

UNITED STATES  
COURT OF FEDERAL CLAIMS

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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. )

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GEORGE AND VICTORIA MEAD, )  
PARENTS OF WILLIAM P. MEAD, )  
A MINOR, )

Petitioners, )

v. )

Docket No. 03-215V

SECRETARY OF HEALTH AND )  
HUMAN SERVICES, )

Respondent. )

Pages: 4101 through 4374

Place: Washington, D.C.

Date: May 30, 2008

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

IN RE: CLAIMS FOR VACCINE )  
INJURIES RESULTING IN )  
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A SIMILAR NEURODEVELOPMENTAL )  
DISORDER, )  
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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 30, 2008

The parties met, pursuant to adjournment, at

9:03 a.m.

4102

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

<u>WITNESSES:</u>	<u>DIRECT</u>	<u>CROSS</u>	<u>REDIRECT</u>	<u>RECROSS</u>	<u>VOIR DIRE</u>
<u>For the Petitioners:</u>					
Dr. Marcel Kinsbourne (Recalled.)	4106	4143	--	--	--
Dr. Elizabeth Mumper (Recalled.)	4175	4244	--	--	--
<u>For the Respondent:</u>					
Dr. Eric Fombonne	4273	4303	4309	--	--
Dr. Jeff Johnson	4314	4326	--	--	--
Dr. Jeffrey Brent	4330	4348	--	--	--

E X H I B I T S

PETITIONERS'

<u>EXHIBITS:</u>	<u>IDENTIFIED</u>	<u>RECEIVED</u>	<u>DESCRIPTION</u>
12	4108	--	Marcel Kinsbourne settlement documents
13	4118	--	NIMH study on riluzole
14	4190	--	Three-slide component of Elizabeth Mumper
15	4213	--	Jordan King video
16	4213	--	William Mead video
17	4270	--	Letter

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P R O C E E D I N G S

(9:03 a.m.)

SPECIAL MASTER VOWELL: Good morning.

Please be seated. We are back on the record for another day of proceedings on the second theory for the omnibus autism proceeding. We are taking rebuttal testimony. Are there any matters that counsel would like to address on the record before we begin today?

MR. POWERS: No, Special Master, not for Petitioners.

SPECIAL MASTER VOWELL: Thank you.

MR. MATANOSKI: No, ma'am.

SPECIAL MASTER VOWELL: Thank you. Mr. Powers, to call your next rebuttal witness.

MR. POWERS: Yes. Thank you, Special Masters. The Petitioners, at this time, would like to call in rebuttal Dr. Marcel Kinsbourne.

SPECIAL MASTER VOWELL: Thank you. Good morning, Dr. Kinsbourne, please be seated. You will continue under the oath that you were administered and took earlier in the proceeding.

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1           Whereupon,

2                           MARCEL KINSBOURNE, M.D.

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

6                           DIRECT EXAMINATION RESUMED

7                           BY MR. POWERS:

8           Q     Good morning, Dr. Kinsbourne.

9           A     Good morning.

10          Q     And since we are making an audio record  
11          here, I'll reintroduce myself. I'm Tom Powers, and,  
12          as you know, I represent the Mead and King families,  
13          as well as Petitioners' Steering Committee.

14                   Now, Dr. Kinsbourne, you were called to  
15          testify during the first week of this hearing.  
16          Correct?

17          A     Yes, sir.

18          Q     And in the subsequent days of the hearing,  
19          after your appearance in that first week, other  
20          witnesses appeared that, based on your review of the  
21          record of the proceedings, addressed some of the  
22          specific points that you raised in your direct  
23          testimony in your report. Correct?

24          A     Yes.

25          Q     So, this morning, what we're going to do

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1 primarily is focus on the specific testimony that you  
2 now would like to respond to, and that is testimony  
3 that we heard from the government's side of the case  
4 in the days after your first appearance here.

5 Correct?

6 A Yes.

7 Q Now, before we go into that, there was a  
8 matter that you might recall from the cross-  
9 examination following your direct examination during  
10 the first week of this proceeding. Do you recall, in  
11 cross-examination, questions regarding your employment  
12 status at the University of Toronto some 31 years ago?

13 A I do.

14 Q Do you recall, in that line of questioning,  
15 you were asked whether you had been terminated from  
16 the university and discussion of the grounds of your  
17 termination? Do you recall that?

18 A I do.

19 Q What was your response, at that point, to  
20 the document that you saw, which was a Grievance  
21 Committee report?

22 A I didn't actually see the document, so I  
23 don't exactly know who sent it to whom. I don't think  
24 it was a formal report. But in terms of the issues  
25 involved, I pointed out that there had been some

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1 allegations made. I filed a grievance. The grievance  
2 prevailed, that all allegations and charges were  
3 withdrawn, and that the protective order was issued  
4 about the whole matter.

5 I have to say, in these 30-some years,  
6 nobody until now has violated that protective order.

7 Q And you indicated that there might be  
8 further information about this matter that might be  
9 available and that you were going to see if you could  
10 track that information down and find it. Did you, in  
11 fact, do that?

12 A I did. I looked in my file and found a copy  
13 of the settlement with the university, which I sent to  
14 you, and --

15 MR. POWERS: Let me interrupt you for just a  
16 second, Dr. Kinsbourne.

17 We're going to mark the settlement documents  
18 as Petitioners' Trial Exhibit 12, and I have given a  
19 copy to Respondent's counsel before we began a little  
20 while ago this morning. I'm going to provide copies  
21 to the Special Masters here.

22 (The documents referred to  
23 were marked for  
24 identification as  
25 Petitioners' Exhibit No. 12.)

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1 BY MR. POWERS:

2 Q Dr. Kinsbourne, on the screen in front of  
3 you, and on the table in front of you, you see a  
4 document. Could you just describe for the Special  
5 Masters that that document is?

6 A Yes. This is the outcome of the grievance  
7 proceedings, and this document was drawn up by the  
8 attorneys for the university and Mr. Jeffrey Sach, who  
9 represented my interests and who currently is, I  
10 believe, general counsel to the faculty at the  
11 university.

12 By the way, I did talk to Mr. Sach, and he  
13 will be available for any questions that the Court  
14 might have to follow up on this.

15 At any rate, this settlement made it clear  
16 that all charges were withdrawn and that I was not, in  
17 fact, terminated.

18 Q And, in fact, you had represented, under  
19 cross-examination, that you, in fact, resigned  
20 voluntarily and were given an opportunity to then seek  
21 another teaching position. Is that statement also  
22 reflected in this settlement?

23 A I'm not sure that it's in that document, but  
24 I've certainly got more papers to make that point,  
25 should the Court require.

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1 Q The document will speak for itself, but I  
2 just wanted to give you an opportunity to let the  
3 Special Masters know that, under cross-examination,  
4 when you referred to additional documentation and a  
5 settlement, that this, in fact, is what you were  
6 referring to.

7 A Yes, sir.

8 Q Okay. So we're going to move on from this  
9 and talk about some of the testimony that addressed  
10 both your expert report and your direct testimony.

11 Do you recall Dr. Rutter's, Sir Michael  
12 Rutter's, testimony?

13 A I do.

14 Q And in a portion of Dr. Rudder's testimony,  
15 he had critiques of your mechanistic model of  
16 neuroinflammation and overactivation. Do you recall  
17 some of those critiques?

18 A I do.

19 Q One theme of the critique seemed to be that  
20 your model and your analysis lacked scientific rigor.  
21 "Scientific rigor," I think, was a term used, a lack  
22 of scientific certainty.

23 So my question for you, Dr. Kinsbourne, is  
24 how you would respond to that critique and explain to  
25 the Special Masters even whether you were attempting

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1 to state an opinion to a degree of scientific  
2 certainty or not.

3 A Yes, sir. Dr. Rutter described himself  
4 accurately as a neuroscientist. He is, and I have a  
5 high regard for his work. Certainly, as I, in fact,  
6 made clear in my report, I was not presenting, as it  
7 were, a scientific discovery, which I could prove to  
8 be the case, and I did not think that that was my role  
9 in these proceedings to do.

10 What I'm presenting is a reasonable medical  
11 mechanism by which this could have happened, and Dr.  
12 Rutter really didn't address the actual purpose and  
13 role of my proposal. When neuroscientists use the  
14 word "speculation," what they are really saying is  
15 that, whether there is evidence or not, if one draws a  
16 conclusion before the evidence is complete --  
17 speculated. I, however, was not drawing a conclusion;  
18 I was offering a possible mechanism.

19 Q And that possible mechanism, as you  
20 described it; in your opinion, is that mechanism  
21 biologically plausible?

22 A It is biologically plausible, and it is  
23 grounded in contemporary scientific literature, as is  
24 reflected in my report.

25 Q And, in fact, there was a specific portion

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1 of the model that Dr. Rutter addressed, and he seemed  
2 to take issue with the glutamate-mediated,  
3 overactivation model that you described. Do you  
4 recall his testimony on that issue?

5 A Actually, not exactly in those terms. I  
6 don't think Dr. Rutter purports to be a neurologist or  
7 a neuroscientist. I think he is very careful to stay  
8 within his discipline, child psychiatry, and I'm not  
9 sure that he actually critiqued the neurobiological  
10 aspect.

11 What he took exception to was the more  
12 global interpretation of the hyperglutamanergic,  
13 hyper-arousal model as a viable model for autistic  
14 behavior.

15 Q And how would you respond, in general, to  
16 that critique of Dr. Rutter?

17 A Well, it seemed to me that he was critiquing  
18 something from his memory of many years ago, which  
19 perhaps has not survived. It certainly hasn't, in my  
20 memory, but he wasn't really talking about what I  
21 presented. He talked about some notion that autistic  
22 children are overly emotional or overly reactive,  
23 which is not, in those words, accurate. That's not at  
24 all what I'm presenting.

25 In fact, although the over-arousal model did

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1 have earlier origins, the first studies that presented  
2 it were EEG studies. They ran studies of children's  
3 emotional behavior. There was evidence presented that  
4 the brain of these children was overactive, as based  
5 on the EEG findings as were available and construed at  
6 that time.

7 Now, this overarousal model has survived,  
8 and it has significant support at this time, and, in  
9 my report, I reference the important document by  
10 Rubenstein and Merzenich, which adopts that model, and  
11 other articles which, in fact, give evidence of this  
12 overarousal in psychophysiological terms.

13 Q And, in addition to that, there is  
14 contemporary scientific evidence supporting the idea  
15 that the role of excess glutamate contributing to this  
16 overarousal, there is contemporary support in the  
17 scientific literature for that aspect of your model.  
18 Isn't that correct?

19 A Right. Again, as with the overarousal  
20 model, the hyperglutamanergic idea is not my idea. It  
21 was presented for -- I proposed it, and, again, I  
22 mention some of the origins in my report, and I could  
23 provide the Court with more documentation of that  
24 fact.

25 My role has been to consider the literature

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1 on glutamate excess in neuroinflammation, consider the  
2 literature on glutamate excess in neuroinflammation in  
3 autistic individuals, put it together with the  
4 evidence for overarousal in autistic children, and  
5 combine the neurobiology and the behavior in what I  
6 take to be a coherent fashion.

7 Q Now, Dr. Kinsbourne, let's talk specifically  
8 about some of the more contemporary scientific  
9 literature that supports a couple of the aspects that  
10 Dr. Rutter is criticizing.

11 First, let's talk about glutamate, and the  
12 first article we're going to refer to is Petitioners'  
13 master --

14 MR. MATANOSKI: I just want to clarify. I  
15 think the witness has already stated that Dr. Rutter  
16 was not criticizing the portion of his data that had  
17 to do with glutamanergic response.

18 MR. POWERS: Well, we're talking about the  
19 glutamanergic response as triggering the  
20 overactivation, and if we want to parse it out, I can  
21 pretend that he is addressing Dr. Rustferti.

22 BY MR. POWERS:

23 Q So, Dr. Kinsbourne, do you recall that Dr.  
24 Rust described a specific critique of you positing the  
25 idea that the glutamanergic response was not

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1 contributory to the appearance of autistic symptoms?

2 A Yes, sir.

3 Q Okay. Let's talk about some of the  
4 contemporary scientific literature that addresses that  
5 issue. This will be Petitioners' Master Reference  
6 List 570. I know the screen is hard to read, Dr.  
7 Kinsbourne, so I'm going to leave the stand here and  
8 give you a paper copy of this article.

9 A Thank you.

10 Q So, Dr. Kinsbourne, if you look at the  
11 document in front of you -- this is Reference List No.  
12 570 -- is this an article by Dr. Aschner that talks  
13 about glutamate and reactive oxygen species and methyl  
14 mercury neurotoxicity?

15 A Yes, sir.

16 Q So I would like to just briefly direct your  
17 attention to page 2 of the exhibit, and if you look at  
18 the right-hand column, about half-way down, there is a  
19 highlighted section, if we could focus on that.

20 A I could if it were highlighted.

21 Q Well, on the screen, it should be.

22 A I see, yes. Okay.

23 Q We're trying to coordinate highlighting  
24 multiple paper copies and the electronic, but on the  
25 electronic, you should see it there.

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1 A I was just reading a spy novel where the  
2 highlighting magically disappears.

3 Q What is the point of the highlighted section  
4 there? We don't need to read it aloud because the  
5 Special Masters, obviously, can read the article.  
6 What do you think the significance of that highlighted  
7 portion is?

8 A It encapsulates a major part of my proposal,  
9 and, indeed, this is one of the sources of my  
10 proposal.

11 Q And this is a 2007 article. Correct?

12 A Right. I'm saying, this is a recent -- of  
13 Dr. Aschner's vary distinguished research program, and  
14 I also refer to other articles from his group.

15 Q We're also going to take a look at  
16 Petitioners' Master Reference List No. 567, and, Dr.  
17 Kinsbourne, is that an article by Dr. Purcell and  
18 others that discusses post-mortem brain abnormalities  
19 of, again, the glutamate neurotransmitter system and  
20 autism?

21 A That's correct.

22 Q I would like to draw your attention to what  
23 is the exhibit page number 9, and if you look at the  
24 right-hand column, what I'm going to highlight for you  
25 is, in the first third of that top paragraph, there is

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1 a sentence that begins, "If the increase in GFAP," all  
2 the say down to Footnote 31. Again, the Special  
3 Masters have the study, and they can read it, but what  
4 is the significance of this particular discussion in  
5 Dr. Purcell's paper for your theory?

6 A I think the relevance is that GFAP is a  
7 protein which is released by astrocytes under stress,  
8 and the article patients out that there may be  
9 reactive gliosis. In other words, there may be a  
10 proliferation of astrocytes in response to that  
11 stress, and that proliferation may contribute to  
12 autism pathophysiology. In other words, it may  
13 contribute to the mechanism by which an individual  
14 becomes autistic.

15 Q And at the very bottom of that page, again,  
16 on the right-hand column, the sentence that begins,  
17 "Disrupted," and it will continue on to page 10 of the  
18 exhibit, so we'll give our folks to get the entire  
19 thing highlighted for you, going from one page to  
20 another.

21 A The first sentence, beginning with  
22 "Disrupted," encapsulates the point that I'm trying to  
23 make for mechanism, that disruptive glutamate  
24 transmission could account for a constellation of the  
25 cognitive deficits of autism. I would just add, in my

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1 case, cognitive and behavioral deficits in autism.

2 Q And if you continue on to the next page,  
3 then, it actually starts to talk about the symptoms,  
4 particularly, that are related to glutamate.

5 A Correct. These are relevant symptoms, which  
6 I do believe can be explained in terms of disruptive  
7 glutamate transmission, and, indeed, as the group  
8 points out, that people have taken this seriously, and  
9 are, in fact, currently, trying to determine whether  
10 drugs that block glutamate receptors might alleviate  
11 autistic symptoms, and, in fact, there are several  
12 ongoing studies using two agents that I could mention  
13 funded by the NIH and by a foundation which is, in  
14 fact, finding out whether glutamate receptor  
15 antagonists could help autistic children.

16 MR. POWERS: In fact, we have what we're  
17 going to mark as Petitioners' Trial Exhibit No. 13 a  
18 brief report of a clinical trial, I think, involving  
19 one of the drugs that you're talking about.

20 (The document referred to was  
21 marked for identification as  
22 Petitioners' Exhibit No. 13.)

23 BY MR. POWERS:

24 Q And, Dr. Kinsbourne, Exhibit 13 that you  
25 have in front of you and is now up on the screen;

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1 could you describe to the Special Masters what this  
2 document is?

3 A Yes. This is a study which has been funded  
4 by the NIMH, the Mental Health Institute, with the  
5 following rationale.

6 Riluzole is a glutamate blocker. It has  
7 been shown to be effective in childhood obsessive-  
8 compulsive disorder. Now, we're going to try to see  
9 the effects of riluzole glutamate blocking also, as  
10 well as in childhood OCD, in children with autism-  
11 spectrum disorders.

12 Now, an important point is that, as I  
13 discuss at length in my report, the neuroinflammation,  
14 which was discovered by the Vargas/Pardo group, is, in  
15 my opinion, more likely harmful to the brain than  
16 helpful, but some people have objected that actually  
17 it might be helpful or protective of the brain  
18 function.

19 If anybody seriously believed that, this  
20 study would never have been funded. Children would  
21 have been put at risk if it was a protective mechanism  
22 that was being blocked.

23 So I think that highly responsible  
24 scientists from Johns Hopkins and from the study group  
25 at NIMH have felt that it was appropriate to attempt

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1 to diminish glutamate transmission in autistic  
2 children.

3 Q And we're actually going to take a quick  
4 look at page 3 of this exhibit -- it's page 3 of 5 --  
5 and at the very top of that page, the first full  
6 sentence that begins, "Glutamate plays," we're just  
7 going to highlight that sentence. What this clinical  
8 trial description says is that glutamate plays a  
9 crucial role in the regulation of excitatory activity  
10 within this circuit and may be involved in the  
11 idiopathogenesis of OCD, which is obsessive-compulsive  
12 disorder.

13 Is this statement about glutamate's role in  
14 the regulation of excitatory activity; is that  
15 consistent with the central theme of your opinion in  
16 this case?

17 A That's correct, yes.

18 Q We can pull that down from the screen.

19 Briefly, back to Dr. Rutter and his  
20 description of your model of overactivation or  
21 overarousal as being somehow historically relevant,  
22 are you aware of contemporary scientific discussion of  
23 this very theory?

24 A Yes, indeed.

25 Q And would that include discussions by Dr.

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1 Casanova, who is one of the Respondent's witnesses who  
2 submitted an expert report but did not appear and  
3 testify?

4 A Correct.

5 Q So I'm going to put up on the screen  
6 Petitioners' Master Reference List No. 274, and if we  
7 could highlight just the title so that Dr. Kinsbourne  
8 can identify it for the Special Masters and for the  
9 record.

10 And, Dr. Kinsbourne, I do have a paper copy,  
11 so I'm providing it for you.

12 So, Dr. Kinsbourne, you have in front of you  
13 a Science Journal article called "Mini-column Nerve  
14 Pathology in Autism" by Dr. Casanova and others. Is  
15 that correct?

16 A Yes, it's an important document.

17 Q Let's go ahead and look at the very last  
18 page of text in that article, which would be, in terms  
19 of the exhibit -- I believe it's page 4 of the exhibit  
20 -- and I would like to highlight for you the last full  
21 paragraph in that article.

22 Now, in this highlighted section, is it fair  
23 to say that Dr. Casanova is discussing the arousal  
24 model in the brain as related to autism spectrum  
25 disorders? Is that the general thrust of this

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1 paragraph?

2 A It's quite specific, yes.

3 Q He goes on to say, in about the third  
4 sentence in this, that the arousal theory is of some  
5 interest because it is consistent with the reduction  
6 of inhibitory into neuronal activity.

7 So the arousal theory is certainly  
8 interesting enough to Dr. Casanova to discuss it in  
9 this article. Correct?

10 A Yes.

11 Q And his discussion is on the flip side of  
12 the coin of the glutamate homeostasis, which is the  
13 inhibitory process, GABA.

14 A Correct. Still addressing the excitation-  
15 inhibition balance.

16 Q And the excitation-inhibition balance, as  
17 you've already testified, is a core concept in your  
18 model that you've described?

19 A It is, and it is a core concept in brain  
20 functioning.

21 Q So it's a functional model, but is there  
22 also some possible implication in recent science that  
23 the excitation and neuroinflammation and glial  
24 activation might actually be causally related to brain  
25 pathology?

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1           A     A number of sources have raised that  
2     question.

3           Q     I'm going to show you what's been filed here  
4     as Petitioners' Master Reference List No. 104 coming  
5     over with a paper copy, and we'll get that on the  
6     screen.

7                     Now, Dr. Kinsbourne, this is an article by  
8     Dr. Courchesne and his group, and it's an article  
9     that's referred to quite often in these proceedings  
10    that's called "Autism at the Beginning." Is that what  
11    you see in front of you?

12          A     It is.

13          Q     I would like to turn, and, unfortunately, I  
14    don't have my exhibit pages marked, but it's page 590  
15    of your copy, Dr. Kinsbourne; 590 is the journal page  
16    number.

17                    SPECIAL MASTER VOWELL: Which, for the  
18    record, is page 14.

19                    MR. POWERS: I was just counting, and you're  
20    much quicker than I. I appreciate it. It's page 14  
21    of the exhibit.

22                    What I would like to do, there is a section  
23    highlighted there, but before even talking about that  
24    one, in the left-hand column, the last full paragraph,  
25    there is a sentence that begins, "Glial cells," and I

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1 would just like to highlight that first full sentence  
2 of the last paragraph on the left-hand column.

3 Now, this sounds that glial cells play key  
4 roles in brain organization during development, as  
5 well as in neuroinflammatory reactions. Correct?

6 A Yes.

7 Q So the bulk of your report describes the  
8 role of glial cells in neuroinflammatory reactions.  
9 Correct?

10 A Could.

11 Q So Dr. Courchesne is acknowledging that role  
12 of glial but also mentioning a little bit new, which  
13 is that it plays a role in actually organizing the  
14 brain. Correct?

15 A Yes. Actually, it isn't even new. I think  
16 it's part of what we know about neurodevelopment that  
17 glial cells actually provide the scaffolding by which  
18 neurons move to their appointed locations.

19 Q Now, you've heard testimony of  
20 neuropathologists, including Dr. Kemper, how have  
21 argued apparently that pathological abnormalities in  
22 the brain cause neuroinflammatory responses in some  
23 cases. They have hypothesized that. Correct?

24 A That's correct.

25 Q What they haven't discussed is the notion

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1 that glial disruption or glial dysfunction might  
2 itself be the cause of the underlying pathology. Do  
3 you recall them discussing that issue at all?

4 A No. That didn't come up.

5 Q Well, let's go ahead and look at the right-  
6 hand column and the first full paragraph, and let's  
7 highlight the first half of that paragraph, ending at  
8 the word "cerebellum."

9 Now, what Dr. Courchesne is talking about  
10 here is that excess glial production of excess glial  
11 activation actually has the potential to produce any  
12 or all of the previously described, microstructural  
13 findings. Correct?

14 A Yes.

15 Q So he is talking about glial disruption  
16 affecting the physical architecture of the developing  
17 brain.

18 A Correct. In a manner so as to generate the  
19 kind of abnormalities that, in fact, have been  
20 reported neuropathologically in brains of autistic  
21 individuals.

22 Q Including the minicolumn abnormalities that  
23 Dr. Casanova -- in fact, there is a specific  
24 discussion of frontal minicolumn abnormalities.  
25 Correct?

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1 A Correct.

2 Q And that would be in reference to Dr.  
3 Casanova's work with minicolumns.

4 A Yes, it would.

5 Q So would you characterize the current  
6 scientific literature as supporting the notion in your  
7 report and in your testimony that glial activation can  
8 cause neuroinflammation leading to the symptoms of  
9 autism, but also that glial overactivation can  
10 actually cause changes to the developing brain's  
11 structure?

12 A Yes. There is support for these  
13 propositions.

14 Q And the support is described in some of the  
15 articles that we just took a look at. Correct?

16 A Yes, sir.

17 Q Now, Dr. Rust also had some comments on your  
18 testimony, and one of the issues that you raise is  
19 that, in a couple of places, you misrepresented the  
20 cited articles. Do you remember some of his testimony  
21 on that?

