have a third test case that will be identified and assigned to Special Master Vowell. We will present case-specific evidence in that third test case. We anticipate that rebuttal, since Respondent's case is going to be open at least until July on

general causation with Dr. Magos and Clarkson, that we will then have some rebuttal to do after that.

SPECIAL MASTER VOWELL: That week?

MR. POWERS: I sure hope it's that week, but I am just expecting, Special Master, in time, I'm just saying the sequence would be after Drs. Magos and Clarkson, which will be the conclusion of Respondent's --

SPECIAL MASTER VOWELL: Let me make sure. I understand what you're proposing. You're proposing that third week -- that week in July to have rebuttal evidence for Drs. Magos and Clarkson, or rebuttal evidence in general?

MR. POWERS: Precisely. We are proposing rebuttal evidence on general causation after the conclusion of their case. If there is any case-specific for Jordan King and William Mead, we would obviously have to put that on before we are done next week.

MR. MATANOSKI: That's not my understanding of how this was going to transpire. My understanding was the last two days of the third week were for rebuttal for the case-in-chief. The record was open concerning the testimony of two toxicologists, Dr. Clarkson and Magos, and we understood that if rebuttal

testimony was going to come in as to those two witnesses, it would come in thereafter.

SPECIAL MASTER VOWELL: That was my understanding as well.

MR. POWERS: The big concern there is with those two witnesses, given the subject of their expert report, we might then have to bring all of our general causation witnesses back. So if we have general causation witnesses coming back say late in the third week of this proceeding, and then have to bring them back again, in terms of efficiency and cost I don't know if that's the best approach.

MR. MATANOSKI: To testify as to what? If they are not a toxicologist, they would be unqualified to testify as to the matters that Dr. Clarkson and Dr. Magos will be testifying to.

If Dr. Clarkson and Dr. Magos testify outside of their area of expertise, it obviously is subject to being objected to by opposing counsel. So if they are testifying within their areas of expertise, then the rebuttal should be limited to that area of expertise.

SPECIAL MASTER CAMPBELL-SMITH: It is difficult to understand, Mr. Powers, how you couldn't introduce rebuttal testimony to what we hear in these

three weeks at the conclusion of these three weeks for these witnesses.

MR. POWERS: Yes, I think the issue is that the idea that we would have to have -- I hate to have two people come back because what Dr. Clarkson and Dr. Magos actually talks about isn't purely the toxicology. I think Dr. Aposhian would be involved with both of those experts, but I think Dr. Kinsbourne would be too.

So again, the Petitioners just don't -- I mean, the Clarkson body of work and the neuroinflammation issues raised in his body of work are issues that clearly are central to Dr. Kinsbourne's opinion and testimony. So having potentially Dr. Aposhian and Dr. Kinsbourne come back next week, then, you know, we're closed on general causation, but then we hear something in Drs. Magos and Clarkson, and have to bring them both back again.

SPECIAL MASTER VOWELL: That's an if. It's probable that you may want to put on more rebuttal, is that what you're telling us.

MR. POWERS: Oh, you mean even on this round? Yes, it's probable.

SPECIAL MASTER VOWELL: Exactly.

MR. POWERS: It is probable that we will

want to put on rebuttal.

SPECIAL MASTER VOWELL: And it is possible that you will have rebuttal have Dr. Clarkson and Dr. Magos testifies?

MR. POWERS: That is true, and then the issue again is just hearing the entire case, in order to offer rebuttal and offer rebuttal that's coherent, the need to hear the full case.

MR. MATANOSKI: I have to object. Not only is this new as far as the procedure, not within the understanding obviously of Respondent, and I believe from what I'm hearing now of the Court, but we are talking about toxicology, and I understand, and I'm in agreement that if Dr. Aposhian wants to come back and talk about toxicology, that's fine, and that would be fine rebuttal.

I'm understanding from some comments now that perhaps Dr. Kinsbourne would come back and talk about what? Toxicology? Because my whole line of questioning whenever, whenever I asked Dr. Kinsbourne about a matter that was toxicological, he said, "Outside my area of expertise. I defer to the toxicologists," which would be Dr. Aposhian.