22 A I do.

23 Q And one of those points was, in looking at  
24 articles by -- I think it's Dr. Friedman. Dr.  
25 Friedman is the lead author on one on the other with

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1 Dr. Petropolous, and he is the second author on the  
2 other, with Dr. Petropolous.

3 We're going to take a look at those, and, in  
4 particular, we're going to start off with Physician's  
5 Master Reference No. 320. This is the article where  
6 Dr. Petropolous is the first author, and Dr. Friedman  
7 is the second author.

8 Q So could you describe for the Special  
9 Masters and for the record what it is that we have on  
10 the screen here?

11 A This is a study of the brain of individuals  
12 with autism spectrum disorder by MRI, conductive  
13 resonance imaging, and it talks about a particular  
14 aspect of imaging which is called the "T-2 phase" of  
15 imaging.

16 Q And do you recall that Dr. Rust  
17 characterized your citation of this particular piece  
18 as inaccurate because his claim was that this article  
19 doesn't talk about directly neuroinflammation leading  
20 to the symptoms of autism.

21 A Could.

22 Q Now, you didn't cite it for that  
23 proposition, did you?

24 A No. I didn't cite it for that.

25 MR. POWERS: Let's go ahead and turn to page

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1 4 of the exhibit, and, actually, it starts on page 3.  
2 I'm sorry. The very last sentence on page 3, and then  
3 going through the first full paragraph on page 4, and  
4 it will take a moment to get that on the screen.  
5 We'll wait for that to happen so it's easier to work  
6 through this.

7 BY MR. POWERS:

8 Q It won't all fit there, so what we can do is  
9 describe, first off, the beginning of the sentence of  
10 interest, is that their findings in children with  
11 autism, and these were findings that came after the  
12 children were diagnosed, as they say, it may reflect  
13 brain mechanisms involving neuroinflammation which  
14 have been implicated in this disorder. Correct?

15 A Right. There are now interpreting their  
16 findings, yes.

17 Q And then, as it goes on to say, such  
18 processes are typically accompanied by edema. Do you  
19 see that?

20 A Yes, I do.

21 Q Now, I'm going to step out of Dr. Rust's  
22 critique for just a moment. If you recall, Dr. Kemper  
23 specifically said that you are incorrect in describing  
24 edema as a consequence of neuroinflammation. Do you  
25 remember him making that specific comment?

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1 A Yes, I do.

2 Q Now, this paper actually says that  
3 neuroinflammatory processes are typically accompanied  
4 by edema. Correct?

5 A Could.

6 Q So that support, in the scientific  
7 literature, for your contention that edema is a  
8 characteristic of neuroinflammation.

9 A It does.

10 MR. MATANOSKI: Your Honor, at this point, I  
11 would like to request of the Court, please let the  
12 witness answer some questions rather than counsel  
13 simply leading him through articles. This is supposed  
14 to be Dr. Kinsbourne's rebuttal, not Mr. Powers'.

15 BY MR. POWERS:

16 Q So, Dr. Kinsbourne, if you would look at  
17 that paragraph, what is the significance of this  
18 paragraph and what these articles are saying to your  
19 report?

20 A The significance is that the findings on MRI  
21 are consistent with ongoing inflammation, and they  
22 also themselves relate their findings to studies to  
23 which I refer in which microglial activity and  
24 cytokines have been found to be associated with  
25 autistic disorders.

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1 Q So let's turn to page 14 of your report, and  
2 I just want you to be able to show the Special Masters  
3 what statement in your report you cite to this article  
4 for support for. Again, it's on page 14, and it's the  
5 very first sentence at the top of the page.

6 A Correct.

7 Q And, again, on the page previous is a  
8 sentence that talks about another Friedman article,  
9 but the one we're talking about is the one that's  
10 highlighted.

11 A Yes. I made the point, briefly, that the  
12 Petropolous article did find evidence of  
13 neuroinflammation in the cerebral gray matter of these  
14 individuals.

15 Q So if that's what you cited, the article  
16 that we just discussed, in support of. Correct?

17 A Yes.

18 Q So it would be your contention that that's a  
19 very fair and accurate citation to the literature that  
20 we just described.

21 A Yes.

22 Q I want to talk a little bit about Dr.  
23 Kemper's testimony with you. Do you recall Dr. Kemper  
24 testifying -- I think it was during the second week of  
25 this hearing --

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1 A Yes.

2 Q Do you recall Dr. Kemper having, on his  
3 direct examination, specific criticisms of your  
4 "expert report" generally but specific components of  
5 your theory?

6 A Yes.

7 Q Now, one was a reference to page 13 of your  
8 expert report. We're going to put page 13 of your  
9 report up, and the first portion of the second full  
10 paragraph. I would like to highlight the first two  
11 sentences there that deal with circulation.

12 Do you recall listening to Dr. Kemper  
13 describe -- he believed that edema was not a  
14 characteristic of neuroinflammation.

15 A He did make that statement.

16 Q And we just discussed the citation in the  
17 scientific literature where you find support.

18 Correct?

19 A Right.

20 Q He did not take issue with activated  
21 microglia. Correct?

22 A Correct.

23 Q Now, he did take issue and say that there is  
24 no local invasion of immune cells. Do you remember  
25 that criticism?

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1 A Yes, I do.

2 Q Do you believe that, in the process of  
3 neuroinflammation, there can be a local inflammation  
4 of immune cells?

5 A Yes. That's documented in the literature.

6 Q Let's talk about where it might be  
7 documented in the literature.

8 We're going to be referring to Petitioners'  
9 Master Reference List No. 72. This is Dr. Pardo's  
10 article that's been much discussed.

11 So you have the article in front of you, Dr.  
12 Kinsbourne. I'm going to draw your attention to page  
13 6, and on page 6 we're going to highlight the last  
14 full paragraph on that page, the bottom right-hand  
15 corner.

16 Now, the third sentence in there talks about  
17 an increase in MCP-1 expression. First off, what is  
18 "MCP-1"?

19 A It's a cytokine that's released by DL cells.

20 Q And is this part of the neuroinflammatory  
21 process?

22 A Yes, it is.

23 Q If you read further in that sentence, what  
24 does it describe about the significance of MCP-1 as it  
25 relates to the pathogenesis of autism?

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1           A     It mentions the relationship of this  
2           chemical not only to microglial activation but also  
3           specifically to the recruitment of monocytes,  
4           macrophages, to areas of neuronal cortical  
5           abnormalities, which is, in other words, the same  
6           phenomenon that I was referring to in my report that  
7           Dr. Kemper took issue with.

8           Q     So monocytes and macrophages; what types of  
9           cells are those?

10          A     These are cells that are not inherent in the  
11          brain but in the body and in the circulation, but I  
12          tracked it into locations around the blood vessels  
13          that supply the brain by the MCP-1.

14          Q     Is it your testimony that this description  
15          is consistent with the statement in your report that  
16          neuroinflammation is associated with the invasion or  
17          the infiltration of immune cells?

18          A     Yes. That's what I was referring to.

19               MR. POWERS: Let's take that down from the  
20          screen, and I'm also going to hand you another very  
21          well-known exhibit number. This is Petitioners' No.  
22          69. It's Dr. Vargas's article.

23               I would like to direct your attention, Dr.  
24          Kinsbourne, to page 5 of this exhibit. There is a  
25          section highlighted there, but we're actually going to

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1 look at something below that. About two-thirds of the  
2 way down the right-hand column, there is a phrase that  
3 begins, "In addition to the presence of activated  
4 macrobriala," and go ahead and highlight down to the  
5 bottom.

6 BY MR. POWERS:

7 Q Do you see a section in there where the  
8 authors describes their observation that there was a  
9 marked accumulation of perivascular macrophages and  
10 monocytes in the cerebellum of the autistic?

11 A Four of the autistic individuals. Well, as  
12 I pointed out, these cells come from the circulation,  
13 and they pass through the walls of the blood vessels  
14 into a perivascular location, which means around where  
15 the blood vessels flow in the brains of at least four  
16 of these 10 autistic people.

17 Q So, again, do you believe that this  
18 statement in the published literature is consistent  
19 with your description of the characteristics of  
20 neuroinflammation?

21 A Yes, it is.

22 MR. POWERS: We can pull that down.

23 BY MR. POWERS:

24 Q If you recall, Dr. Kemper took issue with  
25 something that you said on page 17 of your expert

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1 report. If you look at the third paragraph, it's the  
2 paragraph that begins with a citation to Dr. Vargas,  
3 but the sentence I'm interested in is at the very end  
4 of that paragraph, and it begins, "The inflammation  
5 becomes chronic...."

6 Now, Dr. Kemper took issue with the idea  
7 that cells, particularly astrocytes, are dying. What  
8 he said, if I recall the testimony, was that the  
9 Vargas folks did not find dead astrocytes, and,  
10 therefore, given the lack of dead astrocytes, that  
11 your friendly fire description was inaccurate. Do you  
12 recall that testimony?

13 A I do.

14 Q How would you respond to that criticism and  
15 let the Special Masters know exactly what you're  
16 describing with astroglial activation here?

17 A Well, there are two aspects to this. One is  
18 that what I have seen saying was that there are  
19 circumstances under which the immune attack is so  
20 severe that the astrocytes can, in fact, die.

21 I wasn't arguing that this was generally the  
22 case in autism. My point in autism is that there is a  
23 functional abnormality of astrocytes, and,  
24 specifically, that the astrocytes no longer perform  
25 their function of regulating the flow of glutamate,

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1 which, therefore, can spread and activate neurons that  
2 otherwise would not have been activated.

3 Death is not part of the model that I'm  
4 proposing, although I have no doubt that this is  
5 something that can happen and, on occasion, does  
6 happen.

7 Q In fact, in fairness, let's go ahead and  
8 highlight the remainder of the paragraph here.

9 So, in your report, you actually describe  
10 specifically what you think is going on with  
11 astrocytes. Is that correct?

12 A Yes.

13 Q Anywhere in there do you say that a  
14 necessary part of your model is that astrocytes are  
15 dying?

16 A No, and, in fact, it's a necessary part of  
17 my model that most of them don't die because if they  
18 die, the neurons would die, and we would have a  
19 totally different situation in the brain.

20 Q Now you do say that some will die. Correct?

21 A Yes.

22 Q I want to go back to Petitioners' Master  
23 Reference No. 72 and page 7. Halfway down the left-  
24 hand column, there is a sentence that begins,  
25 "Importantly, cells undergoing...."

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1 Now, you say that some astrocytes might die,  
2 and Dr. Kemper said no astrocytes are dying. Where  
3 can you find any support for the idea that some  
4 astrocytes might be dying, even if you didn't find the  
5 evidence of the actual dead cells?

6 A The substance, TGF-beta-1, as is pointed out  
7 here, is produced mostly by reactive astrocytes and  
8 neurons. That's the sixth line down of the  
9 highlighted section. It then says that this chemical,  
10 the cytokine, may reflect an attempt to modulate the  
11 neuroinflammation or repair injured tissue. In other  
12 words, it's, in a sense, considered to be anti-  
13 inflammatory as opposed to pro.

14 It doesn't the same in this paragraph, but  
15 the understanding is that that substance is, in fact,  
16 produced by astrocytes in the course of dying.

17 Q So if this is a substance produced by  
18 astrocytes in the course of their death, and elevated  
19 levels of this substance are present, what do you  
20 think the significance of that is, in terms of your  
21 description of what goes on with astrocytes in this  
22 process?

23 A Well, some astrocytes, in fact, have  
24 succumbed, but there is a mechanism for holding that  
25 process in check. It's a self-regulatory, protective

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1 mechanism that's being described here.

2 MR. POWERS: We can pull the article down.

3 BY MR. POWERS:

4 Q You also hit on another point that -- I  
5 think it was Dr. Rust specifically said that he  
6 thought your model implausible because he did not  
7 understand how it could be self-regulating. Do you  
8 recall that testimony?

9 A I do.

10 Q How would you respond to that accusation by  
11 Dr. Rust that your model is undermined because it  
12 cannot explain the natural process of  
13 neuroinflammation?

14 A Well, there are two ways of encountering  
15 that. One is that a number of articles in peer-  
16 reviewed journals have, in fact, found the concept of  
17 the overactivated glutamanergic state to be a  
18 feasible, reasonable concept.

19 The second is that, as Dr. Rust himself  
20 described in some detail, there are self-regulatory,  
21 corrective provisions in the brain for holding  
22 neuroinflammation in check so that, up to a point, it  
23 is quite biologically plausible that neuroinflammation  
24 may occur but not escalate to an overwhelming assault  
25 on the brain as a whole.

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1 In fact, this must be the case because both  
2 the Vargas group and, subsequently, the Lopez-Hurtado  
3 group found evidence of neuroinflammation not only in  
4 children but even in adults up to the age in the  
5 forties, and nobody argues that this neuroinflammation  
6 just began in the forties in those cases. So the  
7 evidence is that neuroinflammation can, as it was a  
8 similar way at some low level, continue for many, many  
9 years, which implies both that the pro-inflammatory  
10 factors continue and that anti-inflammatory factors  
11 hold it to some level of check.

12 Q Now, finally, Dr. Rust took issue with your  
13 characterization of the neuroinflammatory process, in  
14 particular, as being an environmental contribution to  
15 autism. Do you recall that critique?

16 A Yes.

17 Q I would like to go back to Petitioners'  
18 Master Reference List No. 72, and we're going to look  
19 at page 9. Again, this is a page that has been oft  
20 discussed, but I want you to discuss this in terms of  
21 responding to Dr. Rust's critique that he saw no way  
22 that neuroinflammation could be an environmental  
23 contributor.

24 Let's go ahead and look at the table at the  
25 top left hand of that page. Let's blow that up.

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1 Can you describe for the Special Masters the  
2 significance of that table in this published paper to  
3 your theory of causation in these cases?

4 A Yes, sir. It, in fact, talks about  
5 interactions between environmental and genetic factors  
6 that influence neuroglial activation and the presence  
7 of autism. Among the environmental factors, on the  
8 top left-hand, he mentions infections and toxins,  
9 maternal factors, and others.

10 So the notion that environmental factors are  
11 of significance mechanistically is embodied in this  
12 sketch, which then centers on neuroglial activation,  
13 and, at the bottom right-hand corner, the flow chart  
14 proceeds to the outcome of the autistic phenotype,  
15 featuring, particularly, regression as part of the  
16 phenotype that's being described here in terms of its  
17 mechanistic origins.

18 Q Let's go ahead and look at the text of this  
19 page, under the "Conclusions" section. In that  
20 section, about half-way through the section called  
21 "Conclusions," there is a sentence that begins, "Our  
22 neuroimmunopathological studies...."

23 Let's go ahead and highlight that, if you  
24 would, please.

25 Now, the authors make a point here that, to

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1 the extent there is an immune response involved, it's  
2 innate rather than adaptive. Correct?

3 A Yes. That's been made very clear  
4 throughout.

5 Q Have you ever, in your testimony or in your  
6 report, implied that what's going on in the brain is a  
7 response of the adaptive immune system.

8 A Not at all.

9 Q What immune response are you describing in  
10 your testimony, your report, and your opinion?

11 A I'm describing the innate immune response,  
12 which, in the body, has to do with macrophages and  
13 mononucleocells, and, basically, it's a kind of  
14 inflammation that one has if one scratches one's arm,  
15 and the area gets red and a bit swollen through edema  
16 and hot and so on. But that immune response, when it  
17 occurs in the brain, is still innate, but it features  
18 the macroglia and the astrocytes as we have discussed.

19 Q Well, move down a little bit further, and  
20 the sentence beginning, "The roles of neuroglial  
21 activation...." Now, in this sentence, there is talk  
22 about some sort of preexisting central nervous system  
23 abnormalities, and it says that "neuroinflammation  
24 might maintain some of those abnormalities." Do you  
25 see that?

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1           A     Yes.  It might maintain them, but it goes on  
2     to say, if not also initiating some of them.  That's  
3     consistent with the Courchesnean point of view that we  
4     have already talked about.

5           Q     Now, Dr. Kemper, the neuropathologist who  
6     testified, it was his position, if you recall, that  
7     the neuroinflammatory responses seen here were in  
8     response to the underlying brain pathology.  Do you  
9     remember that testimony?

10          A     Yes.

11          Q     And, certainly, that's a possibility that  
12     these authors are leaving wide open.

13          A     Right.

14          Q     Does that possibility exclude the possible  
15     that the neuroinflammatory process might initiate some  
16     of the abnormalities in this disorder?

17          A     No, it doesn't at all.  It might be either,  
18     or it might be both.

19          Q     It's just uncertain.

20          A     Correct.

21          Q     Let's look at the very last sentence in the  
22     paper here.  This is the sentence that says,  
23     "Neuroglial and neuroinflammatory responses likely  
24     have polygenic and environmental bases and may have  
25     important clinical and therapeutic implications in

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1 autism."

2 Can you explain what you think the  
3 significance of that concluding statement is to your  
4 opinion and to your report and your testimony?

5 A Yes. I've been arguing that gene  
6 environment interaction is an important factor  
7 potentially in causing autism, and they are saying  
8 that, that "polygenic," the gene component, and the  
9 environmental basis interacting may set up the  
10 neuroglial neuroinflammatory responses, and they, in  
11 turn, may have important implications for autism.

12 Q Is it your opinion, Dr. Kinsbourne, that the  
13 work of Drs. Vargas and Pardo supports your theory and  
14 your mechanism of injury in these cases?

15 A Indeed, I based a lot of it on their work.

16 MR. POWERS: I have no further questions  
17 right now.

18 SPECIAL MASTER VOWELL: Thank you. Is  
19 Respondent's counsel ready to proceed?

20 MR. MATANOSKI: I believe so, ma'am.

21 (Pause.)

22 CROSS-EXAMINATION

23 BY MR. MATANOSKI:

24 Q Welcome back, Dr. Kinsbourne.

25 A Yes.

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1 Q For the record, I'm Vince Matanoski.

2 Doctor, I'm going to begin where Mr. Powers  
3 began, with the settlement that you reached with the  
4 University of Toronto. Now, as part of that  
5 settlement, you agreed to tender your resignation.  
6 Isn't that right?

7 A No.

8 Q As part of Petitioners' Trial Exhibit 12,  
9 paragraph 4, it says, "The applicant tenders his  
10 resignation from the university."

11 A There were two parts to that. The first  
12 part was that the charges were rejected, and they were  
13 quashed, and I was offered the opportunity of staying  
14 at the University of Toronto. However, I elected, as  
15 part of my settlement, to leave.

16 Q And that is part of the settlement that you  
17 attend to your resignation.

18 A That is part of the ultimate settlement  
19 which you have before you.

20 Q Thank you. Doctor, you were asked a series  
21 of questions about criticisms from Dr. Kemper. Did  
22 you listen to his testimony?

23 A Yes.

24 Q Could you tell me what those criticisms  
25 were?

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1 A Do you mean the ones we just went over?

2 Q Yes.

3 A He criticized my statements about neuroglia.

4 Q Can you be any more specific about what his  
5 criticism was?

6 A Yes. He said that there was no edema in  
7 neuroinflammation, and he agreed with the microglial  
8 activation, and then he disagreed with the third item  
9 that I mentioned --

10 Q What was that item?

11 A -- which I've forgotten for the moment.

12 Q Even though you listened to his testimony,  
13 and you were just testifying about it, you can't  
14 remember the number.

15 A Even though we just talked about it, yes.

16 Q Are you sure you listened to his testimony?

17 A Am I sure I listened? Yes, of course, I'm  
18 sure I listened.

19 Q I was just wondering, if you were not being  
20 led through the questions, whether you can even count  
21 what the criticisms were.

22 A Well, I'm sorry you're wondering, but I  
23 listened to his testimony.

24 Q But you can't recall even what the third  
25 matter that Dr. Kemper brought up in his testimony.

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1           A     It will come back to me, if you would like  
2     me to think further.

3           SPECIAL MASTER VOWELL:  Counsel, I'm just  
4     going to  
5     ask -- I'm reading lips, but I would like to further  
6     be assisted by hearing you, as I'm sure everyone else  
7     will, if both Dr. Kinsbourne and counsel would speak  
8     up just a little.

9           MR. MATANOSKI:  I'm sorry.

10          BY MR. MATANOSKI:

11         Q     Dr. Rust; you listened to his criticisms,  
12     too.  Is that right?

13         A     I did.

14         Q     Can you tell me now what those criticisms  
15     are?

16         A     Oh, there were an awful a lot of criticisms.

17         Q     How about giving me --

18         A     I'll give you a few, yeah.  My theory is  
19     unbelievably complex.  My theory is well put.  My  
20     theory is totally novel.  These are my discoveries.  I  
21     have ignored 30 years of neuroscientific research.  
22     There are some highlights.

23         Q     Can you be any more specific about what --

24         A     Well, I'm telling you things that he said,  
25     and I'm using almost exactly his specific words.

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1 Q You can't be any more specific than that.

2 A I haven't finished my response to your  
3 question.

4 He criticized my point of view about  
5 regression as being striking, although Dr. Richler, in  
6 fact, describes regression as being striking.

7 He criticized my scientific approaches,  
8 speculative. He didn't believe that regression could  
9 be interpreted as the cause of an ongoing disease  
10 because, in Rett syndrome, there is regression, which  
11 is attributable to genetic causes.

12 He found my model of overarousal to be  
13 really a misinterpretation of the behavior of children  
14 with autism under stress. He pointed out that once  
15 they are in familiar, calm situations, that they quiet  
16 down, and on the longer -- here are some examples.

17 Q He was pretty broad and pretty much  
18 criticized just about every part of your opinion,  
19 didn't he?

20 A Oh, he did, yes.

21 Q Doctor, you were given a study to look at.  
22 I think this is Trial Exhibit 13 perhaps. Now, that  
23 study was for safety, wasn't it, a drug safety test?

24 A What are you referring to?

25 Q The one you were just handed, Trial Exhibit

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1 13, a study by -- they were recruiting participants  
2 for a drug study.

3 A Oh, the riluzole study?

4 Q Yes.

5 A Yes.

6 Q That was a drug safety study, wasn't it?

7 A Well, it says, on the front sheet, "This  
8 research study will examine the effectiveness of  
9 riluzole for treating such a composite result."

10 Q And on page 3 of that study proposal?

11 A Are you going to direct my attention to it?

12 Q I don't have it in front of me. Actually, I  
13 do. Doesn't it say, "This proposal is for a 12-week,  
14 single-arm, open-label study that will evaluate safety  
15 and estimate dose of children," the second paragraph,  
16 first full paragraph, of that page?

17 A It does say that, yes.

18 Q And is that study limited to children with  
19 regressive autism?

20 A Did you say, is it limited?

21 Q Is that study limited to children with  
22 regressive autism?

23 A As I pointed out, this is an agent which was  
24 initially shown to be of some effectiveness for OCD,  
25 and now it's being extended to the autistic children

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1 as well.

2 Q It is not limited to children with  
3 regressive autism.

4 A No, it's not limited.

5 Q Your opinion is limited to children with  
6 regressive autism, is it not?

7 A I'm talking about children with regressive  
8 autism.

9 Q You limit your mechanism, for purposes of  
10 this proceeding, to children with regressive autism.  
11 Correct?

12 A No, I don't limit it. I am discussing it in  
13 the context of regressive autism. Whether the  
14 mechanism is applicable in other conditions, I haven't  
15 considered.

16 Q So your mechanism is not applicable solely  
17 to regressive autism.

18 A I don't know. I haven't considered that. I  
19 haven't considered it in the context of regressive  
20 autism, and I have not considered it in other  
21 contexts.

22 Q Then consider it now. Would it be  
23 applicable equally to other kinds of autism, not just  
24 regressive?

25 A I don't know. I would need to consider

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1 that, based on the medical literature. I can't give  
2 you --

3 Q Well, why did you consider it only with  
4 respect to regressive autism?

5 A Because the issues before this Court have to  
6 do with possible and environmental postnatal effects  
7 of certain agents, in one case, the measles vaccine  
8 virus and, in other case, the mercury. When postnatal  
9 effects are being considered, the disorders that  
10 appear to be postnatal, such as regressive autism,  
11 which seem to be the relevant disorders to consider in  
12 the first instance.

13 Q But if your mechanism applies equally to all  
14 of the kinds of autism, then it certainly doesn't just  
15 explain away regressive as postnatal. It could be  
16 anything. Correct? It could be any kind of autism  
17 that your mechanism applies to.

18 A I am not giving an opinion about whether or  
19 not my mechanism applies to other kinds of autism.

20 Q So, at this point, you can't say that it is  
21 limited only to regressive autism, your mechanism.

22 A I have not considered the universe of other  
23 possibilities for this mechanism.

24 Q Yet you would use it as part of a  
25 differential diagnosis to determine whether or not

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1 autism occurred as a result of vaccination exposure.

2 A I don't use a mechanism for a differential  
3 diagnosis.

4 Q That's how you came to your conclusion.  
5 Your mechanism is how you came to the conclusion, at  
6 the end of your report, that you would consider  
7 vaccine exposure in the differential diagnosis of  
8 autism.

9 A I did, indeed. I offered a medical reason  
10 or mechanism, and then I said that, given that, among  
11 the potential triggers for neuroinflammation, are  
12 viruses and heavy metals, viruses and heavy metals  
13 should be considered a differential diagnosis, which  
14 would intuit, of course, any source of virus, such as  
15 the measles vaccine virus, and any source of heavy  
16 metal, such as thimerosal.

17 Q And so you concede that your postulate that  
18 you have would apply equally to other exposures, even  
19 if we were just to consider potential postnatal  
20 causes.

21 MR. POWERS: Excuse me, Special Masters.  
22 I'm going to object to the extent that we're now  
23 beyond surrebuttal. These are questions that go to  
24 Dr. Kinsbourne's earlier direct testimony and could  
25 have been raised, and may even have been raised, on

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1 cross, at this point. These are way outside of the  
2 scope of the rebuttal testimony of Dr. Kinsbourne this  
3 morning.

4 SPECIAL MASTER VOWELL: Mr. Matanoski?

5 MR. MATANOSKI: I'll withdraw the question.  
6 I think he has answered this before, actually. I  
7 think it's in his report. I imagine it's clear to the  
8 Court now that his mechanism is not specific to the  
9 mercury vaccine.

10 MR. POWERS: Again, I object to counsel, on  
11 an examination of a witness, making arguments on the  
12 record to the Court here. I just raise the objection  
13 that when counsel is directing questions to the  
14 witness, that they be questions to the witness and not  
15 argument to the Masters.

16 SPECIAL MASTER VOWELL: So noted.

17 BY MR. MATANOSKI:

18 Q Doctor, you talked a lot about glutamate  
19 excess. How do we get to that process of glutamate  
20 excess from vaccines? Is it the inorganic mercury in  
21 your causal mechanism?

22 A Neuroinflammation involves a process that I  
23 have already explained in detail in my report and in  
24 my direct testimony, which raises the discontrol of  
25 glutamate by its normal regulatory mechanisms.

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1           Therefore, whatever might cause  
2           neuroinflammation is also -- cause glutamate excess.  
3           I pointed out the three categories, known categories,  
4           of agents that could cause neuroinflammation.

5           One category would be viruses persisting in  
6           the body, the second would be heavy metals, and the  
7           third would be neurodegenerative disorders.

8           Among that range of causations, vaccines  
9           could play a role in two respects: one, insofar as  
10          delivering a virus which stays in the body of  
11          particular children, and the measles vaccine virus has  
12          been shown to do so in some autistic children; and the  
13          other, a vaccine that contains, or, at least,  
14          contained, mercury as part of its chemical  
15          constitution and, therefore, would be one of the  
16          available vehicles for delivering mercury to the body  
17          and, therefore, to the brain.

18          Q       And that would be in the form of inorganic  
19          mercury, in your opinion.

20          A       Well, it wouldn't enter the brain in that  
21          form, but once in the brain, it would become  
22          decomposed to that.

23          Q       And this glutamate excess is built up  
24          because of inorganic mercury in the brain.

25          A       One of the many possible causes of glutamate

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1 excess in the brain would be a triggering by the  
2 effect of low levels of inorganic mercury.

3 Q And this glutamate excess is going to  
4 exacerbate, or continue to increase, as the inorganic  
5 mercury continues to increase. Correct?

6 A Not necessarily.

7 Q Why not?

8 A Why should it?

9 Q So you don't know why it should or  
10 shouldn't.

11 A No. You don't know why it should or  
12 shouldn't. I never made the claim that the glutamate  
13 excess would necessarily become worse and worse, and I  
14 pointed out today, in testimony that I did give as  
15 opposed to this topic, which we didn't address today,  
16 that regulatory mechanisms, which can also keep the  
17 glutamate excess in check.

18 Q So what causes those regulatory mechanisms  
19 to fail?

20 A I didn't testify that the regulatory  
21 mechanisms failed.

22 Q If they are in check, there isn't excess  
23 glutamate.

24 A No. That's not true. You have a certain  
25 amount of excess glutamate, but it's capped. It is

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1 precluded from becoming out of control by anti-  
2 inflammatory cytokines and regulatory cells.

3 Q And after this initial impact on that  
4 glutamate regulatory system by inorganic mercury,  
5 subsequent amounts of inorganic mercury had no impact  
6 on that regulatory system.

7 A I don't know whether it has no impact. It  
8 might have some impact. It might have no impact in  
9 some people than others. It might have less impact  
10 over time and yet others. This is a level of  
11 specificity which I can testify to and don't need to  
12 establish my mechanism.

13 Q So you're willing to say that inorganic  
14 mercury will induce glutamate excess, but then, after  
15 it induces it, you have no idea what inorganic mercury  
16 might do after that.

17 A As long as it stays there, it will maintain  
18 the neuroinflammation. That is something I have an  
19 idea about, and I've just stated that idea. Whether  
20 that neuroinflammation will become worse, stay the  
21 same, or get better, I'm sure, varies from person to  
22 person.

23 Q And you have no way of determining that.

24 A You would have to show me the persons.

25 Q Well, what would you look for in a person to

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1 determine why there would be more glutamate excess in  
2 one person or another as a result of inorganic  
3 mercury?

4 A First of all, we have to show that there is  
5 more. Secondly, I would then have to consider the  
6 particular case where there are no conceivable  
7 reasons. This goes way beyond my report and will be  
8 in my testimony.

9 Q So you just got us to some will do it, and  
10 you don't know what's going to happen after that.  
11 Some inorganic mercury will do it, create this excess,  
12 but you have no idea what's going to happen after  
13 that.

14 MR. POWERS: Again, I object. You were  
15 talking about a dose issue, and Dr. Kinsbourne, on  
16 rebuttal testimony, wasn't talking about dose at all.  
17 This was an issue that he raised on direct testimony.  
18 He was crossed and re-crossed on that issue, and now,  
19 rather than rebuttal cross-examination, re-re-re-  
20 cross, and, again, I object because we're going way  
21 outside his rebuttal testimony and his cross-  
22 examination.

23 SPECIAL MASTER VOWELL: Mr. Matanoski?

24 MR. MATANOSKI: Ma'am, I don't know why Dr.  
25 Kinsbourne is telling us about neuroinflammation, if

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1 he can't tie it to the vaccine or the inorganic  
2 mercury, and tell us what's going to happen with  
3 respect to the inorganic mercury. He is only telling  
4 us that neuroinflammation, which, he admits, can be  
5 caused all kinds of possible factors.

6 If he can't tie it to inorganic mercury and  
7 explain how he is tying the neuroinflammation, the  
8 inorganic mercury, to reach a conclusion, at the end  
9 of his report, that you should consider thimerosal-  
10 containing vaccine, it is impossible --

11 SPECIAL MASTER HASTINGS: This is not  
12 argument.

13 SPECIAL MASTER VOWELL: No.

14 MR. MATANOSKI: Well, I was trying to  
15 explain why he thought --

16 SPECIAL MASTER VOWELL: -- the questioning.  
17 I think he has limited his answer. He has delimited  
18 his answer with he hasn't considered, or he is  
19 uncertain, and needed to examine the individual. So  
20 perhaps we can move to other lines of questioning.

21 MR. MATANOSKI: Yes, ma'am.

22 BY MR. MATANOSKI:

23 Q How much glutamate excess needs to be built  
24 up before you get the excitatory effect that you are  
25 postulating?

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1 A No one that I know of has quantitated that.

2 Q So that's not capable of being tested.

3 A No. I think it is capable, ultimately. In  
4 fact, I think that magnetic-resonance-spectroscopy  
5 methods are either currently available or will be very  
6 soon available to actually see whether, in the brain,  
7 there is microbial activation; whether, in the brain,  
8 there is inorganic mercury. This is really a good  
9 range, and, within a short time, we'll know whether  
10 it's right or wrong.

11 Q My question was the amount of glutamate, not  
12 neuroglial activation.

13 A No. I understand your question, sir. This  
14 is a question that I can't answer, and I don't believe  
15 Respondent witnesses could either.

16 Q So is there a way of measuring how much  
17 glutamate will be needed, excess glutamate, to create  
18 the excitatory effect that you're postulating and  
19 trying to defend here?

20 A To my knowledge, that cannot be measured in  
21 humans, in living humans. There are in vitro models  
22 in which it can potentially be measured.

23 Q Do you have any idea what the measurement of  
24 excess glutamate would be before it becomes  
25 excitotoxic?

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1 A I offer no quantitative opinions, no.

2 Q Can the glutamate excess that you're  
3 postulating manifest in the overexcitation in the  
4 period of a day?

5 A I based my testimony on medical literature,  
6 and I'm unaware of any medical literature that  
7 addresses that question.

8 Q Could it remain latent for years?

9 A Could it do what?

10 Q Could this process of glutamate excess  
11 remain latent for years without it manifesting itself  
12 in clinical symptoms?

13 A I know of no literature which puts a  
14 timeframe on this.

15 Q You cited the Purcell paper, which was PML  
16 567, I believe -- maybe that was "67" -- that paper  
17 didn't deal with regressive autism, did it? Not  
18 exclusively with regressive autism, did it?

19 A That's correct. It didn't.

20 Q It was 567. In that paper, the authors --  
21 this is a portion that you did not cite or discuss --  
22 didn't the authors state, and this would be on page 9  
23 of 567, "As we are examining postmortem samples long  
24 after one set of the disorder, it is more likely that  
25 we are identifying secondary consequences of the

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1 disorder." Isn't that right?

2 A It probably is, but could you refer me to  
3 where it says that?

4 Q Under "Discussion."

5 A Yes.

6 Q It's up on your screen now, too.

7 A Okay.

8 Q So those authors are saying these are  
9 secondary effects, not causative ones. Correct?

10 A In this particular sentence, they are saying  
11 that, yes.

12 Q They are discussing their article.

13 A Correct.

14 Q Now, you mentioned the article by Dr.  
15 Casanova. You called it an "important article" in  
16 your testimony just this morning.

17 A Yes.

18 Q Now, he proposes a deficit of inhibition,  
19 not an excitation.

20 A Correct.

21 Q That's not what you're postulating. You're  
22 postulating an overexcitation, not an inhibition.  
23 Correct?

24 A I am postulating a change in the excitation-  
25 inhibition balance in favor of excitation.

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1 Q Actually, sir, you were saying it's  
2 glutamate excess.

3 A Yes.

4 Q That's only one side of the balance.

5 A Correct. In other words, the balance is  
6 skewed in the direction of excitation.

7 Q Because of glutamate excess.

8 A What's that?

9 Q Because of glutamate excess in your  
10 postulate.

11 A That's correct.

12 Q How does Dr. Casanova propose that the  
13 deficit of inhibition occurs in his article?

14 A He is arguing that there is a problem with  
15 inhibitory interneurons.

16 Q So this inhibition, this deficit of  
17 inhibition, is actually a function of brain  
18 development. Isn't that right?

19 A Not necessarily.

20 Q Isn't that what he postulates in his  
21 article?

22 A As we have discussed, the question of brain  
23 development is an issue which is postnatal as well as  
24 prenatal.

25 Q In his article, doesn't Dr. Casanova

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1 postulate or say that he believes that it occurs in  
2 the prenatal period?

3 A You may be right, but I don't want to rely  
4 upon that. Can I refer to the article?

5 Q Certainly. Well, would you accept that he  
6 does and that, in fact, he says that it's in the first  
7 trimester?

8 A I think that's perfectly possible. However  
9 --

10 Q Aren't minicolumns formed in the first --

11 A Let me explain the relevance of Dr.  
12 Casanova's statement to my theory. I was not  
13 referring to his article necessarily as corroborating,  
14 or even being pertinent, to my proposals as to the  
15 origin of the new information and the autism.

16 I was pointing to his article in response to  
17 the criticism that the overactivation-overarousal  
18 theory is outdated and not to be considered. He was  
19 considering it very seriously. That was the point  
20 about his article that I was presenting to the Court.

21 Q But his mechanism is one that's prenatal in  
22 origin.

23 A His and some other people's, too. Dr.  
24 Zimmerman has also taken that position, absolutely.

25 Q Now, your attention was drawn to the

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1 Courchesne article. You cited that in your report.  
2 That would be Petitioners' Master Reference No. 104.

3 In your report, and today, you cite this  
4 article for the propositions where support for your  
5 postulate, neuroinflammation, as part of your  
6 causative mechanism. In your report, you cited that  
7 part of the article that dealt with neuroinflammation.  
8 However, you omitted the other factors that the  
9 Courchesne authors were looking at as possible causes,  
10 didn't you?

11 A You have to show me the report and the other  
12 factors. I don't have my report before me. Perhaps  
13 you can refer me to the statement.

14 Q Here is the article. I guess we've already  
15 highlighted the sections that are involved.

16 You cited the last part of this,  
17 "Compensatory neurogenesis during a prenatal or  
18 postnatal life that is triggered by adverse events  
19 such as those that ignite the neuroinflammatory  
20 reactions reported by Vargas, et al."

21 SPECIAL MASTER HASTINGS: Can we identify  
22 that number for the record?

23 MR. MATANOSKI: It's page 8, going onto page  
24 9, sir.

25 SPECIAL MASTER HASTINGS: Thank you.

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1 BY MR. MATANOSKI:

2 Q Prior to that, they listed two other  
3 possible mechanisms here to explain their findings.  
4 Didn't they say, prior to that, that the possibilities  
5 also included a failure to correctly regulate the  
6 number of neurons produced during the neurogenesis  
7 stage of prenatal development in autism, and, as  
8 another possibility, a deficit or delay in apoptosis  
9 so that too many survive into postnatal life?

10 A I would like to make it clear, again, that  
11 I'm not presenting a discovery as to what impact is  
12 the true and scientific cause of autism. I'm  
13 presenting one of a number of medically reasonable  
14 possibilities.

15 I'll make it clear again that there are  
16 other reasonable medical possibilities, and the  
17 responsible articles mention those. I'm not arguing  
18 that my proposal is better or worse, and certainly not  
19 that my proposal excludes other interpretations. Of  
20 course, it does not.

21 Q Your attention was drawn to Petitioner's  
22 Master List No. 247. You described that as an article  
23 of some interest. Is that article and the discussion  
24 therein limited to regressive autism?

25 A I'm sorry. What are you referring to here?

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1 Q You had just discussed, in your direct  
2 testimony this morning, Petitioner's Master List No.  
3 274, which you described as an article of some  
4 interest, and I was just asking you if that article  
5 was limited to a discussion of regressive autism.

6 A I don't have the -- Casanova did this.

7 SPECIAL MASTER VOWELL: Yes.

8 THE WITNESS: No, not specifically.

9 BY MR. MATANOSKI:

10 Q In defending your reliance on an overarousal  
11 model, you did mention, this morning, your list  
12 article, which you actually cite in your report.

13 A Right.

14 Q Your list article came out in 2006, and you  
15 describe overfocusing in that article. Is that  
16 overarousal?

17 A I think overfocusing is caused by  
18 overarousal, yes, but that article was not a  
19 neurobiological article; it was a behavioral article.

20 Q And you didn't mention, in that article,  
21 that glutamate is a model of overflow --

22 A No, because it was not a neurobiological  
23 article; it was a behavioral article.

24 Q Now, you seemed to, in your testimony this  
25 morning, be making it clear that you're not

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1 necessarily saying that it's astrocyte death that's  
2 occurring. Is that right? Would that be a fair  
3 representation of what you were telling us this  
4 morning?

5 A I would like to reword it. It's not that  
6 I'm not necessarily saying it; I'm not saying it. My  
7 model does not postulate astrocytic death as being an  
8 essential component, no.

9 Q In your testimony in Cedillo, you postulated  
10 the same mechanism and described it as one of  
11 astrocyte death. Correct?

12 A You would have to show me that. I cannot  
13 remember that at all.

14 Q You can't remember. And, in Snyder, the  
15 case that you testified last fall with the same  
16 mechanism, you described astrocyte death occurring.  
17 Is that right?

18 A I don't know because you would have to show  
19 me what I said. Obviously, I don't remember the words  
20 I used.

21 Q And in support for your proposition, in both  
22 your report and in your discussion this morning, you  
23 referred to the Aschner article. 570, I think, was  
24 one that you put up, and there is also 568-P for this  
25 Petitioners' Master List References 568 and 570 as

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1 support for your model of glutamate excess.

2 Let me just turn to that quickly. In Dr.  
3 Aschner's discussion of this, as he describes it, an  
4 "excitotoxic model," doesn't he describe this process  
5 in Petitioners' Master List No. 568 as a "vicious,  
6 amplifying cycle of neurotoxic cascade"?

7 SPECIAL MASTER VOWELL: And counsel is  
8 referring specifically to --

9 MR. MATANOSKI: -- page 5 of --

10 SPECIAL MASTER VOWELL: -- PML 568.

11 MR. MATANOSKI: Yes, ma'am.

12 SPECIAL MASTER VOWELL: Thank you.

13 THE WITNESS: Where on page 5 should I look?

14 MR. MATANOSKI: It's actually up on your  
15 screen, sir. Doctor, if you would like to, it's right  
16 up on the screen, so it might be easier for you.

17 THE WITNESS: Right. I can see the words.

18 BY MR. MATANOSKI:

19 Q And, in 570, that you were discussing this  
20 morning, page 2, the same page you were on, if you  
21 went down the paragraph a little bit further from  
22 where your attention was directed, to the very end of  
23 that same paragraph, second column -- if we can bring  
24 that up -- perhaps we can't.

25 Do you still have 570 from where you were

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1 discussing it this morning?

2 A 570.

3 Q Is that in front of you, Doctor?

4 A Yes, it is.

5 Q Page 2. Right above the paragraph that  
6 begins with the bold, "Role of --" could I draw you  
7 attention to that?

8 A The paragraph that begins with what?

9 Q The paragraph that immediately precedes the  
10 bolded part that says, "The Role of Reactive --"

11 A Yes, okay.

12 Q Your attention was drawn, this morning, to  
13 some discussion in the text a little bit before that  
14 that talked about astrocytic glutamate uptake being  
15 inhibited, and you were using that as support for a  
16 proposition of not necessarily astrocyte death but  
17 just an inhibition in the function of the astrocytes  
18 could result in this glutamate imbalance.

19 A That's correct.

20 Q If you could carry that discussion down from  
21 Dr. Aschner to the end, doesn't he conclude that it  
22 sets in motion an unimpeded cytotoxic cycle?

23 THE WITNESS: The exact phrasing, if we can  
24 go back to it -- can we go back to it?

25 MR. MATANOSKI: It's right there in the

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1 document that you were looking at.

2 THE WITNESS: My screen is blank,  
3 unfortunately.

4 MR. MATANOSKI: It was page 2 of the  
5 document that you had in front of you.

6 THE WITNESS: Okay.

7 MR. MATANOSKI: The same paragraph you were  
8 reading from, Doctor. It's now up on your screen.

9 BY MR. MATANOSKI:

10 Q My question to you was, if you carried the  
11 discussion on past where you were relying on this as a  
12 proposition that inhibition, without necessarily  
13 astrocytic death, can lead to glutamate excess, don't  
14 the authors here, Dr. Aschner, in particular, say that  
15 it sets in motion an unimpeded cytotoxic cycle?

16 A Yes. There was a statement about if it  
17 became synchronous, and I'm trying to find that  
18 statement again. You showed it to me earlier. I  
19 don't see it now, but the point is that, indeed, the  
20 end point is potentially a cytotoxic death, indeed,  
21 but, as I pointed out, in the living brain, there are  
22 regulatory mechanisms that could preclude that cycle  
23 from coming out of control in this fashion.

24 Q And in your report, in further discussing  
25 the role of astrocytes and trying to tie it into

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1 mercury, you discussed the Charleston article, and, in  
2 that article -- that's PML 116 -- and on page 10 of  
3 your report, you state that the astrocyte population -  
4 - you describe that article as standing for the  
5 proposition that the astrocyte population in the brain  
6 decreased significantly.

7 A Yes, it did.

8 Q But, in Vargas, which you described this  
9 morning and talked about, PML 69, the authors did not  
10 find any astrocyte loss. Is that right?

11 A That's correct.

12 Q And that was true in all of the autopsy  
13 samples of all of the autism patients they looked at.  
14 Isn't that right?

15 A That is correct.

16 Q Both regressive and nonregressive.

17 A Right.

18 Q And, in Lopez-Hurtado, PML 446, another  
19 autopsy study in autistic individuals, they reported  
20 no astrocyte loss. Isn't that correct?

21 A That's correct. And the Charleston people  
22 also didn't report any at 12 months and at 18 months,  
23 only at six months.

24 Q So then, in those articles, they actually  
25 recovered, and it was not a lasting impact.

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1           A     Either it recovered, or there was a  
2           compensatory proliferation of astrocytes, and, in  
3           fact, in Charleston, people point out that toxic  
4           insults often do cause a reactive proliferation of  
5           astrocytes.

6           Q     In compensation.

7           A     Yeah.

8           Q     So the astrocytes are available, then --

9           A     Right.

10          Q     -- to mop up the excess glutamate.

11          A     I think that may or may not be an outcome of  
12          that. They don't talk about that.

13          Q     But the astrocytes would be available.

14          A     Would be available?

15          Q     Yes. You discussed Dr. Pardo's article,  
16          which is PML 72. You would agree that Dr. Pardo is in  
17          the best position to interpret the significance of his  
18          own work, don't you?

19          A     I think that's true of Dr. Pardo, I'm sure,  
20          yes.

21          Q     I'm sorry?

22          A     Yes, of course, yeah.

23          Q     And your opinion that mercury from a vaccine  
24          is a potential cause of autism was only formed in the  
25          last few months. Correct?

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1           A     It's true that I've only studied this  
2           seriously in the last few months, yes.

3           Q     And you only came to that opinion in the  
4           last few months.

5           A     I have been aware since I first began to  
6           study for this cycle of cases that one of the causes  
7           of neuroinflammation is heavy metals, and I believe I  
8           mentioned that in previous reports, but I haven't paid  
9           serious consideration to the issue specifically of  
10          mercury until recently.

11          Q     When I asked you that question in November  
12          in Snyder, you indicated to me that you had not formed  
13          a conclusion at that point.

14          A     That's true.

15          Q     And this postulate that you've laid out  
16          before the Court; you've never presented that for  
17          publication or peer review. Correct?

18          A     I only came to this conclusion quite  
19          recently.

20                 MR. MATANOSKI: Thank you. I have no  
21          further questions.

22                 SPECIAL MASTER VOWELL: Thank you. Anything  
23          further from Petitioners' counsel?

24                 MR. POWERS: No, Special Master. Nothing  
25          further.

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1 SPECIAL MASTER VOWELL: Any questions from  
2 my colleagues?

3 (No response.)

4 SPECIAL MASTER VOWELL: Okay. It looks like  
5 it's about ten-forty. I do see that Dr. Mumper has  
6 arrived, but I understood, from our earlier off-the-  
7 record discussions that -- apparently, it's ten-forty-  
8 seven. My computer is now connected up to the  
9 chronometer I've just been handed.

10 But my question is, did counsel want to  
11 prepare for a brief break to address the item that  
12 counsel had indicated that they wanted to take up off  
13 the record?

14 MR. MATANOSKI: I'm sorry, ma'am. Are you  
15 proposing that we take that up right now or take the  
16 break and then take it up, or during the break?

17 SPECIAL MASTER VOWELL: We take a break so  
18 that we can take up the item before we get to Dr.  
19 Mumper's testimony.