I don't understand why there would be rebuttal to Drs. Magos and Clarkson from a neurologist

who said, "I have to defer to a toxicologist on toxicology matters." If Dr. Clarkson and Magos step outside their area of expertise, it's subject to objection.

MR. POWERS: And just a substantive issue. When one looks at these studies, one of them -- I mean, there are issues in the monkey studies, for example, particularly the adult monkey studies, that get into what regions of the brain certain toxicological events -- what regions of the brain, and whether that's significant or not, the types of cells that are involved. It's not pure toxicology. These papers really do deal with the intersection of pharmacokinetics and toxicology with brain function in terms of the inflammatory process, and where these processes occur, and we've heard lines of questions from Respondent directed to Dr. Kinsbourne that was apparently important enough to inquire what regions of the brain were affected.

MR. MATANOSKI: And that was because Dr. Kinsbourne seemed to be stepping outside of his area of expertise when he talks about monkey studies that have to do with the effect of mercury on the brains of monkeys. Nevertheless, and every time I ask him a question about that that was specific to toxicology,

he would defer.

This entire theory of neuroinflammation was sprung on Respondent three weeks before trial, and at this point for Petitioners to be asking for the opportunity to come back later and work this case up further because they don't have it ready for trial, they had ample opportunity if they want to explore this theory before now. They are trying to explore this theory through this trial. We've had nothing but new evidence for the first two days of trial. This has got to stop. This whole proceeding is just expanding and is just going to be endless at this point.

They had a clear deadline of when they had to file their expert reports. That came and went without hearing this theory. This theory was, again it came up after Respondent's reports came in. I think an assumption that can be made or an implication or inference that can come from that is that Petitioners put on one theory. They saw Respondent's response to it. They understood the problems with that theory, and there were many.

And then they went out, as you heard from Dr. Kinsbourne, and asked him for an opinion. After they had seen Respondent's case, they tried to come up

with a new theory because they knew that the theory they were presenting on was not sufficient to carry the burden, and then three weeks before trial, before trial we prepared for for six years, this is six years in coming, the Petitioners come up with this theory.

I think this theory has no merit, and I think you will see that. I would submit you already have seen that. However, there has to be an end to this record. There has to be an end to this proceeding. I know that this can come up in another case. We might see it again. I think it will be ended right now, this theory of neuroinflammation. I think there is nothing to it, and I believe that at the end of this trial you will believe that too.

However, the notion that because we have two toxicologists coming in, in July, that this is going to be an extension of time for the case-in-chief of the Petitioners has to be resisted. There is nothing that Drs. Clarkson and Magos can be talking about that Dr. Kinsbourne can address within an area of expertise, and vice-versa.

To the extent Dr. Clarkson and Dr. Magos venture out of their area of expertise, you are not going to be giving it any weight, obviously, and it's going to be objected to. I submit that when Dr.

Kinsbourne stepped outside his area of expertise and talked about toxicology, that should be given no weight too.

SPECIAL MASTER HASTINGS: Let me ask a particular question. Have you and counsel talked any further about the exact schedule for that week in July exactly when Dr. Magos and Dr. Clarkson are going to testify? We talked about generally the Thursday and Friday.

MR. MATANOSKI: No, sir.

SPECIAL MASTER HASTINGS: Nothing further?

MR. MATANOSKI: No, sir.

SPECIAL MASTER HASTINGS: So what exactly, I want you to be as specific as possible, what are you specifically asking us to do or rule, Mr. Matanoski?

MR. MATANOSKI: I'm asking you to stay on the same procedural schedule that we had, which is that in that time frame Drs. Clarkson and Magos will come in. Rebuttal to that toxicological evidence will come in thereafter.

SPECIAL MASTER HASTINGS: So you are asking us to limit any rebuttal that come in after Magos and Clarkson to only rebuttal of their testimony?

MR. MATANOSKI: That's correct.

SPECIAL MASTER HASTINGS: You're asking that

the Petitioners, if they have any rebuttal to the rest of the government's case, that they bring that in at the end of the third week of this trial?

MR. MATANOSKI: That's correct, sir.

SPECIAL MASTER HASTINGS: Did you want to respond?

MR. POWERS: Yes. The same position that we had taken before, which is that to do all the rebuttal at once and to do all the rebuttal at the end of Magos and Clarkson.

SPECIAL MASTER HASTINGS: So you're saying right now you're going to foreswear any rebuttal week after next. You don't want to do any then. You want to save it all until after Magos and Clarkson?

MR. POWERS: Again, we are not talking case-in-chief. As I said from the outset, case-in-chief, we are done. I have already said that we are done. I'm not proposing extending our case-in-chief except for the one individual test case. So it's not new evidence that we are proposing at any point in support of our case-in-chief. It's rebuttal, and rebuttal makes sense to have it come in after their entire case, just as Drs. Clarkson and Magos will be coming in now after the conclusion of our case so that we don't have to have the position where there is is --

bringing people back for split rebuttal.

MR. MATANOSKI: Your Honor, again, this is a change in the procedure, and it actually may have affected, because this late change in the procedure, whether we would even call those witnesses or not. We want to be at an end of this proceeding.

SPECIAL MASTER HASTINGS: That's a good point. I mean, almost a year ago we agreed on these three weeks, and both sides knew it, and we were certainly hoping everything would be done during these three weeks. It was the government that needed to extend beyond these three weeks.

MR. MATANOSKI: I understand.

SPECIAL MASTER HASTINGS: I know it wasn't your fault, and I don't recall that we ever specifically talked about this specific issue. I'm sure we are not going to rule on it this minute. I'm sure we'll be talking before we rule on what you're presenting to us now.

MR. POWERS: Nothing further. You asked what our position was and I'm hoping I --

MR. MATANOSKI: In fact, sir, one other thing that I should caveat as you rule on this or consider this, maybe this is a matter we will need to take up next week, whether Respondent's position about

whether the late filing, three weeks before trial, of an entirely new theory of the case may have been different had this been presented, this proposal by Petitioners' Steering Committee had been presented at the time, we may not have ever agreed to have this proceeding go on with this new theory thrust upon us three weeks before trial when we had been preparing for an entirely different case.

SPECIAL MASTER CAMPBELL-SMITH: I think what we'll do is take this matter under advisement, to revisit it. We obviously need to confer, and we will take this matter under advisement, and we will return and have conversations with counsel again next week. We can anticipate commencing on Monday with Respondent's case at 9 a.m., and I understand Respondent will keep us apprised if there is a schedule shift during one of the days next week so for those who are listening at home they can adjust their schedules accordingly, and that's possible midweek for Dr. Rust. Am I correct with that?

MR. MATANOSKI: That's correct, ma'am.

SPECIAL MASTER CAMPBELL-SMITH: Thank you.

MR. POWERS: And aside from a potential shift, not the day but the timing I understand on Dr. Rust's testimony Wednesday. At this point it's

Petitioner's understanding that there are no other plan changes in the order of witnesses and the appearance schedule that has been shared with us already.

MR. MATANOSKI: That's correct.

MR. POWERS: So that does leave, depending on what you all decide, there are still the two -- the Thursday and Friday, if they, (a) are still available, for spillover, but whatever is available those days, if you direct us to, would be available for rebuttal.

SPECIAL MASTER CAMPBELL-SMITH: Thank you. And before we conclude, I do want to thank William Mead's mom for being here with us today, the last parent in one of the two cases. We appreciate, again, as I expressed to William Mead's dad earlier, we appreciate very much your willingness to go public with this case for the same of the omnibus autism proceeding.

Dr. Mumper, you're excused. Thank you for being very patient there.

(Witness excused.)

SPECIAL MASTER CAMPBELL-SMITH: I think that with that we have concluded with a robust piece of business that we will address next week, and we are adjourned for the afternoon.

MR. MATANOSKI: Thank you.

(Whereupon, at 3:43 p.m., the hearing in the above-entitled matter was recessed, to reconvene at 9:00 a.m. on Monday, May 19, 2008.)

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REPORTER'S CERTIFICATE

DOCKET NO.: 03-584V, 03-215V

CASE TITLE: In Re: Claims for Vaccine Injuries

HEARING DATE: May 16, 2008

LOCATION: Washington, D.C.

I hereby certify that the proceedings and evidence are contained fully and accurately on the tapes and notes reported by me at the hearing in the above case before the United States Court of Federal Claims.

Date: May 16, 2008

Christina Chesley
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