20 MR. MATANOSKI: That sounds fine, Your  
21 Honor.

22 SPECIAL MASTER VOWELL: I suggest we take it  
23 up so that we have some opportunity to think about it,  
24 have a little bit of a break, and resolve, if there is  
25 anything that needs to be resolved, and then proceed

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1 into Dr. Mumper's testimony.

2 MR. POWERS: Thank you.

3 SPECIAL MASTER VOWELL: Okay. Let's see, if  
4 we're here, my thought would be just about a 15-minute  
5 break for the mid-morning break, which would put us  
6 roughly  
7 at -- I'm shortchanging you a couple of minutes, but  
8 roughly at 11, or we'll say five after so we can have  
9 our brief conversation here.

10 MR. MATANOSKI: Thank you, ma'am.

11 SPECIAL MASTER VOWELL: Okay. Thanks. We  
12 are in a brief recess.

13 (Whereupon, a short recess was taken.)

14 SPECIAL MASTER VOWELL: Please be seated.  
15 We are back on the record, and I understand that,  
16 based on our off-the-record discussion, whatever  
17 concerns Respondent had pertaining to the videotaped  
18 testimony that will accompany Dr. Mumper's testimony,  
19 there is no objection but a reservation by Respondent,  
20 as I understand, to counter with video as necessary.  
21 Does that accurately reflect your position?

22 MR. MATANOSKI: That's correct, ma'am.

23 SPECIAL MASTER VOWELL: Thank you. To  
24 proceed, Petitioners' counsel, just let me draw to  
25 your attention, Dr. Mumper, that you will continue

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1 under the same oath that was administered and you took  
2 earlier in the proceeding. Thank you.

3 Whereupon,

4 ELIZABETH MUMPER, M.D.

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

8 DIRECT EXAMINATION

9 BY MR. POWERS:

10 Q Good morning, Dr. Mumper.

11 A Good morning.

12 Q We, obviously, have been in this position  
13 before earlier when you gave direct testimony, but, to  
14 make it clear on the record here, my name is Tom  
15 Powers, along with Mr. Williams.

16 We represent the King and Mead families, as  
17 well as the Petitioners' Steering Committee, and we  
18 have you on the witness stand today to respond to  
19 specific testimony that was offered on direct  
20 testimony by the Respondent's experts. Is that your  
21 understanding of why you're on the stand today?

22 A Yes, sir, I understand.

23 Q Did you have opportunity to listen to the  
24 direct testimony and cross-examination of Dr. Rust?

25 A Yes, I did. I listened to the audio and

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1 took 40 pages of notes.

2 Q And when you say you listened to the audio,  
3 did you listen to the live version, the audio  
4 download, or some of both?

5 A Some of both.

6 Q Did you listen to the entirety of his  
7 testimony?

8 A Yes, I believe I did.

9 Q I want to go through some of the issues that  
10 Dr. Rust specifically raised as they apply to William  
11 Mead and Jordan King. Obviously, Dr. Rust covered a  
12 lot of ground, but we're going to focus on the case-  
13 specific testimony of Dr. Rust. Again, is that your  
14 understanding of what your testimony today is directed  
15 to?

16 A Yes, sir.

17 Q Now, do you recall some of his testimony  
18 about Rett's syndrome?

19 A Yes. I recall quite a bit of testimony on  
20 Rett's.

21 Q And as it applies to these cases, that  
22 testimony about Rett's syndrome, do you have a  
23 response to that for the Special Masters to explain  
24 how you think that testimony fit or didn't fit with  
25 his analysis of the two cases?

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1           A     Well, I had thought that he was on  
2     elaborating on Rett's syndrome a lot in order to lay  
3     some groundwork and then make some type of  
4     extrapolation or determination as specific to these  
5     two cases, and, as time went on, I had the same  
6     question in my mind that Special Master Hastings did,  
7     in terms of where it was going.

8                 Rett's syndrome is a very well-described  
9     syndrome that, the vast majority of the time, occurs  
10    in girls, and the genetics of it have been identified,  
11    in that we know actually the MECP-2 gene is involved,  
12    and, at some point, Dr. Rust seemed to be making the  
13    extrapolation that Rett's syndrome has a lot of  
14    autistic-type features, and we know the genetics of  
15    that, and, therefore, we can extrapolate that other  
16    versions of autism may well be genetic, and, you know,  
17    we still need to determine the genetics.

18                No doubt that that is true, to some extent,  
19    but to spend so much time on a disorder that is so  
20    fundamentally different from the way these two boys  
21    presented was very puzzling to me. He did make the  
22    point that Rett's is now being described in some boys,  
23    which I actually find intriguing from the standpoint  
24    of it opening the door to certain environmental causes  
25    because if boys typically do not get Rett's, one

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1 wonders what it is about those boys that they are now  
2 being identified, even though they are in the vast,  
3 vast, vast majority.

4 We do know, from work that Jill James has  
5 done and other people who work in oxidative stress  
6 literature that there are more challenges for boys in  
7 terms of handling environmental toxins, specifically  
8 with regard to the role of glutathione because, in  
9 general, females tend to have better preserved  
10 glutathione, and boys are at higher risk because of  
11 their relatively lower levels of glutathione.

12 So the Rett's issue, to me, was a lot of  
13 time spent on a disorder that is not really relevant  
14 to these two particular boys, and I understood that  
15 what I was supposed to do when I reviewed the records  
16 was to look at case-specific analyses, given the  
17 information from the medical records and the videos  
18 about these two particular boys, and try to generate  
19 some hypothesis about factors in their case that might  
20 be more specific to them as individuals.

21 Q Was there anything about Rett's syndrome  
22 that was informative to your opinions on individual  
23 causation in either of these cases?

24 A No.

25 Q There was also discussion by Dr. Rust about

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1 the possible that William Mead had a trajectory of  
2 head size or head circumference that reflected a  
3 pathological and congenital cause of autism. Do you  
4 recall that testimony?

5 A I do.

6 Q What is your response to the testimony that  
7 Dr. Rust offered?

8 A Well, I think the opinion that he was trying  
9 to make was that William initially had a relatively  
10 low- sized head circumference, and then he showed an  
11 increase in his head trajectory with a subsequent  
12 decline.

13 The thing that seems inconsistent to me is  
14 the fact that his newborn's head circumference was  
15 well in proportion to his body length and weight and  
16 was in around the 80 to 85th percentile at the time of  
17 birth.

18 Now, on -- I believe it was cross-  
19 examination, that Dr. Rusk said that, Well, perhaps  
20 that measurement was not very accurate because, you  
21 know, the child had just been through birth drama, and  
22 maybe he had bruising on his head.

23 So I went back to the medical records to see  
24 if, in William Mead's specific case, I could find any  
25 evidence of that, and I found three different

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1 references.

2 One is Exhibit 1, page 00031, which is an  
3 intake form with a physical exam in which it says,  
4 "Healthy male newborn and head and neck normal."

5 The other was a nursery record from  
6 Providence St. Vincent titled "Newborn Nursery  
7 Admission Assessment," and, Scott, I'm sorry, but my  
8 copy does not have an exhibit number, but I can turn  
9 it over to the Court where it says, "Head and face  
10 symmetrical, normal," and the skin says "normal," and  
11 there are opportunities there to check off a box for  
12 either bruising or petechiae, and that is not checked.

13 So whereas I can accept, in concept, Dr.  
14 Rust's observation that, in certain cases, the newborn  
15 head circumference might not be reliable, if there is  
16 significant trauma. Again, I don't think that we can  
17 just speculate about cases that he has known of. I  
18 think we have to look specifically at the child he was  
19 talking about, and I do not find any evidence in the  
20 medical record that substantiates that claim.

21 Q And is it your testimony that if that claim  
22 was, in fact, true, there would have been ample  
23 opportunity in the medical record to reflect that?

24 A I believe that is true.

25 Q And a reasonable physician would have made

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1 note of something that was abnormal as Dr. Rust  
2 described.

3 A Typically, you will see physicians  
4 documenting things like cephalhematoma if there is  
5 bleeding that's causing a large bruise on the brain  
6 that might interfere with the head circumference  
7 measurements. I know that it's certainly my practice  
8 to do that.

9 There is another thing called a "caput  
10 succedaneum" that is another term that physicians  
11 could document on their initial newborn physical  
12 examination. Neither one of those notations appears  
13 in the records, to the best of my observations of the  
14 records.

15 Q Do you also recall testimony by Dr. Rust  
16 that he believes that both Jordan and William were not  
17 normal in their development prior to regression? Do  
18 you recall that testimony?

19 A Yes.

20 Q Do you recall Dr. Rust being able to cite to  
21 any specific piece of evidence in the medical record  
22 in support of that contention?

23 A No. I did not see him point to anything  
24 specifically. He talked in terms of generalities, and  
25 I was actually struck by the fact that he seemed

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1 somewhat confused about the cases as he was  
2 testifying.

3 For example, one striking thing for me, when  
4 I reviewed William Mead's videos, is that his sister,  
5 Eleanor, appears in virtually all of the videos, and  
6 Dr. Rust was not able to remember that William had a  
7 sister.

8 So I would submit for the Court that I had  
9 paid some close observation to that fact and that it  
10 concerned me about the level of Dr. Rust's scrutiny,  
11 that he was unable to recall that, for example.

12 Q So it's your understanding, from listening  
13 to his testimony, that he was not able to identify  
14 anything in the videos with particularity, but also  
15 nothing in the medical records with particularity.  
16 Correct?

17 A I do not recall him saying anything  
18 specific. It's been a week since I listened, and I am  
19 open to a point where he may have said something, but  
20 I honestly do not recall it right now.

21 Q Now, Dr. Rust did describe the importance of  
22 talking to the parents and getting a good parent  
23 history as part of making any assessment of autism and  
24 the onset of autism. Do you recall that testimony?

25 A Yes. He was describing his practice in his

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1 own clinic at the University of Virginia in  
2 Charlottesville, and he went so far as to describe  
3 setting aside extra time at the end of the day so that  
4 he would have time to get a careful history.

5 What was not clear to me was how much of  
6 that history was actually performed by him versus  
7 taken by his residents, which is a very typical  
8 practice at most universities, including that  
9 university where I had some many years of experience.

10 Q In your clinical practice -- let me back up  
11 a little bit.

12 Do you agree, in principle, that a thorough  
13 parental history and thorough information from the  
14 parents; is that important information you have when  
15 diagnosing and treating autism?

16 A Yes. I think the history from the parents  
17 is probably the most crucial piece of information in  
18 putting together a picture of the entire child, not  
19 just with respect to his autistic symptoms but also  
20 with respect to his other medical problems. So I  
21 think it's absolutely very important.

22 Q In your practice, are you the physician that  
23 actually conducts that interview and collects that  
24 information?

25 A That is correct.

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1 Q And that's your standard practice.

2 A That's correct.

3 Q In listening to Dr. Rust's description of  
4 the development of Jordan King and William Mead, do  
5 you recall whether he ever heard the parents' history  
6 at any point?

7 A I believe that he testified that he had not  
8 been here when they testified, that he had not  
9 listened to the audio transcripts of their testimony,  
10 and that he had not read the written transcripts of  
11 their testimony.

12 Q And it's your testimony, is it, that having  
13 that information is, again, critical to assessing  
14 both the diagnosis but also the timing of onset of an  
15 autistic disorder?

16 A I believe that to be very important, yes.

17 Q There was a significant part of Dr. Rust's  
18 testimony that critiqued the care and treatment that  
19 Jordan King and William Mead got. Do you recall that  
20 testimony?

21 A I do.

22 Q And this was testimony that specifically  
23 took issue with some of the treating pediatricians',  
24 Dr. John Green's, medical intervention, as well as a  
25 general critique of the type of interventions that you

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1 do at ARI. Is that correct?

2 A That is correct.

3 Q Could you describe your response, just in  
4 general, to that critique, including the critique  
5 that, in large part, these interventions are not  
6 science based?

7 A I was disappointed with the way Dr. Rust  
8 handled that line of questioning because these are  
9 issues that I have studied in some detail in an effort  
10 to try to figure out how to help these children, and  
11 it seemed to me that he dismissed various  
12 interventions almost out of hand, saying things like,  
13 "There is no evidence that IVIG helps children with  
14 autism," and stating that as if it were a fact.

15 In reality, there is published science about  
16 that very fact, and so I think that if you want to  
17 state something like that, you should be more specific  
18 and say, "IVIG only helps certain children with  
19 autism," for example. So that's one objection I had.

20 The second objection I had is that he tended  
21 to go down this laundry list as if Dr. Green was  
22 trying to cure autism with these different  
23 interventions and almost as if he would try one thing  
24 and then move to something else if the first thing  
25 didn't work.

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1           It's a crucial distinction here to realize  
2           that John Green was taking care of his whole patient,  
3           and he was addressing the specific medical problems of  
4           the child. So it wasn't that he was moving from one  
5           supplement to another to another, hoping that  
6           eventually he would hit on something that would cure  
7           his autism.

8           He was following, I think, a very rational  
9           approach, given what he knew about the child and the  
10          interventions he had available to him, and looking at  
11          the risk/benefit ratios of those interventions with  
12          respect to the biochemical and the underlying medical  
13          problems that the child had.

14          For example, the most egregious example from  
15          Dr. Rust was when he was asked about valtrex, and he  
16          said something along the lines, and I'm paraphrasing  
17          here, I don't have any idea why that would be helpful  
18          in autism. That's used for genital herpes.

19          Well, I would like to demonstrate later that  
20          there is a very well-established biochemical reason  
21          that John Green would have considered that and that it  
22          has nothing to do with treating genital herpes in, you  
23          know, a two- or three-year-old little boy.

24          Then the third thing I would just like to  
25          say is that if Dr. Rust, you know, didn't know why we

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1 would use some of these things -- for example, he said  
2 that he has never heard of eskimo oil, so he doesn't  
3 understand why that would be helpful.

4 We live in the Internet age, and he can  
5 Google it and, within just a few moments, find out  
6 that that's a type of omega-3 essential fatty acids,  
7 and then, if you look at the literature on omega-3  
8 essential fatty acids, there is a broad amount of  
9 information in the literature that documents the value  
10 for that for immune regulation and for being able to  
11 help cell-to-cell communication and to help heal the  
12 lining of the intestine.

13 So, in general, that describes some of the  
14 issues I had with the way he handled that line of  
15 questioning.

16 Q You mentioned specifically that Dr. Rust  
17 characterized some of Dr. Green's interventions as  
18 attempts to cure autism. You described, generally, it  
19 was to address the whole patient.

20 What do you believe were some of the  
21 underlying medical conditions that William Mead and  
22 Jordan King had that were being addressed by the  
23 therapies that Dr. Green recommended and that you used  
24 in your own practice, again, from the perspective of  
25 the ones that are being used to cure autism but to

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1 treat the whole patient?

2 A Right. They both had evidence of chronic  
3 diarrhea and, I think, subtle signs of abdominal pain,  
4 and when we get a history from the parent that they  
5 have ongoing chronic diarrhea for as much as a year, I  
6 think we need to take that seriously and not just  
7 write it off to toddler's diarrhea in a child who is  
8 losing function and deteriorating before our very  
9 eyes.

10 Q So whether it's curing autism or not, it's  
11 to treat a significant medical condition, which is the  
12 chronic diarrhea.

13 A Right. And another thing that John Green  
14 was trying to address were abnormalities in  
15 methylation and transsulfuration biochemistry, and  
16 that was the underlying reason that he would choose  
17 things like certain B vitamins or methylcobalamin  
18 injections or folinic acid.

19 By examining the record, one can, at least,  
20 get reports back from the parents that the child  
21 seemed to improve when those interventions were  
22 undertaken.

23 When I had the opportunity to listen to  
24 Mylinda King's testimony here, and then also further  
25 interview her, she says that, even now, if she takes

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1 away his methylcobalamin injections or misses a day,  
2 she sees deterioration in his performance.

3 So I think that for the Special Masters to  
4 understand that it was Dr. Green's perspective in  
5 trying to treat those methylation abnormalities that  
6 would make his choices seem more reasoned and more  
7 rational.

8 SPECIAL MASTER VOWELL: Pardon me. Can we  
9 just take a moment and everyone check your electronic  
10 devices? I was handed a note that there is a little  
11 feedback coming through the system.

12 THE WITNESS: Special Master, I'm wondering  
13 -- I have two mikes in front of me. Do I need both of  
14 them, or is this the live one?

15 SPECIAL MASTER VOWELL: Yes. You need both.  
16 You need to keep a little bit of a distance. Okay.  
17 To proceed. Thank you.

18 BY MR. POWERS:

19 Q So, Dr. Mumper, you just mentioned this idea  
20 of the methylation cycle. Do you recall Dr. Rust,  
21 again, describing some of these interventions as  
22 having no basis in science?

23 A Yes.

24 MR. POWERS: I would like to put up on the  
25 screen, and, unfortunately, we don't have copies right

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1 now, but we'll have copies over the break, three  
2 slides that you prepared and brought with you today.

3 SPECIAL MASTER VOWELL: Tom, I have a set of  
4 copies, if that would be helpful.

5 MR. POWERS: If Respondent would like to  
6 take a look at -- it's not marked up, is it?

7 SPECIAL MASTER VOWELL: No.

8 MR. POWERS: Okay.

9 SPECIAL MASTER VOWELL: Well, if it's on the  
10 screen, I guess it's --

11 MR. POWERS: If it's on the screen, I think  
12 we can look at the screen, and, Counsel, we'll have  
13 copies for you on the break.

14 I should stop for a second. This would be  
15 Petitioners' Trial Exhibit 13? -- 14, I'm sorry.

16 SPECIAL MASTER VOWELL: It's going to be the  
17 three-slide component from Dr. Mumper.

18 MR. POWERS: Yes. That's correct. So it  
19 will be page 1, page 2, and page 3 of Exhibit 14.

20 (The document referred to was  
21 marked for identification as  
22 Petitioners' Exhibit No. 14.)

23 BY MR. POWERS:

24 Q So, Dr. Mumper, can you describe for the  
25 Special Masters what you have there as page 1 of

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1 Exhibit 14 on the slide?

2 A Yes. This is the methylation and  
3 transsulfurations biochemistry that Dr. Deth talked so  
4 much about, and this is the way that we teach it to  
5 doctors who are learning how to take care of children  
6 with autism.

7 Q Let me interrupt you for just a second.  
8 Now, did Dr. Deth actually prepare this chart, or is  
9 this something that somebody else prepared, or is it  
10 adapted from Dr. Deth?

11 A This is Dr. Jill Janes' slide, and then,  
12 several place on the third of these, I have made some  
13 notations about treatments that she did not put in but  
14 that are my notations, and I'll be sure to clarify  
15 those.

16 Q Okay. I just wanted to make it clear that  
17 this is not something taken from Dr. Deth's testimony  
18 so that the record here is clear that this is  
19 something that was prepared for you in your clinical  
20 work. Correct?

21 A That's correct.

22 Q Okay. I'm sorry for interrupting, but go  
23 ahead and explain again what this slide is.

24 A Basically, this is just showing, number one,  
25 is the folate cycle, and the five

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1 methyltetrahydrofolate at the bottom is converted up  
2 to tetrahydrofolate at the top, which is the active  
3 form of folate that's utilized in the body to support  
4 methylation reactions.

5 Q And that's the part of the diagram with a  
6 box that has the number one in it.

7 A That's correct.

8 Q Okay.

9 A I was just going to say that the "MS" in the  
10 little green box there is methionine synthase, which  
11 is the enzyme that Dr. Deth discussed that has that  
12 crucial role for helping make that conversion.

13 Q Okay. Now, let's move on to the portion of  
14 this slide that has a box with the number two in it.  
15 What is that?

16 A That is the methylation cycle where  
17 homocysteine is remethylated back up to make  
18 methionine. Methionine is a very important, essential  
19 amino acid, and it is converted to sand, which is  
20 S-adenosylmethionine.

21 That is the major methyl donor in the body,  
22 and so that allows you to assess methylation  
23 potential, and one of the values that's been very  
24 important for us to assess, as a result of Dr. James'  
25 work, is the SAM/SAH ratio and the fact that the

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1 proper balance of that needs to happen in order for  
2 methylation to continue properly. So this slide is  
3 simply setting the stage for a normal cycle.

4 Q And then the portion of the slide that has  
5 number three in a box next to it --

6 A -- is the transsulfuration cycle, and this  
7 process by which homocysteine is converted to reduced  
8 glutathione, which is the important kind, as opposed  
9 to oxidized glutathione, is what is labeled here as  
10 "anti-oxidant potential," and a lot of our  
11 interventions are designed to try to help the child  
12 make more reduced glutathione. So this is normal  
13 methylation and transsulfuration biochemistry with the  
14 folate cycle.

15 I might mention that this process is so  
16 important to nature that it's built in a couple of  
17 redundant mechanisms, the remethylation mechanism as  
18 well as the folate cycle, in order to supply methyl  
19 groups to make methylation happen.

20 Q So let's go to page 2 of Exhibit 14.

21 A This is also from Dr. Jill James, "The  
22 Effect of Oxidative Stress on Methionine  
23 Transsulfuration," and, basically, what this shows is  
24 the same cycle that I've shown you but with an idea of  
25 what happens to this very crucial cycle when children

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1 are under oxidative stress.

2 For example, they, therefore, are not able  
3 to remethylate their homocysteine, and they end up  
4 with lower levels of methionine to start. That leaves  
5 them with lower S-adenosylmethionine, and as these  
6 methyl transferase enzymes are trying to enable the  
7 child to methylate their DNA, their RNA, make  
8 proteins, make membrane phospholipids, make creatine,  
9 which is the power currency of the cell, and make  
10 neurotransmitter. If all of those reactions are  
11 inhibited, it's going to be very difficult for  
12 children to turn their genes on and off.

13 One of the things that I did find intriguing  
14 about Dr. Rust's testimony is that he talked about how  
15 methylation is such an important process and how being  
16 able to methylate genes is gene regulation in action.

17 We have a lot of concerns about that because  
18 we are concerned about the epigenetic effects of any  
19 toxin or environmental factor that would impact on the  
20 cellular biochemistry.

21 Q Okay.

22 A And then, ultimately, at the end, I just  
23 wanted to point out that the glutathione in the  
24 reduced form, which is the good guy, is down, and the  
25 glutathione in the oxidized version is up and that

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1 cysteine is depleted in this model.

2 Q And, again, to make it clear, your  
3 discussion here is not based on your personal  
4 expertise as a biochemist, is it?

5 A No.

6 Q It's not based on any expertise or original  
7 research you've done as a molecular biologist.

8 A No. This is based on my initial reading of  
9 the Jill James work, my sheer honor to get to work  
10 with her on some research projects, my having heard  
11 her explain the cycle in lectures that we mutually  
12 attended probably 10 to 20 times, and the way that I  
13 use it to impact on my clinical practice and the  
14 teaching of the doctors that we teach.

15 Q So let's talk about that final point, I  
16 guess, on Slide 3. Can you describe for the Special  
17 Masters what this chart represents?

18 A In this chart, and I have added some  
19 notations of my own here, I have tried to look at the  
20 interventions that physicians like John Green and I  
21 use to try to help these children, and to put it into  
22 context into this crucial pathway so that it becomes  
23 clear that we are trying to fit the intervention to  
24 the scientific profile, the metabolic profile, of the  
25 child.

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1 Q I want to direct your attention to a  
2 particular portion of this slide, then.

3 A Okay.

4 Q To the left, there is a note that you added  
5 that says, "M-B12 methylcobalamin."

6 A Right.

7 Q Can you describe for the Special Masters the  
8 significance of that notation that you made?

9 A Yes. One of the theoretic interventions  
10 that one could do to make the methylation cycle work  
11 better would be to give methylcobalamin, which is also  
12 called "M-B12," to help generate methyl donors. A  
13 methyl group is a carbon and three hydrogens and four  
14 methionines so that it can take it through the cycle.

15 Jill James' work actually looked at children  
16 with autism, compared them to controls, documented  
17 that the children with autism had low methionines, low  
18 cysteines, and low reduced glutathione. So she  
19 designed an intervention trial in which she would give  
20 substrates and nutritional interventions that would  
21 help that methylation cycle work better.

22 She used methylcobalamin, she used betaine,  
23 which is the same as TMG, and she used folinic acid.  
24 So you can see that all of those interventions are  
25 working on helping that remethylation cycle take

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1 place.

2 So in a university lab, using well-  
3 demonstrated scientific techniques, and, ultimately,  
4 published in peer-reviewed literature, she was able to  
5 demonstrate the normalization of those methylation  
6 metabolites in the children that she treated, to quite  
7 a dramatic extent, and that's why we cited her paper  
8 in my expert report.

9 So that is why Dr. Green, when he got  
10 laboratory evidence implying impairments in this  
11 methylation cycle, chose to use things like  
12 methylcobalamin and folinic acid, and sometimes we use  
13 TMG, and sometimes we use DNG.

14 Q Can you direct the Special Masters'  
15 attention to any other notations that you made on this  
16 slide indicating the type of treatments and therapies  
17 that you would use as a clinician and that you would  
18 teach to other doctors that you work with?

19 A Well, another thing that I think that's very  
20 interesting is, if you look at B-6 in magnesium, which  
21 is to the left of a circle that says "CBS," which  
22 stands for cystathione beta synthase, there are about  
23 22 studies in the medical literature that have shown  
24 efficacy of B-6 or B-6 plus magnesium, and these go  
25 back several decades.

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1 Dr. Rimland, at ARI, was noticing this many  
2 years ago, a couple of decades before Jill James' work  
3 was published. Now, I think, in retrospect, we can  
4 postulate that one mechanism by which those vitamins  
5 and minerals make a difference is in helping the body  
6 generate cysteine, which is this rate-limiting amino  
7 acid for glutathione production.

8 You'll recall from Dr. Deth's testimony a  
9 fair amount of information about how important it is  
10 for kids, when they are trying to detoxify substances,  
11 to be able to have adequate cysteine and make adequate  
12 glutathione in order to feed their detoxification  
13 pathways and how poorly their cellular biochemistry  
14 works when they have a decrease and reduced  
15 glutathione and an increase in oxidated glutathione,  
16 which is what the big pink box with the "GSSG" is  
17 showing, high oxidized glutathione.

18 So that's another nutritional intervention  
19 that ties specifically to this cycle.

20 Q Can you identify any other nutritional  
21 interventions that you believe provide scientific  
22 support for the clinical practices of yourself and Dr.  
23 Green?

24 A The other thing that I think that's very  
25 interesting is, on the sort of right-hand part of the

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1 slide where I've labeled "DPP-4, casein-free, and  
2 gluten free." This relates to some biochemistry  
3 related to adenosine, and if you'll bear with me for a  
4 few moments, I will ultimately take you back to the  
5 valtrex issue that Dr. Rust was asked about.

6 Adenosine, in the pink box there, when it is  
7 elevated, as we have documented, per Dr. James' work,  
8 seems to be the case in about 20 percent of children  
9 with autism, there is a feedback loop that makes it  
10 have an adverse effect on S-adenosylhomocysteines such  
11 that that builds up. That leads the children to be in  
12 a situation where they have an abnormal SAM/SAH ratio,  
13 where the SAH part is too high, and the SAM part is  
14 too low.

15 By negative feedback, the effect of that is,  
16 once again, that they are not able to methylate their  
17 DNA. Remember the concerns we have about effects on  
18 gene regulation and gene expression when that's the  
19 case. They are not able to make proteins adequately.  
20 They are not able to make their phospholipid membranes  
21 nor their creatine, which is the power currency of the  
22 cell.

23 So another potential target for adenosine  
24 intervention, for interventions to work on helping  
25 methylation function well, is to try to get the

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1 adenosine down when it's too high.

2 So, in our group at Defeat Autism Now, one  
3 clinician noticed that a mother who was on valtrex had  
4 given her child valtrex three different times -- given  
5 it, taken away, given it, taken away, given it, taken  
6 away -- and there was always a challenge-rechallenge  
7 effect, where the child started speaking when he was  
8 on the valtrex and regressed when he was off of that.

9 So we took that anecdotal experience as a  
10 reason to look into the biochemistry, and Dr. Baker  
11 and Dr. James ultimately did a study in which they  
12 looked at adenosine levels and found that, in the kids  
13 who had high adenosine levels and were given a  
14 acyclovir, which valtrex is broken down to acyclovir,  
15 that the adenosine levels normalized, and as the  
16 levels normalized, the children improved, in terms of  
17 their speech, language, communication, and social  
18 reciprocity.

19 So this is a situation in which we are  
20 looking at valtrex not as treating genital herpes in a  
21 two-year-old but as in trying to, in a very  
22 fundamental way, correct this very important, cellular  
23 biochemistry.

24 Now, if you'll bear with me for a minute,  
25 there is something called "adenosine deaminase binding

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1 protein" that is important for the adenosine deaminase  
2 enzyme to work appropriately on adenosine. That  
3 binding protein, we know, is impaired with heavy  
4 metals, including mercury, and that was shown in 1982  
5 by a scientist called Pershell. I believe he was from  
6 Germany, but I'm not positive about that.

7 The binding protein for ADA, DPP-4, which I  
8 told you last time was dipeptidylphosphatase 4, which  
9 is that enzyme that works on gluten and casein, and a  
10 lymphocyte called CD-26 are all essentially the same  
11 thing, and this is very confusing initially. But the  
12 point is --

13 MR. MATANOSKI: Your Honor, I'm going to  
14 object at this point. At the outset of this, I  
15 thought there was going to be some rebuttal. I  
16 thought it was in the form of criticism about  
17 treatment.

18 At this point, we're way beyond any  
19 qualified testimony on these matters, these charts,  
20 everything that's going on now about Dr. Deth's  
21 pathways. I would just say, can we move on to  
22 something that's actually rebuttal to this case  
23 specific instead of something that the witness is not  
24 qualified to know.

25 SPECIAL MASTER VOWELL: Mr. Powers?

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1 MR. POWERS: We are getting very close to  
2 the conclusion of this line of questioning, but it's  
3 appropriate on rebuttal because a fundamental topic of  
4 Dr. Rust was that Dr. Green, the treating physician,  
5 as well as Dr. Mumper in her approach to these cases  
6 generally, are not relying on science for the  
7 treatments and the interventions. Dr. Mumper is  
8 detailing her reliance on the science explanation.

9 MR. MATANOSKI: Which we would submit she is  
10 not qualified, or has not been qualified, to explain  
11 how this could happen, how she could rely on this.  
12 She is a pediatrician.

13 SPECIAL MASTER VOWELL: Dr. Mumper has  
14 stated that she is not testifying as a biochemist and  
15 has no expertise in that area, and your comment, Mr.  
16 Powers, to the last one, do you have any further  
17 comment?

18 MR. POWERS: No further comment.

19 SPECIAL MASTER VOWELL: We are at the end of  
20 the casein-free, gluten-free matter.

21 MR. POWERS: Yes. We're about to move off  
22 of this slide.

23 THE WITNESS: I will move off the slide just  
24 to say that the reason for trying to show the  
25 chemistry here was Dr. Rust said that he was never

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1 aware of any children that had benefitted from a  
2 gluten-free, casein-free diet.

3 I think this is one of the scientific bases,  
4 that there is a subset of children that improved, and  
5 I am very surprised that, in his population of many  
6 hundreds of children, he has not seen improvements in  
7 at least a subset.

8 BY MR. POWERS:

9 Q Now, we'll take that slide down, and, Dr.  
10 Mumper, you just expressed surprise that Dr. Rust  
11 hasn't seen improvements related to the gluten-free,  
12 casein-free diet. What is your experience, as a  
13 clinician with your own practice, as well as somebody  
14 who is working with a network of doctors, what is your  
15 opinion on the efficacy of the diet?

16 A We tend to recommend the diet based on a  
17 clinical picture in which we have some history of the  
18 child either craving dairy or craving gluten or  
19 otherwise deteriorating when they eat these foods.

20 Our best clinical estimates, when we ask  
21 people to try the diet, is that about 30 percent of  
22 the children will improve dramatically, another 30  
23 percent will have some significant improvements, and  
24 there is probably about 30 percent or so who do not  
25 seem to improve where that is not part of their

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1 pathology.

2 But, in this situation where the parents  
3 were reporting chronic diarrhea, I think it is an  
4 entirely reasonable thing to do, and when we use those  
5 diets, we're careful to supplement calcium. So I  
6 think that, in given situations, there is rational  
7 reason to use those diets in children with autism.

8 Q Now, you were describing your reliance on  
9 peer-reviewed, published literature, as well as  
10 materials prepared by people like Dr. James, a little  
11 while ago. Are you relying on any other review or  
12 compilation of scientific literature beyond what you  
13 just described in the slides?

14 A Well, we are constantly upgrading our  
15 bibliographies of scientific articles. I've got one  
16 now that I'm tasked to review that is looking at five  
17 or six different categories. So, yes, we try to look  
18 at the whole literature. We're specifically interested  
19 in autism in the gut, autism in the metabolic  
20 pathways, autism and immune dysregulation, and autism  
21 as relates to detoxification.

22 Q And when you say "we," who are you referring  
23 to?

24 A The scientists, researchers, and clinicians  
25 associated with the Autism Research Institute.

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1 Q Now, Dr. Rust described a concern that, in a  
2 lot of these care and treatment interventions, there  
3 seems to be an absence of controlled clinical trials  
4 and an absence of placebo controlled clinical trials,  
5 in particular, you know, the double-blind, crossover  
6 placebo studies. Do you recall that testimony?

7 A Yes, I do.

8 Q And that that was a criticism of your work  
9 and that because of the lack of that evidence, he  
10 found that the care and treatments that you  
11 recommended as being ineffective and not based in  
12 science. Do you recall that?

13 A I do.

14 Q How would you respond to that point that Dr.  
15 Rust made?

16 A I will acknowledge that we need many, many  
17 more placebo-controlled, double-blind studies, but  
18 we're very concerned about only using that model. Our  
19 paradigm is that these children have multiple medical  
20 problems and that if you are not careful when you pick  
21 your placebo-controlled trial, if you have a very  
22 heterogeneous constellation of children with multiple  
23 medical problems, you may stumble upon something that  
24 does not look as if it will be helpful, even though  
25 it's helpful for a subset.

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1 I think the best example of that is a  
2 secretin study, which Dr. Rust referred to, expressing  
3 surprise that William Mead had gotten secretin, saying  
4 that the gold standard study had shown it was not  
5 efficacious, but I have two points to make about that.

6 One is, if you actually look at the Herlihy  
7 study, there were clear responders who did  
8 dramatically well, and then there were a lot of other  
9 patients who did not do well with secretin. We  
10 discussed this at length in the think tank, and the  
11 scientists from the ARI that were involved in that  
12 study were concerned from the beginning that the  
13 population was too heterogenous.

14 So in kids who had certain kinds of gut  
15 symptoms, there were several of the children that got  
16 dramatic responses, and if you'll recall from William  
17 Mead's laboratory data, he had pretty significant  
18 laboratory findings in which, at Harvard Hospital, his  
19 pancreatic enzymes were shown to be dramatically low  
20 and then shown to improve dramatically after a  
21 secretin infusion. That's Exhibit 15, pages 51 and  
22 52.

23 So, again, for Dr. Rust to just paint a  
24 broad brush that secretin is not useful in a placebo-  
25 controlled study and, therefore, can't be useful in

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1 this particular patient, who clearly shows a need for  
2 it, I think, reflects a very superficial understanding  
3 of the importance that we place on taking care of the  
4 individual patient based on their individual problems.

5 Q Now, in response to the criticism that there  
6 are no clinical trials and no placebo trials, you, in  
7 your practice, or you, in your role with ARI, are you  
8 endeavoring to conduct such trials?

9 A Yes, we are.

10 Q Can you describe, very briefly, for the  
11 Special Masters what type of trials you are planning,  
12 either that are underway or that are planned to start  
13 soon?

14 A Well, we've submitted grants for a placebo-  
15 controlled, double-blind crossover on diflucan. We've  
16 submitted, for looking at trying to work with the NIH  
17 on a chelation study, looking at DMSA probably  
18 initially.

19 We're trying to do what are called "single-  
20 subject, multiple-baseline studies," where we can take  
21 a single subject and do lots of initial measurements  
22 and then do interventions and measure their response  
23 so that we can deal with the issue of the fact that  
24 different biochemistry and different medical problems  
25 in a single child may need a certain constellation of

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1 interventions.

2 That's used very widely in behavioral  
3 psychology, and we are working on adapting that to the  
4 medical model, working with a guy named Ted Carr, who  
5 is a behavioral psychologist, well published, who  
6 wants to do some initial studies in that model with  
7 gut disease in my clinic at the Rimland Center.

8 Q Now, the last issue that Dr. Rust raised  
9 that I wanted to discuss with you, you sort of touched  
10 on a second ago, and that is chelation. Do you recall  
11 his testimony that chelation is harmful, or  
12 potentially harmful, potentially fatal, that it's  
13 painful, and that he didn't understand how it could  
14 possibly have any efficacy in treating a disorder that  
15 one would postulate is caused by inorganic mercury and  
16 inflammation?

17 A Right.

18 Q Do you recall that testimony?

19 A Yes.

20 Q How would you respond, for the Special  
21 Masters, to that particular critique on the chelation  
22 issue that Dr. Rust raised?

23 A I have a couple of thoughts. One is to  
24 point out that chelation is a well-recognized and  
25 widely used pediatric modality in children that have

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1 blood poisoning and lead toxicity, and, in many of the  
2 children we treat who show mercury in their chelation  
3 urines, they also show evidence of lead.

4 So we think that it's important to go after  
5 the lead, and we've had many discussions at ARI about  
6 how we're just as concerned, if not more so, about  
7 lead than mercury in many of these children because,  
8 even though we took lead out of paint and gasoline, we  
9 just found out, a year or so ago, that we put it in a  
10 bunch of toys we got from China, and so they are still  
11 being exposed to lead.

12 With regard to the dangers and the  
13 fatalities, I would like to comment that when both Dr.  
14 Green and I use chelation in our offices, it's  
15 primarily oral chelation, and we tend to use blood  
16 count monitoring for complete blood counts and  
17 chemistry screens very four to eight weeks, and so  
18 that's why you saw some white counts in chemistry  
19 screens in those boys' charts.

20 When IV is used, John Green is one of the  
21 ones that has a vast amount of experience with that.

22 The death that Dr. Rust was referring to was  
23 actually a pharmaceutical error in which sodium EDTA,  
24 not calcium EDTA, was given to the boy, who died. We  
25 would expect that if the child was given sodium EDTA,

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1 it would have horrible consequences on his calcium and  
2 probably lead to asystole, which is probably how that  
3 child died. But that does not paint all of chelation  
4 as being dangerous or potentially fatal. That was a  
5 pharmaceutical error.

6 Q Now, Dr. Rust also just raised the question,  
7 or made the statement, that he couldn't understand how  
8 chelation could possibly have any efficacy,  
9 particularly since the theory in these cases is that  
10 there is inorganic mercury in the brain that,  
11 obviously, chelation is not going to bring back out of  
12 the rain across the blood breaker. Do you recall his  
13 comments on that issue?

14 A I do.

15 Q How do you think it is that chelation could  
16 possibly assist in the treatment of the symptoms of  
17 children that you see?

18 A A lot of work remains to be done in this  
19 area, but we are able to mobilize mercury, lead, and  
20 other toxins from where they are hiding. Typically,  
21 mercury hides in the brain and in the kidneys and in  
22 the liver and in the fat, and we know that we're not  
23 typically removing anything from the brain, but, by  
24 working on the rest of the body burden and taking off  
25 the chronic stress that mercury provides, we're

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1 enabling cysteine to be regenerated, and we're  
2 enabling glutathione to function more productively,  
3 and we are eliminating some heavy metal burden by  
4 doing the chelation.

5 It's also entirely possible that some of the  
6 chelating agents are working by an antioxidant  
7 mechanism. For example, DMSA is a good anti-oxidant,  
8 and so sometimes we wonder if we are actually  
9 achieving an anti-oxidant rather than a chelating  
10 effect.

11 I will say that our preference is to try to  
12 mobilize the body's own mechanisms to do a natural  
13 form of chelation, so that's why we promote working on  
14 the methylation biochemistry and the nutritional  
15 support as a crucial component and not relying just on  
16 chelation.

17 Q Now, finally, Dr. Mumper, I want to move  
18 away from the specific-treatment discussion that Dr.  
19 Rust engaged in and that you've now replied to.

20 Do you recall Dr. Rust saying that he  
21 reviewed videotape of both Jordan King and William  
22 Mead?

23 A Yes, I do.

24 Q And do you recall Dr. Rust saying that, upon  
25 his review of the video that he watched, he thought

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1 that both boys were abnormal before they actually  
2 regressed?

3 A Yes.

4 Q Do you recall him citing to any specific  
5 portions of video in his testimony in support of his  
6 conclusion?

7 A I do not recall that he did.

8 Q Okay. Have you identified specific portions  
9 of video that you think are responsive to Dr. Rusks's  
10 characterization of the preregressive symptoms of both  
11 Jordan and William?

12 A Yes. I tried to do that after taking notes  
13 on his testimony.

14 Q Now, you had an opportunity to review the  
15 video -- I think you described this on your direct  
16 testimony -- but you reviewed the video before Dr.  
17 Rust testified. Correct?

18 A Right.

19 Q Are you saying now that you reviewed the  
20 video again after you heard Dr. Rust testify?

21 A Yes. When I reviewed the videos the first  
22 time, I took extensive notes about the ages of the  
23 child and things that they were doing that either  
24 appeared age appropriate to me or not.

25 The second time when I went through, I was

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1 trying to look at specific criticisms or suggestions  
2 that had been made that the boys had deficits and  
3 address specific criticisms about they must not be  
4 talking, they must not have gestural language, they  
5 must not have social reciprocity, those kinds of  
6 things, and I tried to find very short clips that  
7 would demonstrate it. I think the whole total of each  
8 child is less than 10 minutes or so.

9 MR. POWERS: So I'll interrupt asking  
10 questions of you and just address the Special Masters.  
11 We do have video that we're going to show. We have  
12 done what is essentially an index of the video.  
13 Jordan King would be the next exhibit, I guess, 15?

14 SPECIAL MASTER VOWELL: Yes.

15 MR. POWERS: And William Mead's would be 16.

16 (The documents referred to  
17 were marked for  
18 identification as  
19 Petitioners' Exhibit Nos. 15  
20 and 16.)

21 MR. POWERS: What we will do, and propose  
22 doing, is that we will take the video clips that are  
23 going to be shown here today, put those onto one  
24 compact disk, and have an electronic index in that  
25 disk that allows one to match up what you're about to

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1 see on this piece of paper with the contents of the  
2 CD, and we'll file that as soon as we can get that  
3 produced.

4 If that sounds sufficient to the Court,  
5 that's how we propose proceeding, and, obviously,  
6 providing copies to counsel.

7 SPECIAL MASTER VOWELL: That's fine.

8 MR. POWERS: So before showing the video,  
9 I'm just going to take a moment and pass out to the  
10 masters and to Respondent's counsel Exhibits 15 and  
11 16.

12 SPECIAL MASTER CAMPBELL-SMITH: Thank you.  
13 Also, counsel, without disturbing your plan for how  
14 you plan to proceed, but to the extent that Dr. Mumper  
15 could lay some groundwork before we look at the video  
16 as to why she picked this particular clip, what we  
17 should be paying attention to.

18 THE WITNESS: Right.

19 SPECIAL MASTER CAMPBELL-SMITH: Because  
20 without your sort of guidance even beforehand, we  
21 might get it after, but subtleties are certainly lost  
22 if we don't have a sort of a preview before we get  
23 into it.

24 MR. POWERS: And that's what we anticipated  
25 doing here, both a little setting some context and

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1 then any description of what is actually seen.

2 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

3 MR. MATANOSKI: Your Honor, just for the  
4 record, as you know from your prior orders that this  
5 designation was exactly what the Court had asked for  
6 prior to trial. The Respondent gave you the  
7 designation of the particular points of the videos  
8 that the Respondent will be looking at or relying on.  
9 Petitioners declined to do that, instead saying that  
10 they would have to wait, they were just going to  
11 essentially rely on the entire video and then  
12 designate later what they were going to rely on.

13 And with respect to replying to this, we  
14 will rebut what we can today, if necessary, however we  
15 reserve the right to designate or counterdesignate  
16 other parts of the video later for your review in a  
17 similar fashion to see -- what you saw from us before  
18 where we designated certain portions that we could be  
19 potentially relying on.

20 MR. POWERS: And Petitioners, as you also  
21 know, made it clear that we didn't designate anything  
22 early on because we would not anticipate relying on  
23 that in our case-in-chief. The designations now we're  
24 in rebuttal it's impossible to designate ahead of time  
25 what one might use in rebuttal because you haven't

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1 heard the testimony of the witness that you might be  
2 rebutting. So these are offered in rebuttal. And if  
3 Respondent is saying they would want to reserve the  
4 right to designate more video, then if the record is  
5 open in these cases and more designations are needed  
6 and you want to see more information from video we are  
7 happy to do that and designate whatever the Special  
8 Masters think they need to see to get the full picture  
9 of the video.

10 SPECIAL MASTER CAMPBELL-SMITH: I would ask  
11 that, Dr. Mumper, as you go forward with this that you  
12 make clear in your preliminary comments what portion  
13 of Dr. Rust's comment to which you are specifically  
14 addressing.

15 THE WITNESS: Okay.

16 SPECIAL MASTER CAMPBELL-SMITH: You  
17 indicated you had taken very careful notes.

18 THE WITNESS: Right.

19 SPECIAL MASTER CAMPBELL-SMITH: And you were  
20 trying to respond to concerns about that.

21 THE WITNESS: Okay.

22 SPECIAL MASTER CAMPBELL-SMITH: So if you  
23 could make that clear as to what portion of Dr. Rust's  
24 testimony to which you are responding that would be  
25 very helpful.

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1 THE WITNESS: Okay.

2 MR. MATANOSKI: And then if I may, just to  
3 clarify our position in this, we designated, our  
4 experts designated certain parts of the record, the  
5 video record that they would be relying on, provided  
6 that to the Court and to opposing counsel. During  
7 their testimony they did not refer to any other parts  
8 of the record, the video record, so these designations  
9 that Respondent has were well available to the  
10 Petitioners in advance. The notion that they are  
11 rebutting something other than that is a bit strange  
12 at this point since neither witness that referred to  
13 videotapes actually referred to any specific part  
14 other than the ones that have been designated.  
15 Indeed, they didn't even refer to those. But those  
16 had been designated already.

17 So the notion that rebuttal would come in  
18 now without prior designation is again a bit strange.  
19 And that is the reason why we'd ask that some relief,  
20 if necessary be given.

21 SPECIAL MASTER CAMPBELL-SMITH: I think that  
22 request has been granted Respondent.

23 MR. MATANOSKI: Thank you.

24 SPECIAL MASTER CAMPBELL-SMITH: That to the  
25 extent that you need or want to counter with

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1 additional video testimony that we are certainly  
2 willing to entertain that.

3 MR. MATANOSKI: Thank you, Ma'am.

4 SPECIAL MASTER CAMPBELL-SMITH: To proceed.

5 MR. POWERS: Thank you.

6 So and, Dr. Mumper, before I ask about the  
7 first thing I want to make sure we are technologically  
8 ready to go. Okay.

9 BY MR. POWERS:

10 Q Excuse me. Now, we are going to talk about  
11 Jordan King first. You designated segment number one,  
12 which you have entitled "cooing"?

13 A Right.

14 Q Can you explain to the Special Masters why  
15 the little segment about 30 seconds long that they are  
16 going to see is significant in responding particularly  
17 to Dr. Rust's testimony?

18 A There was discussion in Dr. Rust's testimony  
19 about non-verbal language and other measures to  
20 communicate that did not involve actual words. And it  
21 was related to the, later to the topic of word count.  
22 And so this is an early language marker. The child in  
23 this video is about 3 months of age. And by showing  
24 it I show normal language development at that time  
25 plus a to and fro reciprocal relationship with the

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1 mother who is cooing with the child.

2 Q Okay.

3 SPECIAL MASTER HASTINGS: Now wait a minute.  
4 I'm not sure I understand this exhibit. Part one you  
5 taped 12-98. What does taped 12-98 mean?

6 THE WITNESS: That's a date, December '98.

7 SPECIAL MASTER HASTINGS: So this is, it is,  
8 means December '98.

9 Now you just said age 3 months. And I have  
10 it Jordan King, born September 29, 1997. Is that not?

11 THE WITNESS: Is that a typo?

12 MR. POWERS: Yeah, no, it's not the date  
13 it's just the title of the tape.

14 SPECIAL MASTER HASTINGS: All right, the  
15 title of the tape.

16 MR. POWERS: It's just it is a number that  
17 is the title of the tape but it's not a reference to a  
18 date.

19 SPECIAL MASTER HASTINGS: It's not a  
20 reference to the date of the tape.

21 MR. POWERS: Right.

22 SPECIAL MASTER HASTINGS: All right.

23 THE WITNESS: Okay, Tom, in that tape we had

24 --

25 SPECIAL MASTER HASTINGS: Let me also ask,

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1 again what did Dr. Rust particularly say about the  
2 word development that you are trying to rebut here?  
3 If you can before we go into this on Jordan King, tell  
4 me what in general he said about Jordan King that you  
5 are taking issue with?

6 THE WITNESS: The concern was that he talked  
7 about how he thought it was a artificial distinction  
8 between regressive and classic autism and that he  
9 thought if you really look carefully and ask careful  
10 questions you'd find out that the child were not  
11 initially normal but that they had subtle signs of  
12 abnormality. And when I looked at these tapes as a  
13 pediatrician I thought that the things that we picked  
14 out to show showed some very normal developmental  
15 milestones both for non-verbal language, gesturing, as  
16 well as social reciprocity, as well as appropriate toy  
17 play initially. And --

18 SPECIAL MASTER HASTINGS: All right, let me  
19 stop you there.

20 THE WITNESS: Yes.

21 SPECIAL MASTER HASTINGS: Certainly lots of  
22 experts in this proceeding have said when you look at  
23 people who are said to have regressive autism and you  
24 look back, study videos, you'll find evidence of  
25 abnormality. That in general has been said.

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1 Help me with my memory, to what extent did  
2 Dr. Rust say that about Jordan? Did he point to  
3 specific evidence of abnormality in Jordan?

4 THE WITNESS: Yeah, in --

5 SPECIAL MASTER HASTINGS: To the best you  
6 remember.

7 THE WITNESS: Yeah. On the basis of the  
8 notes that I took which I -- it's on page 25 of my  
9 notes, so that's about a little over halfway through  
10 his testimony, but I don't have the clarity to know if  
11 that was specific for Jordan King or not. So maybe if  
12 I can't do that, and we're not allowed to show the  
13 video, just tell me what the rules are.

14 SPECIAL MASTER HASTINGS: I'm not going to  
15 stop you from --

16 THE WITNESS: Yeah.

17 SPECIAL MASTER HASTINGS: -- showing any  
18 videos, I just want to understand what point you are  
19 trying to refute here because I don't, I'm not sure I  
20 recall.

21 Mr. Powers, do you understand what point you  
22 are trying to refute here?

23 MR. POWERS: Yes. Certainly as Dr. Mumper  
24 described it was general testimony that Dr. Rust  
25 offered that in his opinion relying on video that he

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1 reviewed, and certainly without any reference to  
2 specific frames, that Jordan King and also William  
3 Mead were not normal prior to their regression.

4 SPECIAL MASTER HASTINGS: Okay.

5 MR. POWERS: And again, he did that without  
6 reference to specific frames but definitely was  
7 placing the onset of symptoms, or conversely the  
8 absence of normalcy, further and further back in time.  
9 So these are simply offered to show in that time  
10 continuum that describes the onset what Dr. Mumper has  
11 identified from her skill and experience and training  
12 as indications that Dr. Rust was either mistaken or  
13 was not looking at the appropriate signs. And that's  
14 her approach here.

15 SPECIAL MASTER HASTINGS: And I do think he  
16 said some general comments to that effect. I couldn't  
17 remember any specific comments. And okay, so very  
18 good.

19 THE WITNESS: I actually have found my notes  
20 now that are specific to Jordan King. I have that he  
21 said he had looked at the record regarding the timing  
22 of loss of speech. And then he had a discussion about  
23 the fact that it's not so much the number of words but  
24 it's important that he was communicating and then  
25 stopped talking.

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1           And then he also said in the record it said  
2 something like he was never a "I want to be held"  
3 baby. And he always takes that very seriously.

4           And in the cooing video even though he is  
5 not being held there is a social reciprocity that  
6 speaks to social interactions that I thought would be  
7 valuable.

8           SPECIAL MASTER HASTINGS: Very good. Please  
9 go ahead.

10          MR. POWERS: Thank you.

11          BY MR. POWERS:

12          Q     So let's go ahead and show what is  
13 designated on Jordan King's video clip index as Video  
14 Segment Number 1.

15                   (Jordan King Video Clip No. 1 played.)

16          A     So the good eye contact, the social  
17 reciprocity with the mother, and the fact that he is  
18 doing the appropriate language for a 3-month-old baby.

19          Q     Let's move to Clip Number 2 please, Dr.  
20 Mumper. Can you give the Special Master some context  
21 for Clip Number 2 briefly?

22          A     One of the frustrations in looking at these  
23 clips is that the timing wasn't clear, the actual  
24 dates did not show up on Jordan King. So the videos  
25 that I am going to show next I can tell you are

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1 between the ages of 13 and 16 months but I can't tell  
2 you specifically how old the child was. We can get  
3 some clues perhaps from the progress of his  
4 development.

5 But that seemed to be a critical time in  
6 which Dr. Rust was saying that already, you know, the  
7 child was showing signs of autism, impaired showing  
8 language improvement or failure to progress, loss of  
9 social reciprocity. And so I wanted to address some  
10 areas in which he seems to be demonstrating age-  
11 appropriate normal behavior in that time period that  
12 was questioned.

13 SPECIAL MASTER HASTINGS: Now, when you got  
14 the tape, you just referred to a time frame of  
15 sometime between 13 and 19 months of age. Is that  
16 what you --

17 THE WITNESS: Yes.

18 SPECIAL MASTER HASTINGS: -- just said?

19 THE WITNESS: To the best of my ability to  
20 interpret the tape, the next one, two, the next series  
21 of tapes are in that time frame.

22 MR. POWERS: But was it 13 to -- I think Dr.  
23 Mumper said 13 to 16 months.

24 SPECIAL MASTER HASTINGS: Okay. Well, I  
25 wanted to inquire where you got that? Because now in

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1 Exhibit, Trial Exhibit 15 here it's been identified as  
2 this is the tape from 1999, January to June. Now, on  
3 my calculations that would be 15, 15 to 21 months. So  
4 I want to know where you got the idea of 13 to 16?

5 THE WITNESS: Okay. In my original dating  
6 of the one that's marked "Playing Marimba" the date is  
7 October '98 to January '99.

8 SPECIAL MASTER HASTINGS: Right. Right, I  
9 see that.

10 THE WITNESS: Which is 13 to 16.

11 SPECIAL MASTER HASTINGS: So that's the 13  
12 to 16. All right.

13 THE WITNESS: I'm sorry. The "Drop the  
14 Objects, Smile at the Camera" would be --

15 SPECIAL MASTER HASTINGS: Anyway, I think  
16 you've answered my question.

17 THE WITNESS: Yeah.

18 SPECIAL MASTER HASTINGS: You're getting the  
19 date just from the dates of the tape. So the tape was  
20 marked January through June of 1999.

21 THE WITNESS: Right.

22 SPECIAL MASTER HASTINGS: And you're just  
23 getting the dates from that?

24 THE WITNESS: Right.

25 SPECIAL MASTER HASTINGS: All right.

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1 MR. POWERS: And not to delay actually  
2 seeing the video, but just as a technical matter the  
3 way that the parents maintained these on 4-hour VCR  
4 tapes. And so 4 hours of tape would have -- this is  
5 the date range they wrote on there. In many cases  
6 there's not a stamp on the film itself.

7 SPECIAL MASTER HASTINGS: I understand.

8 MR. POWERS: So that explains some of the --

9 SPECIAL MASTER HASTINGS: My videotapes at  
10 home are marked exactly the same way.

11 MR. POWERS: Okay.

12 BY MR. POWERS:

13 Q So, Dr. Mumper, let's go ahead and show Tape  
14 Number 2, please.

15 A So I think we've established the child's  
16 actually older here per Special Master Hastings.

17 (Jordan King Video Clip No. 2 played.)

18 So what I wanted to demonstrate there was  
19 the child dropping the toy and then looking to see  
20 where it went has to do with the establishment of a  
21 concept called object permanency and making the  
22 connection in his brain that when you do something  
23 with an object and it goes out of site that it  
24 continues to exist beyond what you see.

25 It also can be interpreted as processing

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1 cause and effect.

2 And then I also wanted to show that in that  
3 age range above 16 months he was still smiling and  
4 having social reciprocity with whoever was running the  
5 video camera.

6 Q Now Clip Number 3 please. This is one that  
7 is called "Playing the Marimba."

8 A And now, Tom, I believe we are back to the  
9 somewhere in the 13 to 16 month age range now. And  
10 this is looking at reciprocity in terms of play with  
11 another person and being able to socially interact in  
12 a musical game.

13 (Jordan King Video Clip No. 3 played.)

14 Q Okay.

15 A And he was also looking around at the  
16 videographer again in that.

17 Q Clip Number 4, "Playing with the Cat."  
18 Let's go ahead and cue that up, please. And can you  
19 describe what the Special Masters ought to have an eye  
20 out for here?

21 A Yes. In this situation it seemed like very  
22 appropriate interactive play with an animal and with  
23 the grandmother.

24 Q Okay.

25 (Jordan King Video Clip No. 4 played.)

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1 A And again he looked at the camera.

2 Q Clip Number 5, quick context? And I think  
3 we've got our process down for going through these.

4 A Yes, right.

5 Q So if you could provide some context here  
6 for the Special Masters and then we'll show the clip?

7 A One of the discussions in testimony was  
8 about not being able to use gestures, that it wasn't  
9 just language but that children also had gestures that  
10 were postulated to be absent or of poor quality in  
11 these children. And this is demonstrating gesture to  
12 be picked up, which is typically around, emerges  
13 around 9 months of age as a skill.

14 (Jordan King Video Clip No. 5 played.)

15 And again still making eye contact with the  
16 people in the scene.

17 Q Okay. What's been designated as Clip Number  
18 6 called "Dancing" I'm guessing is Jordan dancing.  
19 But if you could provide again some context for the  
20 Special Masters?

21 A Showing ability to enjoy play, ability to  
22 interact and look at the person who is filming him.  
23 You will see when you see the tape that he is very  
24 engaged.

25 (Jordan King Video Clip No. 6 played.)

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1 Q Okay. Now, the next one it's called  
2 "Toolbench," and this is Clip Number 7.

3 A In this it demonstrates his ability to use  
4 tools in a functional way and an appropriate way to  
5 play as opposed to lining up toys or playing with them  
6 in an inappropriate way.

7 (Jordan King Video Clip No. 7 played.)

8 Q And is that Maya, his sister?

9 A That's Maya, his sister. So we know that he  
10 is at least 15 months old in this video.

11 (Jordan King Video Clip No. 7 playing.)

12 Q This is from the -- that was on the original  
13 tape.

14 Now, Clip Number 8 is called "Harmonica."  
15 Again quick little context for the Special Masters and  
16 we'll play that clip?

17 A Showing the social reciprocity between him  
18 as he plays a harmonica and the other people in the  
19 room, showing interactive play.

20 (Jordan King Video Clip No. 8 played.)

21 Q And actually if we could stop it there for  
22 just a quick second. Jordan was shown being held. Do  
23 you recall Dr. Rust making comments that one of the  
24 things that he thought might have been going on with  
25 Jordan early on was an aversion to touch and an

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1 aversion to being held?

2 A Yes, I do recall that. And, in fact, there  
3 are many, many, many examples in the video I reviewed  
4 where he was being held by various people,  
5 grandmother, mother and father.

6 Q Okay. Let's go ahead and complete rolling  
7 this clip please.

8 (Jordan King Video Clip No. 8 played.)

9 A So he perked up when he was told he had a  
10 good job. And so he was responding to the mother  
11 there. He clearly was smiling brightly and  
12 interacting. And so at that point he was also showing  
13 gestural language.

14 Q Now, you also, tell me if you did, recalled  
15 Dr. Rust saying that Jordan had splinter skills?

16 A Right.

17 Q You have to stay back from the microphone.  
18 What was his description of splinter skills  
19 relative to Jordan King?

20 A Well, I believe he was postulating that he  
21 had musical abilities. And so between the fact that  
22 he came from a musical family and had demonstrated his  
23 work with the harmonica and marimba he may have been  
24 referring to that. Whether it's a true savant skill,  
25 you know, time would tell.

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1 Q But at this point there's nothing savant-  
2 like -- I mean not to denigrate Jordan's harmonica  
3 playing -- but there is nothing savant-like that you  
4 would identify in any of the musical sequences? And I  
5 say it jokingly, but since Dr. Rust did mention this,  
6 there is nothing savant-like that you've identified in  
7 any of the musical scenes here involving Jordan, is  
8 there?

9 A Yeah, I would say that was very rudimentary,  
10 age-appropriate for a toddler harmonica playing.

11 Q Okay.

12 A Yes.

13 Q We're going to go to Video Clip 9 then,  
14 please. And this is "Building a Marimba."

15 A And the thing to look for here is his  
16 ability to use a nail in a functional way and to  
17 imitate his father trying to put a nail into a hole  
18 and repeatedly getting it out of the bag, the nail  
19 bag.

20 (Jordan King Video Clip No. 9 played.)

21 Q And so demonstrating the nail but did you  
22 see anything else in that video that would be  
23 significant, any interactions with his dad or anything  
24 of particular mention?

25 A Yeah, he was looking back and forth for

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1 approval from and interaction with his father.

2 Q And then we're going to show Clip Number 10.

3 A And the reason I chose this is that there  
4 was some speculation about Jordan withdrawing around  
5 the time of the birth of his sister and being  
6 withdrawn and not socially interactive with her.

7 (Jordan King Video Clip No. 10 played.)

8 Q Okay.

9 A And --

10 SPECIAL MASTER HASTINGS: Before we leave  
11 this tape, in that segment his sister looked like a  
12 very, very young newborn.

13 THE WITNESS: Right.

14 SPECIAL MASTER HASTINGS: Would that be your  
15 interpretation?

16 THE WITNESS: Yes. I think she was a very  
17 new newborn. So my best guess on his age would be  
18 that he was around 15 months at that age.

19 SPECIAL MASTER HASTINGS: All right.

20 THE WITNESS: And that's all we have to show  
21 looking at normal characteristics. We have two brief  
22 clips post-regression that show a clear, I think,  
23 contrast to what we've been looking at.

24 BY MR. POWERS:

25 Q And before we move to those I just want to

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1 note in watching the videos here there were a couple  
2 of videos where he was vocalizing and babbling but  
3 honestly I didn't hear a lot of fully-formed words.  
4 What's your assessment of his language skills based on  
5 the video clips that we've seen here?

6 A I agree that we don't hear a lot of clearly  
7 articulated words in these video tapes. I did hear  
8 Mrs. King testify, and I found here to be a very  
9 reliable historian, and she gave word counts which  
10 would suggest that he did have normal language  
11 development. But what's striking in these videos is  
12 that he almost always either has a pacifier in his  
13 mouth or he's eating something or he's playing the  
14 harmonica and so I don't see a lot of language. So I  
15 think we have to be clear that for that aspect of  
16 those three domains of his development I don't have  
17 good examples on video and I am relying on parental  
18 history.

19 Q So you mentioned that the last two clips  
20 that we'll see here for Jordan King, 11 and 12, these  
21 are two that represent a presentation of post-  
22 regression symptoms?

23 A Right. And I mainly want to have the  
24 Special Masters look for a qualitative change on his  
25 facial expression, how much more detached he is now,

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1 how he doesn't show the same kinds of social  
2 reciprocity, and how he is oversensitive to auditory  
3 stimuli and exhibiting hand flapping.

4 Q Okay. So we will go ahead first and show  
5 Clip Number 11. And this is, the short title of this  
6 is he has "hands on his ears."

7 (Jordan King Video Clip No. 11 played.)

8 Is that a behavior that you noted in  
9 multiple videos after regression?

10 A Oh, after regression, yes. Did not see it  
11 before.

12 Q And finally for Jordan we're going to show  
13 Clip Number 12.

14 (Jordan King Video Clip No. 12 played.)

15 So what's significant about this videotape,  
16 particularly as you would compare it to the clips that  
17 we viewed and that you've reviewed before he  
18 regressed? What are the significant things to take  
19 from that clip?

20 A So it shows to me a significant qualitative  
21 change in the interactions with his father. Whereas  
22 before he was so engaged and now he seemed to be  
23 withdrawing. And he also is demonstrating a lot of  
24 hand flapping. Again the vacant expression in his  
25 face. And whereas before he seemed to enjoy

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1 manipulating tools in an appropriate way, now he seems  
2 to have lost that higher level of toy usability.

3 Q And particularly with the toys you are  
4 talking about the way that he just kept flipping the  
5 puzzle piece back and forth?

6 A Right; as opposed to putting it into the  
7 form board.

8 Q Okay. So now we're going to talk about some  
9 of the videos from William Mead?

10 A Yes.

11 Q And this is the list that is Petitioners'  
12 Trial Exhibit Number 16. We will use the same process  
13 here, Dr. Mumper, in introducing the context for the  
14 Special Masters and then showing the clips in  
15 sequence.

16 A Okay.

17 Q So Clip Number 1, can you explain what they  
18 are going to see and what they should be looking for?

19 A Actually, if I could just get a minute to  
20 get organized here? Because I was trying to find the  
21 specific things in Rust's testimony, which I have  
22 done. So now I just need to get oriented to his  
23 videos.

24 Q Okay.

25 SPECIAL MASTER CAMPBELL-SMITH: You

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1 anticipated my question, Dr. Mumper.

2 THE WITNESS: Say it again?

3 SPECIAL MASTER CAMPBELL-SMITH: You  
4 anticipated my question.

5 THE WITNESS: So on my page 5 of Dr. Rust's  
6 testimony he talks about kids with autism being head  
7 shy, not wanting to have their head touched or hair  
8 washed, that this is a very striking finding that  
9 comes on very early.

10 He also talked about aversive eye contact  
11 and how that was a systems problem that was worthy of  
12 careful scientific investigation.

13 And he also talked about parents in the  
14 family history tending to be rigid and aloof and  
15 hypersensitive to criticism. Through multiple video  
16 clips I did not find that to apply to either set of  
17 these parents.

18 And so with that as a background, the first  
19 tape that's called --

20 SPECIAL MASTER CAMPBELL-SMITH: Just a  
21 moment.

22 MR. MATANOSKI: I would just observe that I  
23 believe those comments by Dr. Rust were general  
24 comments. As I think the Court has observed before  
25 about some of the other comments that he had made they

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1 were to apply more generally to descriptions of  
2 autism.

3 MR. POWERS: And, Special Masters, he was  
4 describing why those general comments informed his  
5 opinion that these two boys demonstrated what he  
6 described as abnormal courses of development before  
7 their regression and that they had early onset. So  
8 again his comments were general but he was applying  
9 them in a way to support his opinions on the case-  
10 specific determination that both of these boys were  
11 abnormal at particular stages of their development.

12 SPECIAL MASTER CAMPBELL-SMITH: Okay.

13 MR. MATANOSKI: I recognize the Special  
14 Master can go back at the testimony of Dr. Rust. And  
15 we'd submit that our recollection of that is that it  
16 was general in nature and that I think it's pretty  
17 clear in his report and through his testimony that he  
18 did not dispute that either the King or Mead child had  
19 regressive autism.

20 SPECIAL MASTER CAMPBELL-SMITH: That is what  
21 I recollect. But Petitioners' counsel, if you would  
22 like to proceed with this demonstration in the absence  
23 of any objection from Respondent.

24 MR. POWERS: Yes. We would like to proceed  
25 and just with the note that while Dr. Rust did not

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1 dispute the what ultimately was regressive autism, he  
2 did make reference to both boys having -- he was very  
3 non-specific about it so we can't be more specific,  
4 but he did make reference to both boys being not  
5 normal before the regression. And if he had been more  
6 specific we could point to a particular page of his  
7 testimony, but he did make a general observation about  
8 the lack of normalcy before the regression that we all  
9 concede.

10 SPECIAL MASTER CAMPBELL-SMITH: I will  
11 observe that it was my recollection of Dr. Rust's  
12 testimony that at the time that it was documented one  
13 could assume that it had appeared earlier. But it  
14 was, there was an inability to determine, and he  
15 acknowledged he had not met the children and had not  
16 examined them personally. But based on what the  
17 reflections were in the medical record that the time  
18 that you are documenting something there is an  
19 understanding that the activity was a loss of the  
20 activity or function occurred before the notation.

21 But with that in mind we will -- and again,  
22 Dr. Mumper, if you would just describe, as you have.

23 THE WITNESS: Okay. And I will try to be  
24 brief.

25 The "Johnny Jump Up" tape is showing

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1 reciprocal social interactions with his, one of his  
2 parents.

3 SPECIAL MASTER CAMPBELL-SMITH: Do you have  
4 an estimated age for this? These are pretty well  
5 dated but I don't know what this is.

6 THE WITNESS: Hang on one second. On my --  
7 Scott, if you can help me, what I am looking for is  
8 not the recounted notations that you did this morning  
9 and last night but the ones that I set you by e-mail  
10 that had better ages?

11 BY MR. POWERS:

12 Q For "Johnny Jump Up" does it sound accurate  
13 to believe that this was something from November of  
14 1998 when he was about 5 months old?

15 A Yes. Yes, that is correct.

16 (William Mead Video Clip No. 1 played.)

17 The next one is marked "Pushing Up" and --

18 SPECIAL MASTER CAMPBELL-SMITH: What is the  
19 purpose of "Johnny Jump Up"?

20 THE WITNESS: To show the reciprocal  
21 interaction, the smiling, the fact that he's bright-  
22 eyed and alert, that he looks like a normal 5-month-  
23 old.

24 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

25 BY MR. POWERS:

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1 Q So the next clip is Clip Number 2 on Exhibit  
2 16, it's called "Pushing Up"?

3 A Right. And that's demonstrating great eye  
4 contact.

5 (William Mead Video Clip No. 2 played.)

6 Reciprocal smile, bright-eyes, laughter with  
7 the father.

8 Q Clip Number 3, "Bath Time." And I will note  
9 that it is with his sister, to Eleanor's everlasting  
10 embarrassment perhaps, but this is actually a  
11 significant clip, as Dr. Mumper will explain?

12 A And this clip speaks to the issue of Dr.  
13 Rust's testimony that not wanting to have their heads  
14 touched or their hair washed is a very early sign of  
15 children with autism. And in this video you will see  
16 that he tolerates that from his sister and he also  
17 does a fair amount of babbling.

18 (William Mead Video Clip No. 3 played.)

19 Q And again what was significant about that  
20 clip?

21 A Well, he seemed to tolerate or perhaps even  
22 enjoy the head touching. But he's showing a lot of  
23 reciprocal smiling and giggling and laughing and  
24 normal appearing bathtime play.

25 Q Tape Number 4, this one is called "Hi, Dad."

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1 What is the context and the significance of this clip?

2 What should the Special Masters be looking for here?

3 A This is a tape that was done around the time  
4 of his first birthday when he was around a year old.  
5 And he demonstrates the words "Hi, Dad." Two-word  
6 phrases typically come in around 18 months. It's  
7 demonstrating that at a year he at least had several  
8 words which is very much in keeping with the history  
9 given by the parents.

10 This tape also shows some reciprocal play  
11 with the sister again.

12 (William Mead Video Clip No. 4 played.)

13 Q And then we will just keep going, sort of an  
14 extension of this is Clip Number 5 which is called  
15 "Play Nice."

16 (William Mead Video Clip No. 5 played.)

17 Q And what was significant about that clip  
18 again having seen it again?

19 A He says "Hi, Dad" again and he's having  
20 reciprocal interactions with both the parent and the  
21 sibling.

22 Q Everything about that video was  
23 developmentally and age appropriate?

24 A It seemed very age appropriate to me, yes.

25 Q Okay. Now, the last two videos can you

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1 describe to the Special Masters what we'll be looking  
2 at?

3 A The last two are after his regression. The  
4 first one is showing him covering his ears and hand  
5 flapping as a stark contrast to his initial prior  
6 normal behaviors that we've attempted to demonstrate  
7 here.

8 And the second one I believe to be  
9 demonstrating that he has abdominal issues.

10 (William Mead Video Clip No. 6 played.)

11 Q Okay, now that, the date on that was July  
12 2000, so he would have been about 27 months old?

13 A That's correct. And I think you can  
14 appreciate the deterioration in the quality of his  
15 language. Whereas at a year he was able to say "Hi,  
16 Dad," he is pretty much reduced at this point to these  
17 guttural utterances. I think that there is a  
18 qualitative change to his facial expression, he has  
19 more of a vacant look. And he wasn't able to imitate  
20 saying cheese. It just is a way of demonstrating the  
21 regression.

22 Q And several times he was holding his hands  
23 to his ears?

24 A Yes. As if he had hyperacusis or was trying  
25 to modulate the incoming sensory stimuli.

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1 Q And was he also flapping his hands?

2 A And he also was flapping his hands, yes.

3 Q Had you seen any behavior like that prior to  
4 the, say, 16 months of age?

5 A I did not.

6 Q The final clip we're going to show is Clip  
7 Number 7. This is William at the computer?

8 A Yes.

9 Q Okay.

10 A And I would like to set up the Special  
11 Masters to look for his abdomen in this picture. One  
12 of the things that John Green did for which he was  
13 criticized by Dr. Rust was to work on aspects related  
14 to the child's diarrhea and bowel movements. And I  
15 believe that this tape shows inferential evidence that  
16 he was having abdominal pain. You will notice that  
17 his abdomen seems quite distended, quite bloated, that  
18 he pushes on the lower part of his abdomen. That at  
19 one point he is pulling on the skin. And this, these  
20 are behaviors we frequently see in children with  
21 autism. And I believe that we at least need to be  
22 open to the possibility that they are trying to  
23 communicate with us that their stomachs hurt in ways  
24 that they have to use since they no longer have  
25 language.

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1 (William Mead Video Clip No. 7 played.)

2 Q So, Dr. Mumper, in addition to the stomach  
3 issues was there anything else in that video clip that  
4 merits description or mention to the Special Masters?

5 A That he seemed nonresponsive to multiple  
6 efforts by his dad to engage him, that he was sort of  
7 staring fixed on the computer screen but not  
8 attempting interactive play.

9 Q So based on your listening to the parents'  
10 testimony, your review of the medical records, and now  
11 the videos that you've identified here, do you agree  
12 or disagree with Dr. Rust's testimony that each of  
13 these boys was likely not normal prior to their  
14 regression?

15 A I don't find evidence, so I disagree with  
16 him.

17 MR. POWERS: I have no further questions.

18 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

19 CROSS-EXAMINATION

20 BY MR. JOHNSON:

21 Q Good to see you again, Dr. Mumper.

22 A Hi.

23 Q As you know, my name is Vo Johnson. I am  
24 representing the United States.

25 Doctor, you covered a lot of different

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1 issues in the early part of your direct, and so I just  
2 want to make sure that in the last two weeks since  
3 you've testified that you have not become an expert in  
4 biochemistry; is that correct?

5 A That is absolutely correct.

6 Q And you've not become an expert in  
7 neurology?

8 A That's true.

9 Q You've not become an expert in psychiatry?

10 A That's true.

11 Q And you have not become a clinical  
12 psychologist?

13 A That's true.

14 Q Okay. And you have not become an expert in  
15 toxicology?

16 A That's true.

17 Q And you've not become an expert in  
18 neurotoxicology?

19 A That's true.

20 Q And you've not become an expert in genetics?

21 A That's true.

22 Q Would it be fair to say that your knowledge  
23 of those areas is based on what you have learned from  
24 your colleagues at the Autism Research Institute?

25 A And through my reading of other literature,

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1 yes.

2 Q What other literature are you specifically  
3 referring to?

4 A We maintain bibliographies not just of the  
5 papers written by people officially associated with us  
6 but other articles in the autism literature. So the  
7 works of, you know, Pardo, Zimmerman, Vargas, Martha  
8 Herbert, people that are in Italy working on the  
9 environmental components of autism, people at the Mind  
10 Institute that are working on immune dysregulation and  
11 autism, Federico Balzola in Italy that's working on gut  
12 abnormalities in autism. So the list could go on but  
13 we don't limit ourselves to what is just within our  
14 institute's publications.

15 Q Would it be fair to say that you give more  
16 weight to those articles that your colleagues at the  
17 Autism Institute are feel are helpful?

18 A That would be a fair statement. And I also  
19 give more weight to articles where I've had the  
20 opportunity to discuss them with the authors.

21 Q Doctor, you were asked a number of questions  
22 about the different treatment therapies that Dr. Green  
23 provided to both William Mead and Jordan King, and  
24 that I think you also use to some extent in your own  
25 practice?

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1 A That's true.

2 Q One issue that was asked about was  
3 chelation, the use of chelation therapy. And I  
4 believe that you said that, you testified that even  
5 though or that the justification for the use of  
6 chelation in your practice was really targeted towards  
7 the lead that was present; is that what you testified  
8 to?

9 A That's not exactly the way I meant to say  
10 it. I was saying that we look at various types of  
11 toxicity, and lead is very, very common. So when we  
12 do porphyrin analyses the two main things that we're  
13 looking for in those porphyrins are lead and mercury.  
14 And we have come to very much appreciate how much they  
15 co-exist. And so treating lead toxicity is well  
16 within something that pediatricians have had  
17 experience with in terms of treating that with DMSA,  
18 which was also used in these boys.

19 Q What symptoms of lead toxicity are you  
20 relying on for the justification to do chelation  
21 therapy?

22 A Well, the classic symptoms of lead toxicity  
23 include irritability, hyperactivity, or declines in  
24 cognitive performance. But as you may know, the  
25 American Academy of Pediatrics and the CDC have

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1 recommended ongoing lead screening for children  
2 because it is not felt to be prudent to rely on  
3 development of symptoms as opposed to trying to  
4 address lead toxicity if it exists in a child somewhat  
5 unsymptomatically at the time.

6 Q So you would require some testing showing an  
7 abnormal lead level in the blood before you would do  
8 chelation therapy in that child?

9 A No. Because the lead levels in the blood  
10 only persist for a relatively short time. The blood  
11 turns over very quickly within two to three months.  
12 So unless you are getting the child at the age of the  
13 acute lead exposure, you may miss the exposure in the  
14 blood. And so you are left with indirect  
15 measurements.

16 Q So it's your testimony that blood testing is  
17 not a reliable measure of lead body burden?

18 A Right. It can be used to look for acute  
19 exposure.

20 Q Doctor, you would at least agree that people  
21 have died from chelation therapy; correct?

22 A Yes.

23 Q And I believe you mentioned one case that  
24 Dr. Rust raised in his testimony but that's not the  
25 only case in which someone has died from chelation

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1 therapy; is that right?

2 A He mentioned four. And I consulted with  
3 several of my colleagues and we could not find four  
4 cases. We were only aware -- I'm mostly aware of the  
5 one that I mentioned. And I think that there was one  
6 other one of which I'm not familiar. But I do not  
7 know the third or fourth case.

8 Q Are you aware of one case that actually  
9 involved there was a lawsuit that was brought and one  
10 of the defendants that was named in that lawsuit was  
11 Metametrix which is one of the labs that's done  
12 testing in these two cases?

13 A I was not aware that Metametrix was named in  
14 that lawsuit, no.

15 Q You talked a little bit about some of the  
16 therapies that Dr. Rust criticized. And I wanted to  
17 ask you a couple of questions about those. And let's  
18 start out with IVIG since that was the first one that  
19 you discussed.

20 A Uh-huh.

21 Q How does IVIG treat persistent inorganic  
22 mercury in the brain?

23 A I am not saying that it does.

24 Q So whether IVIG treatment is effective in  
25 any given case really doesn't speak to the issue of

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1 whether thimerosal from vaccines contributed to  
2 autism; is that correct?

3 A It speaks to the issue that he was treating  
4 documented low IgG levels in those children.

5 Q And that's not specific to persistent  
6 inorganic mercury in the brain; is that right?

7 A Not to my knowledge.

8 Q How does Eskimo oil treat persistent  
9 inorganic mercury in the brain?

10 A I am not aware of any studies that have  
11 assessed that specifically.

12 Q So the ineffectiveness or effectiveness of  
13 Eskimo oil in treating symptoms of autism really  
14 doesn't speak to whether thimerosal from vaccines  
15 contributes to autism; is that right?

16 A That's correct. It's being used for  
17 intestinal reasons and the other reasons that I  
18 articulated.

19 Q How does valtrex treat persistent inorganic  
20 mercury in the brain?

21 A Actually I think that if you use valtrex to  
22 decrease adenosine which would then allow methylation  
23 biochemistry to proceed, the ultimate result of that  
24 would be an increase in glutathione as demonstrated in  
25 Dr. James' work. Cysteine and glutathione are part of

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1 the integral mechanisms for handling mercury. And so  
2 whereas John was not doing it specifically with the  
3 target of working on inorganic mercury in the brain,  
4 that is actually quite a biologically plausible way to  
5 improve detoxification capacities through the body's  
6 own natural mechanism since glutathione is the primary  
7 thing that we rely on to try to handle mercury  
8 toxicity.

9 Q And remind me again, what is valtrex, what's  
10 the primary clinical use for valtrex?

11 A It's an antiviral agent but it's also a  
12 purine analog and so that's where its utility in  
13 dealing with adenosine comes in.

14 Q And when you say "antiviral" I believe you  
15 said Dr. Rust testified that it was used for use in  
16 genital herpes; is that correct?

17 A Right. And it's also been looked at for  
18 other types of viral infections, HHV6, Epstein-Barr  
19 virus, cytomegalovirus. I'm not sure from John's  
20 notes if he was primarily using the viral mechanism or  
21 the adenosine mechanism, or both.

22 Q Are you aware that valtrex has never been  
23 tested in a pediatric population?

24 A I would not be surprised because many drugs  
25 that we use are not tested under the age of 12.

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1       However, acyclovir is the drug of indication for  
2       newborn herpes encephalitis. And valtrex breaks down  
3       to acyclovir. So we have clear precedent in standard  
4       medical practice for using it in even newborns.

5             Q       Would you be more comfortable using that,  
6       using valtrex if it had been tested in the pediatric  
7       population?

8             A       We have become used to not always having  
9       that luxury, but it's always great when the studies  
10       are done on the children. So, yes, I would be more  
11       comfortable.

12            Q       And I think you alluded to earlier that  
13       there aren't case-controlled studies on the use of  
14       many of these therapies; is that right?

15            A       That is true.

16            Q       Would you feel more comfortable as a  
17       pediatrician treating children if these various  
18       treatments had been tested in a case-controlled study?

19            A       As long as the case-controlled study took  
20       into account the medical problems of the child and was  
21       not very heterogeneous, yes, I would.

22            Q       And are you testifying here today that you  
23       believe that your clinical judgment about the  
24       effectiveness of these treatment therapies is more  
25       reliable than a case-controlled study would be?

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1           A     I'm testifying that when we are trying to  
2     take care of a generation of children and being  
3     overwhelmed by their medical problems that we are in a  
4     position where we are trying to take care of the  
5     individual patient and we feel some urgency that we  
6     can't wait for 10 or 20 years.  These children seem to  
7     have a window of opportunity where if you treat their  
8     medical problems they get better.  And with the  
9     timeline of applying for grants, getting the studies  
10    completed and analyzing the results and then the meta-  
11    analyses, we are proceeding in good faith, using our  
12    best clinical judgment, realizing that we don't have  
13    good case control studies for all that we do.

14           Q     And I've heard you use the phrase in the  
15    past, refer to the concept the child is your  
16    laboratory.  Is the approach you just described what  
17    you are referring to when you say the child is your  
18    laboratory?

19           A     That's a shortcut way of saying that when  
20    you're doing intervention your biggest outcome is how  
21    it affects a particular child.  We certainly use a lot  
22    of laboratory values in helping us assess the child.  
23    But if we are able, for example, to give valtrex and  
24    document that for a particular child their adenosine  
25    level went from high to the normal range, as it did in

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1 Jill James' work, that is more important to me than  
2 what acyclovir did in 20 kids that are not my patient,  
3 because for that patient it demonstrated an  
4 improvement.

5 Q So in other words, you're willing at least  
6 at this point to rely on your clinical judgment, even  
7 in the absence of case-controlled studies showing that  
8 these treatments are effective?

9 A There are case controlled studies looking at  
10 a number of these treatments showing their efficacy.  
11 That has been demonstrated, for example, for B6.  
12 There are about 22 studies demonstrating efficacy.  
13 There have been studies looking at omega-3 and  
14 demonstrating efficacy. We have looked at multiple  
15 vitamins and we demonstrated efficacy.

16 But much more work remains to be done.

17 Q And those treatments that you just talked  
18 about are those the ones that are targeted at the  
19 oxidative stress issue?

20 A Many of them are, yes.

21 Q And oxidative stress is not specific to  
22 mercury toxicity; is that right?

23 A That's correct.

24 Q Those, other things can cause oxidative  
25 stress?

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1 A That's correct.

2 Q Doctor, did you listen to the testimony of  
3 Dr. Rutter or Dr. Lord or Dr. Fombonne?

4 A No. I was not able to hear Dr. Rutter or  
5 Dr. Lord. And I only heard part of Dr. Fombonne's  
6 testimony when I was driving up yesterday.

7 Q I believe you testified when you were here a  
8 couple of weeks ago that you don't diagnose autism; is  
9 that right?

10 A Yes. I do rely on other psychologists,  
11 psychiatrists to be the one who makes the diagnosis.  
12 I'm concerned that if I were to diagnose them and then  
13 take care of them and they get better that the  
14 criticism would be levied that I must have  
15 misdiagnosed them in the first place.

16 Q Do you know what the ADIR is?

17 A Yes.

18 Q Can you tell us?

19 A Autism Diagnostic Interview Revised.

20 Q Do you use that tool in your practice?

21 A I do not. I get reports on it from other  
22 people but we do not do those intakes ourselves.

23 Q Do you know what the ADOS is?

24 A The Autism Diagnostic Observation Scale.

25 Q Do you use that in your practice?

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1           A     I typically do not. We did use the ADOS in  
2     one of our clinical trials in which we hired a  
3     psychologist to administer it to our patients. But I  
4     do not have any experience administering it myself.  
5     Again I rely on reports from other doctors.

6           Q     Do you know whether the ADIR or ADOS have  
7     any questions that are targeted to the issue of  
8     determining whether a regression has occurred?

9           MR. POWERS: I'm going to object. This is a  
10    re-do of the cross-examination of this witness and is  
11    not addressed to any of the issues that she discussed  
12    in her rebuttal testimony today. I believe that  
13    opportunity to raise these issues would have been  
14    during cross or during re-cross following her direct  
15    testimony. This is not surrebuttal.

16          MR. JOHNSON: Special Masters, we have heard  
17    a great deal of testimony from Dr. Mumper today based  
18    on the videos in which she is purporting to identify  
19    abnormal development as opposed to normal development.  
20    I think this goes directly to her qualifications for  
21    being able to offer that testimony.

22          SPECIAL MASTER CAMPBELL-SMITH: Proceed.

23          MR. JOHNSON: Thank you.

24          THE WITNESS: My memory is that the ADIR  
25    does have some targeted questions that work on

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1 identifying regression.

2 BY MR. JOHNSON:

3 Q Do you know what those questions are?

4 A No, I do not.

5 Q In your own practice do you use any  
6 standardized questionnaire when you are taking a  
7 parent history or a patient history from the parent?

8 A We use an intake form. It is not  
9 standardized.

10 Q What questions do you ask to determine if  
11 there's been a regression?

12 A We look for -- we ask questions about age-  
13 appropriate language, social and reciprocal behavior.  
14 We look for a time at which the child seems to be  
15 meeting milestones. Then we look for a period where  
16 they clearly lose those milestones.

17 The classic example is to expect that the  
18 skills are obtained and then they're clearly lost and  
19 that there's a period of time, some people use three  
20 months, between the time that they clearly  
21 demonstrated that and then have clearly lost it.

22 Q Can you give some examples of specific  
23 questions that you ask?

24 A How many words did your child have at one  
25 year? Momma, Dadda, hi, bye and Nonna.

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1           How many questions did your child have at 18  
2 months? He was no longer speaking any words but  
3 sometimes he's just "ummm" or "mmmm."

4           Q     Were you giving examples of both the  
5 question and an answer to the question?

6           A     Right. Right. The question initially is  
7 assessing language at a certain point and then  
8 assessing language at another certain point. And I  
9 was trying to give an example of regression.

10          Q     You determined in this case that there was  
11 totally normal development based solely on the review  
12 of -- your review of the medical records; is that  
13 right?

14          A     I made the judgment that up to some point  
15 that the child appeared to me to be normally  
16 developing based the notations in the well-baby  
17 checkups which I went through month by month, and on  
18 the basis of the videos that I was able to review in  
19 which the normal development seemed to correlate with  
20 what had been annotated in the pediatrician's records.  
21 And then at some other point there was loss of  
22 language, loss of social reciprocity, loss of  
23 reciprocal behavior and appropriate play versus  
24 ritualistic play.

25          Q     Now, you didn't review the videos until the

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1 Thursday before you testified; is that correct?

2 A That is correct. I did not get them until  
3 then. So at the time that I wrote the report I was  
4 very much dependent on the pediatric records which  
5 seemed to be doing a state of the art kind of  
6 assessment at the well-baby visits and then clearly  
7 documenting a regression.

8 Q And you also at the time of your report had  
9 not interviewed the parents; is that correct?

10 A That is correct, yes.

11 Q Did you hear Drs. Rutter, Lord and Fombonne  
12 all testify that parents often don't recognize early  
13 subtle signs of abnormal development?

14 A I did not hear that testimony but I do know  
15 that Rutter and Lord and Fombonne have written about  
16 that and talked about the subtle signs.

17 Q Do you disagree with their testimony on that  
18 issue?

19 A I think there are certainly cases in which  
20 parents overlook subtle signs. And if there are  
21 subtle signs that I missed on these videos I will be  
22 open to learning from those colleagues.

23 I think the fundamental issue though is do  
24 these kids look like they're abnormal from birth or do  
25 they look like they're on a normal developmental

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1 trajectory and then something happens that interferes  
2 with that. So that for me is the crucial kind of  
3 issue.

4 Q I think you testified that you did not see  
5 evidence on the videos of Jordan King not wanting to  
6 be held; am I characterizing your testimony correctly?

7 A I think I said that I saw a number of cases  
8 where he was being held. Now, there were a couple of  
9 examples on the videotape where he did try to get out  
10 of the parent's arms. And so I just was trying to  
11 point out that the sort of all or nothing situation  
12 doesn't exist. And I think that he was able to  
13 tolerate being held many times.

14 Q You would agree that there are notations in  
15 the medical record that indicate his mother reporting  
16 that Jordan didn't like to be held as an infant,  
17 wouldn't you?

18 A Yes.

19 Q In fact, at Jordan King Exhibit 8, page 109,  
20 notes "Mother noted that Jordan was more content not  
21 to be held as an infant." Would that be one of the  
22 notations that you saw when you reviewed the record?

23 A I think so. I'd just like to look and see  
24 where it is.

25 (Witness reviews document.)

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1 Yes.

2 Q And also at Jordan King Exhibit 8, page 87 -  
3 - no, sorry. Yes, page 87, does it say "at 3 months  
4 he was never an I-want-to-be-held child but did allow  
5 it then grew out of that"?

6 A Uh-huh.

7 Q Doctor, I think you testified a couple of  
8 weeks ago that you don't typically use videos in your  
9 own practice; is that right?

10 A That is correct.

11 Q That you normally just don't have time to  
12 view the videos?

13 A Right.

14 Q Were you ever asked -- let me ask this.  
15 When were you first asked by counsel to go through the  
16 videos and identify clips that you thought or that in  
17 your opinion showed normal development?

18 A Gosh. I can't, I can't really remember the  
19 timing. I think it was sometime about two weeks after  
20 the DAN conference, which would have put it maybe in  
21 the third week of April. But I'm not at all sure  
22 about that.

23 Q And you obviously didn't view the videos at  
24 that time, is that right, because you only saw them  
25 for the first time the Thursday before you testified?

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1           A     The -- I'm trying to remember now. I  
2     remember there was a Saturday morning that I spent a  
3     great deal of time looking at them. I guess what I  
4     really need to determine is when I actually received  
5     them. And, I'm sorry, I can't remember the timing on  
6     it.

7           Q     So as you sit here you just can't recall  
8     when you first viewed the videos?

9           A     All I can say with certainty is that it was  
10    sometime after our big Defeat Autism Now conference  
11    which was sometime in early April.

12          Q     And did you prepare any notes at that time  
13    regarding the videos?

14          A     I have a bunch of notes. One set the first  
15    time I reviewed them, another set trying to hone in on  
16    what was testified on by Dr. Rust. So the honing in  
17    happened this past weekend. The first review I think  
18    I only, I think I only got the notes the Thursday  
19    before I was due to testify the following Friday, so I  
20    had looked at it the weekend before my testimony.  
21    That's my best recollection. So I have two sets of  
22    notes from the first review the weekend before I  
23    testified and from the second review the weekend  
24    before this testimony.

25          Q     Was it communicated to you when you first

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1 received the videos approximately two weeks after the  
2 Defeat Autism Now conference that you're referring to,  
3 was it communicated to you at that time that the  
4 Petitioners were asked to designate specific portions  
5 of the videos that they contended showed normal  
6 development?

7 A I may have misunderstood the timing on that  
8 because I didn't realize I was supposed to submit that  
9 way ahead. I'm sorry. I guess I may have  
10 misunderstood that.

11 Q And have you ever been provided a copy of  
12 Respondent's video designations?

13 A No.

14 MR. JOHNSON: Thank you. I have nothing  
15 further.

16 SPECIAL MASTER CAMPBELL-SMITH: Any further  
17 questions from Petitioners' counsel?

18 MR. POWERS: Not at this time, no.

19 SPECIAL MASTER CAMPBELL-SMITH: Any  
20 questions from my colleagues?

21 SPECIAL MASTER HASTINGS: Yes. I have just  
22 a couple I think, Dr. Mumper.

23 According to your report and my view of the  
24 record as well, Jordan received the thimerosal in  
25 question at birth and at age 2, 4 and 6 months. So

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1 according to your report your summary is that by the  
2 time he was 7 months old he had received a total of  
3 187.5 micrograms of ethyl mercury?

4 THE WITNESS: Yes.

5 SPECIAL MASTER HASTINGS: Is that right?

6 THE WITNESS: I think that sounds correct.

7 SPECIAL MASTER HASTINGS: So I want to ask  
8 you then is the timing of the onset of Jordan's  
9 symptoms is it crucial to your ultimate opinion,  
10 you've indicated the opinion that in Jordan's case you  
11 think it's probable that the thimerosal and that  
12 series of vaccines contributed to his autism?

13 THE WITNESS: Yes.

14 SPECIAL MASTER HASTINGS: Would it matter in  
15 that opinion whether the first symptoms occurred at 18  
16 months or 13 months or 9 months? Does it matter?  
17 Would your opinion be the same?

18 THE WITNESS: No, sir, it really doesn't  
19 matter to me because I think the crucial thing here is  
20 that mercury can be latent for a period of months  
21 before it manifests. The classic example of that is a  
22 lab researcher who got two drops of mercury on her  
23 gloved hand and seemed fine for about three or four  
24 months, then go dramatically ill and ultimately died.  
25 So the concept of it being there and not causing overt

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1 symptoms for a while as yet to be determined until we  
2 study this better is entirely consistent with what I  
3 believe to be the case here.

4 So for me the crucial thing is more than at  
5 least initially he seemed to be developing normally  
6 and then he had the development of autistic symptoms.  
7 And whether they started at 15 months, 18 months, 20  
8 months or 22 months doesn't really change my mind  
9 about the plausibility that thimerosal was a  
10 contributing factor.

11 SPECIAL MASTER HASTINGS: All right. And  
12 that wouldn't change if they occurred even earlier  
13 than that, say 13 months?

14 THE WITNESS: Right. Because in this case  
15 his exposure, his first exposure was a hepatitis B  
16 vaccine at birth which he got when his mother had been  
17 given antibiotics for a fever and he had just been  
18 born. So the initial exposure was quite early on. So  
19 it's very difficult for me to tie an exact timeline to  
20 overt symptoms.

21 SPECIAL MASTER HASTINGS: All right. That's  
22 all I have.

23 SPECIAL MASTER CAMPBELL-SMITH: I have a  
24 couple.

25 Would that same observation apply in the

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1 Mead case?

2 THE WITNESS: Yes.

3 SPECIAL MASTER CAMPBELL-SMITH: The other  
4 matter I wanted to get from you, you had indicated  
5 there were three particular record citations regarding  
6 the normal head size at birth --

7 THE WITNESS: That's correct.

8 SPECIAL MASTER CAMPBELL-SMITH: -- for  
9 William. I only noted one. Perhaps you didn't say  
10 all three of them but I'd like to get those.

11 THE WITNESS: Yeah. Let me see if I can  
12 find that page. The problem is is that the second one  
13 I cited did not have an exhibit number on my copy. So  
14 I will turn my paper copy over and one page has two  
15 different citations, one about head size and one about  
16 skin. And so it's titled Providence St. Vincent  
17 Medical Center nursery admission record.

18 SPECIAL MASTER CAMPBELL-SMITH: Right.

19 And the third one?

20 THE WITNESS: The third one is on the same  
21 page as the second one.

22 SPECIAL MASTER CAMPBELL-SMITH: Okay.

23 THE WITNESS: It was just three different  
24 places that addressed the issue of maybe the head size  
25 was off because of trauma.

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1 SPECIAL MASTER CAMPBELL-SMITH: Let me  
2 inquire, I recall that your testimony on -- during  
3 your initial time, and I can't remember whether it was  
4 direct or cross, but you look for deviations from the  
5 standard that would cause you to be concerned about  
6 head circumference. Dr. Rust gave some testimony that  
7 really it didn't matter what the head, the birth size  
8 or birth time measurement for the head circumference  
9 was, he looks for trends.

10 THE WITNESS: True.

11 SPECIAL MASTER CAMPBELL-SMITH: Do you  
12 disagree with the looking for trends in your personal  
13 practice or what I'm trying to get at is do you think  
14 that is an invalid way or are you challenging the  
15 validity of what he said?

16 THE WITNESS: No. I agree completely with  
17 him that trends are important. And I also am open to  
18 the possibility that any isolated point could be an  
19 error. That's why when he postulated an error in this  
20 case I went back to see if maybe the head was noted to  
21 be misshapen or have a cephalhematoma or caput  
22 succedaneum because he was postulating that the higher  
23 head circumference at birth might have been  
24 artifactual.

25 The trend he's looking for is a normal or

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1 low head size that then goes up and then comes back  
2 down. And that has been classically described in many  
3 different cases of researchers that are looking at  
4 head circumference as one way of understanding autism.  
5 And that model fits well with a lot of the published  
6 literature. I was just pointing out that we didn't  
7 seem to have that model in William Mead and that if  
8 you were going to throw away that first measurement  
9 because it was high and therefore the trend wouldn't  
10 have been as dramatic, it would be nice to have more  
11 than just speculation that it might have been wrong or  
12 that the child might have had head trauma.

13 And it just seems like from the medical  
14 records that potentially limited as they are that we  
15 do not have reason to think that his head really  
16 wasn't that size at birth, that it really wasn't 80th  
17 to 85th percentile at birth.

18 But I agree, trends are important, much more  
19 so than individual numbers; correct.

20 SPECIAL MASTER CAMPBELL-SMITH: And just to  
21 be clear about your position in this particular case,  
22 you don't think that that trend exists, it is your  
23 interpretation of William Mead's records that that  
24 trend does not exist for William Mead, the trend to  
25 which Dr. Rust referred?

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1 THE WITNESS: I think for me it's going to  
2 be an unanswered question. It seems to me that we  
3 have evidence that he started out on a growth  
4 percentile for his head that was very much in keeping  
5 with the rest of his body. He did show some  
6 elevations in his head circumference at the 4, 6 and 9  
7 month checkup. And then he comes down a little bit  
8 above the 50th percentile. So it is a little bit of a  
9 trend that shows the decrease in head circumference  
10 after an initial higher point. I just don't want to  
11 leave out the possibility that initially he was  
12 already at a high point.

13 SPECIAL MASTER CAMPBELL-SMITH: Thank you.  
14 Have my questions of Special Master Hastings'  
15 questions provoked further questions from counsel?

16 MR. POWERS: They have not, Special Master.  
17 Just to note for you that the exhibit number that Dr.  
18 Mumper was referring to, and this is in William Mead's  
19 individual file, it's Exhibit 3, page 13.

20 SPECIAL MASTER CAMPBELL-SMITH: Thank you  
21 very much.

22 Does that conclude Petitioners' presentation  
23 of your rebuttal witnesses?

24 MR. WILLIAMS: We have one more matter.  
25 Special Master Vowell had asked me to lay a foundation

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1 for the limitations on the data that Dr. Young and  
2 Geiers had to deal with when they produced their  
3 study. And I have a letter from Dr. Young explaining  
4 that and also responding to some of the criticisms  
5 that Dr. Fombonne made two days ago. And we've marked  
6 this as Petitioners' Exhibit 17.

7 We will file it.

8 (The document referred to was  
9 marked for identification as  
10 Petitioners' Exhibit No. 17.)

11 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

12 Are you planning to address these?

13 MR. WILLIAMS: No. Although I will state I  
14 checked with her and she is available that week in  
15 July if the Special Masters would want to ask her  
16 questions or if Respondent wants to ask her questions,  
17 she'll be here. I think she could come any one of  
18 those five days. And I doubt if her testimony would  
19 be very lengthy in any way, so.

20 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

21 MR. MATANOSKI: Your Honor, I will consider  
22 this new trial exhibit and determine whether we have  
23 any objection to the extent it may constitute a  
24 rebuttal evidence to prior testimony that was  
25 previously unexplained, unanticipated as it were.

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1 SPECIAL MASTER CAMPBELL-SMITH: So noted.

2 Should you like to lodge a formal objection you will  
3 draw it to our attention?

4 MR. MATANOSKI: Yes, that's correct, ma'am.

5 SPECIAL MASTER CAMPBELL-SMITH: Let me take  
6 a look here. We are at about 1:20. And my question  
7 to Respondent's counsel, do you have witnesses that  
8 you intend to introduce or put on this afternoon?

9 MR. MATANOSKI: Yes, ma'am, we do.

10 SPECIAL MASTER CAMPBELL-SMITH: Do you have  
11 an idea about does it make sense for us to press on a  
12 little bit longer or is this an appropriate time for I  
13 will call it a lunch break?

14 MR. MATANOSKI: I think it would be the  
15 appropriate time for a lunch break, ma'am.

16 SPECIAL MASTER CAMPBELL-SMITH: With that  
17 said, how much time would counsel require to eat and  
18 for your working lunch?

19 MR. MATANOSKI: If I may have a moment, Your  
20 Honor?

21 SPECIAL MASTER CAMPBELL-SMITH: Please. We  
22 won't charge this minute.

23 (Pause.)

24 MR. MATANOSKI: Forty-five minutes should be  
25 fine for us, ma'am.

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1 SPECIAL MASTER CAMPBELL-SMITH: Okay. That  
2 puts us roughly at 2:05 that we will return. And we  
3 will take a lunch recess and return then.

4 MR. MATANOSKI: Thank you, ma'am.

5 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

6 (Whereupon, at 1:20 p.m., the hearing in the  
7 above-entitled matter was recessed, to reconvene at  
8 2:05 p.m. this same day, Friday, May 30, 2008.)

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1 Jordan King and William Mead had been receiving for  
2 their treatments for autism and other related,  
3 allegedly related conditions. Are the majority of  
4 those treatments recommended by the majority of autism  
5 experts?

6 A No. Actually none of them is recommended by  
7 autism experts. And there are actually published  
8 guidelines about the evaluation and the management of  
9 treatment of autism by the American Academy of  
10 Pediatrics or neurologists, and none of them  
11 recommends these practices.

12 Q Is there any evidence as to the efficacy of  
13 those treatments?

14 A No. That's one of the reasons that there is  
15 no evidence for their efficacy, no evidence for the  
16 reason for them to work, but there is no published  
17 studies which would suggest that it would change the  
18 course of autism.

19 Q Are any of those treatments dangerous?

20 A Yes. Often these treatments are thought to  
21 be innocuous by parents who are trying to do  
22 everything they can. And we understand that. But  
23 some of these treatments might actually be detrimental  
24 to the health of the children. So chelation therapy  
25 could be, as we know, dangerous if it is not well

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1 controlled. The use of megavitamins, B-12, B-6 and  
2 magnesium treatment has been associated with cases of  
3 neurotoxicity at times. And the diet with greater  
4 frequency in fruit, for instance, has been studied two  
5 years ago where it has been shown that the children  
6 who were strictly on this diet actually had lower  
7 levels of plasma amino acids which are essential for  
8 growth and brain growth in particular.

9 So the belief that these treatments can be  
10 tried and would be harmless anyway is actually not  
11 supported by the data.

12 Q Are there standards that are used by the  
13 medical and scientific community before a treatment is  
14 recommended?

15 A Yes. There are different kinds of standards  
16 the efficacy of interventions. The evidence which is  
17 the most conversed which stems from randomized  
18 clinical trials which are obviously double blind  
19 placebo controlled and for this method there is no  
20 study which has been relying on this method for the  
21 practices of the treatment which has been discussed  
22 this morning.

23 Q Do you have experience with randomized  
24 clinical trials?

25 A Yes, actually I did. I started working on a

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1 randomized clinical trial, did a thesis on that, my  
2 first two publications I think had to do with  
3 randomized clinical trials. And I am currently we are  
4 testing the efficacy in a randomized clinical trial of  
5 a treatment which is not biomedical which a language-  
6 based intervention to improve communication skills in  
7 young children with autism. And we did a randomized  
8 clinical trial. It's a 12-weeks treatment. And I  
9 looked at it at random parents and attached them to a  
10 group where they were immediately treated with this  
11 intervention. And there was a waiting list control  
12 group of 36 families or children in each group, so  
13 it's quite powerful in terms of the statistical power.

14 I just want to share with you our findings  
15 that it's an intervention that everybody likes. When  
16 we did the trial we had all the impression that it was  
17 actually achieving some of the positive results.  
18 Parents were happier, were convinced that the methods  
19 were showing efficacy. And we did too. But as we did  
20 the study well we didn't analyze the data before the  
21 data were finally collected. And when we broke the  
22 blind and looked at the results and there is no  
23 evidence for a big difference between the two  
24 treatment groups which is breaking my heart in some  
25 ways. But that also shows that our experience as

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1 clinicians and as parents can be misleading.

2 And I think the field of autism has been  
3 replete over the last 30, 40 years of treatments and  
4 interventions that practitioners engage into and their  
5 parents apply to that treatment. And the story has  
6 been that when you take these practices and put them  
7 to redraw some clinical tests, that of the randomized  
8 clinical trial usually the story is much more  
9 disappointing. And a case in point is this secretin  
10 study.

11 Q Is it the secretin study that you're  
12 referring to?

13 A Yes, yes. And again that was a usual  
14 progression after acute case reported by the  
15 researcher, by practitioners. It is changing with  
16 autism, and it was to the extent that parents  
17 worldwide were wanting to have their children using  
18 secretin. And I think the NIH at that time founded  
19 three separate randomized clinical trials which were  
20 converted, it took about five years to do that. And  
21 when the results were released all these three  
22 randomized clinical trials were negative, there was  
23 no, absolutely no advantage for secretin over placebo.  
24 So that had to result in question. But still you had  
25 like five or six years of practices where people

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1 believed in it, both practitioners and parents.

2 And the point is that the clinical  
3 experience is in no way a measure of the efficacy of a  
4 treatment, including mine.

5 Q Dr. Mumper also discussed IVIG treatment and  
6 she took issue with Dr. Rust's criticism of IVIG  
7 treatment. Do you have any experience with IVIG?

8 A Yes. Actually we did publish, we see the  
9 first offer in my C.V. is, last term, is a study which  
10 I supervised. It's a small study of a group of about  
11 20 children. We were assessed in the Immunology  
12 Department of the Montreal Children's Hospital at the  
13 time when this treatment became very fashionable,  
14 should I say, so many parents wanted to have access to  
15 this treatment. And rather than to do nothing with  
16 that our immunologists reluctantly initially they  
17 said, well, let's explore the immune system and see if  
18 there is really a deficiency in, immune deficiency  
19 genes in the children.

20 And we published this study in an  
21 immunological journal. And in fact we didn't, we  
22 failed to find evidence that there was a deficiency in  
23 leukocidins and there was just one child who had an  
24 unusual pathology. We see that as a sort of attempt  
25 to repeat that, there was no documented evidence for

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1 efficacy.

2           So the story is that as a routine treatment  
3 it has no place in the management of autism unless you  
4 have a documented deficit, immunological deficit which  
5 has to have some certain characteristics.

6           Q     Now, Dr. Mumper also discussed single-study  
7 baseline studies. You take one child and you look at  
8 the efficacy of various treatments applied to that one  
9 child. Do you have any experience yourself with  
10 single study baseline studies?

11           A     Yes. We want to have randomized clinical  
12 trials but often we don't have this level of evidence,  
13 so there are lower levels of evidence to ascertain the  
14 efficacy of interventions, particularly in the  
15 behavioral domain but also in the use of medication or  
16 biomedical intervention. So the single subject  
17 designed with multiple baseline evaluation is a way to  
18 test.

19           You measure a child without doing anything  
20 with him at several points in time so you assess his  
21 baseline of behavior. And then you administer the  
22 treatment that you think might make a difference and  
23 then you follow by several assessments and then you  
24 remove the intervention and you expect that the child  
25 would respond to the treatment. And you would go back

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1 to baseline to remove the interventions. So that's a  
2 way to observe over time the child before, during and  
3 after treatment in a way which is more rigorous and  
4 allows to draw some meaningful inferences. The  
5 causality inference might sometimes be subject to  
6 coaching, but it's a progress compared to the simple  
7 clinical acumen that people have when they say that's  
8 what I like to do, that's what I do, it seems to work;  
9 that is not enough.

10 And I was told that this design has been  
11 available for years and the people who support the  
12 chelation therapies and all this sort of treatment  
13 have failed completely to publish data which is  
14 rigorous and can be analyzed in this sort of  
15 preliminary way.

16 Q Doctor, I'd like to discuss the videos. We  
17 were shown some videos this morning by Dr. Mumper that  
18 allegedly show Jordan King and William Mead's normal  
19 development before their regression. Do you have any  
20 comments, before we look at some specific videos that  
21 we saw this morning do you have any comments with  
22 regard to Dr. Mumper's methodology, the way she  
23 assessed the videotapes?

24 A Yes. I think there are some videos in which  
25 her comments were not actually supported by what's on

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1 the video. And I will give some examples of that.

2 As I said the other day, what is very  
3 important when we assess them and we look at them is  
4 to look at the quality of the behavior. It's not only  
5 it's there or not, we need to evaluate the quality.  
6 So when we talk about social reciprocity we need to  
7 assess if the child, for instance, initiated  
8 interaction or responded to an initiation of  
9 interaction by someone. Then you look at the quality  
10 of the interactions and how it goes on back and forth.  
11 That's the quality that we want to evaluate.

12 So that the child initiated at one point  
13 behavior is not evidence that there is good quality or  
14 good reciprocity in social interaction. That's one  
15 aspect.

16 And the same for babbles. For instance,  
17 there are some utterances, babbles or even words which  
18 are used you need to assess how spontaneously they are  
19 used by the child, do they have a communicating  
20 function, is there communicating intent, and what  
21 happens in the event they're responded to and is there  
22 really a conversation or interchange when the child  
23 babbles or a child who has a few words. So you need  
24 to assess these qualities otherwise it can be  
25 misleading.

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1           So I think that we would represent a few  
2 clips again and look at them in a different way.

3           I also wanted to correct some comments that  
4 Dr. Mumper made about object permanency. Object  
5 permanency has to do with cognitive development.

6           Q     I'm sorry; what?

7           A     Object permanency. When she was looking at  
8 Jordan King dropping the toys and he was looking at  
9 his toys, that has nothing to do with object  
10 permanency. This is a concept that has to do with  
11 cognitive development in children which is assessed  
12 when you present an object to a child and then you  
13 remove it from his visual field and then you see if he  
14 is looking for this object once it has disappeared.  
15 In that particular case comments are not appropriate  
16 to what we saw.

17           And that -- yes?

18           Q     Any other general comments before we look at  
19 the clips?

20           A     The other comments is that I think the  
21 debate was that their evidence of abnormal development  
22 before the regression, okay, we determined that the  
23 child was developing absolutely normally up to the  
24 point of regression. We can re-discuss this issue  
25 about the timing of regression later. But I think

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1 it's, again, Dr. Mumper should know that we in the  
2 field do not consider that we can actually detect  
3 abnormalities in most children with autism before the  
4 age of 12 months. So her showing clips at 3 months or  
5 5 months of age are not informative at all because  
6 it's not a period of the development where you would  
7 expect to pick up the specific abnormalities seen in  
8 autistic children.

9 We have ongoing, it's documented in so many  
10 studies that I don't want to overwhelm you with the  
11 literature, but we have ongoing prospective studies  
12 where we follow children who are siblings of already  
13 diagnosed children. And this is an ongoing project in  
14 which there are several teams worldwide. And so the  
15 siblings, a proportion of which is as high as 15  
16 percent, would later develop autism, is followed from  
17 birth. And later we have an opportunity to observe  
18 prospectively the development in order to identify the  
19 first signs of what will become autism in some of  
20 them. And up to the age of 10 months, 11 months we  
21 usually when we compare them to those who will not  
22 develop autism or to typically developing children we  
23 don't find much even with standardized assessment  
24 procedure.

25 It is mostly around the age of 12 months

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1 that we start to see subtle abnormalities in social  
2 communication which indicates autism but not before  
3 that age. So I think using clips up to the age is not  
4 evidence of anything.

5 Q Would you like to play some of the clips  
6 that we saw?

7 A Yes. So maybe we should go with maybe, I  
8 don't know, we should go to Jordan King's?

9 Q King's first. How about Number 4, "Plays  
10 with Cat"?

11 A Number 4, yes. And let me before seeing the  
12 tape, what is important because this is an example of  
13 an observation that would fool many people who don't  
14 look at the right things. And it's natural. I just  
15 want to draw the attention of the Masters of the  
16 amounts of vocalization that the child is producing  
17 during that clip, and also to look at how he interacts  
18 with others who are around him and does he pay -- for  
19 instance does he orient to them, does he respond, does  
20 he give eye contact to any of them, does he produce  
21 any vocalization or any gesture? That is the kind of  
22 thing that we would like to see that a normal child in  
23 that circumstance should have showed.

24 So let's have it.

25 Q And just Dr. Mumper identified this segment

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1 as Jordan's age approximately 13 to 16 months old?

2 A Yes.

3 Q Would it be possible to use Number 4.

4 (Jordan King Video Clip No. 4 played.)

5 A So he's interested in the cat, absolutely.

6 But he doesn't really look at his parents or look at  
7 the grandmother. He will go and follow the cat, which  
8 is very interesting.

9 You see, she approaches, she touches him, he  
10 doesn't really give eye contact at any time. And  
11 there is no babble, no vocalizations at all.

12 I want to say that it's a small thing but I  
13 want to remind here that the father wrote he was never  
14 a babbler. These video are highly consistent with  
15 what the parents reported at the time. And just as a  
16 point, I reviewed all the videos of Jordan King, I  
17 have never heard one word. There is no word that he  
18 used, very few vocalizations. When there are a few  
19 vocalizations they are usually not socially directed  
20 and their communicative intent is dubious.

21 Q The next clip I would like to play Number 5.

22 A On the next clip this clip was used as  
23 evidence of his gesturing by Dr. Mumper. And you will  
24 see a partial gesture. But what matters is the  
25 quality and the spontaneity of gesture. And here you

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1 will see that indeed he's responding to another,  
2 trying to engage the other, opens his arms. And it's  
3 to that initiation by the other of this movement that  
4 Jordan starts to respond. And we will see the  
5 response is actually partial, he doesn't really get --  
6 doesn't complete the gesture.

7 But what is important for us when we assess  
8 these tapes is to look at the spontaneous initiation  
9 of communicative acts by the child. This is not  
10 spontaneous, he is responding partially to the  
11 initiation of an adult.

12 ( Jordan King Video Clip No. 5 played.)

13 A You see, she engages him and then, then he  
14 responds. But it's not, it's not initiated by him.  
15 And this is a quality that you need to assess.

16 Do we have some clip 9?

17 Q Yes, Number 9, "Building the Marimba."

18 A Yes.

19 ( Jordan King Video Clip No. 9 played.)

20 This is the sequence where he is building up  
21 something with his father. And, yes, he is interested  
22 in toys. And again, children with autism usually are  
23 very interested in manipulating toys, doing physical  
24 activities, so this type of physical functional play  
25 is often present. And what is lacking is imaginative

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1 play or creative play at this stage. So the fact that  
2 he is interested in toys or musical objects is  
3 absolutely fine and doesn't rule out autism at all.

4 But I think on that tape, on that clip again  
5 let's look at the amount of vocalization that the  
6 child used. Does he direct the attention of his  
7 father to something that he does? Does he respond?  
8 Is there interchange between the two? You will see  
9 there is not much, it's more the father is here, he's  
10 there observing it what he does, and then following  
11 sort of an agenda in playing with his toys, but there  
12 is no really social interaction which I think that's  
13 quality which is not there.

14 ( Jordan King Clip No. 9 played.)

15 You see, he's remarkably quiet. I mean  
16 there are a few, a few moments where his father  
17 imitates the drill and he copies that, but he doesn't  
18 progress. There is no more vocalization and then he  
19 doesn't initiate any attempt to an interaction with  
20 his dad. Just he follows passively. And there is no  
21 words here heard at all.

22 Q Now, Dr. Mumper described this clip as  
23 showing that Jordan was seeking approval from his  
24 father. Do you see that in this clip?

25 A Approval for?

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1 Q She just said it was evidence of normal  
2 behavior seeking approval from his father.

3 A No, I don't think it is. That's an  
4 inference which cannot be held. I think this is  
5 subtle. I appreciate that for most people it would  
6 subtle deficits. But again it's the pattern which is  
7 consistent across all videos which I have seen. And  
8 again I have not heard any word to the fullblown  
9 autism. And he's quiet, doesn't vocalize. When he  
10 vocalizes it's very limited, it's not used really to  
11 communicate socially, and he usually doesn't  
12 reciprocate with vocalization.

13 Q All right. I believe there's another clip  
14 that we wanted to show that we had previously  
15 designated of Jordan King.

16 A Yes.

17 Q And I'm referring to Number 2 on our  
18 designation, disk one, file one. The time period is  
19 1999, January through June.

20 A This is just on that clip, if I recall, he  
21 is playing, he has a pacifier which indeed comes into  
22 the way of babbling. But the thing to look at is that  
23 his mother is filming him. She made comments several  
24 times. She calls him. And he doesn't, he never  
25 orients to her at all. So he doesn't look at her, he

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1 doesn't orient to her despite her calling him several  
2 times. And in terms of his social behavior he's  
3 manipulating toys in an appropriate way but you have a  
4 sense that he's following his own agenda and he's  
5 really on his own world in some way.

6 SPECIAL MASTER HASTINGS: What age is Jordan  
7 in this video you are about to show?

8 THE WITNESS: This would be before the 18  
9 month mark. I don't --

10 MS. RICCIARDELLA: The video just indicates  
11 we have sometime between January and June of 1999.

12 SPECIAL MASTER HASTINGS: All right. Oh,  
13 this is the one from January to June. Okay.

14 MS. RICCIARDELLA: Number 2 on our file one.

15 SPECIAL MASTER HASTINGS: Right, right.

16 Thank you.

17 MS. RICCIARDELLA: Disk one.

18 (Pause.)

19 SPECIAL MASTER CAMPBELL-SMITH: The silence  
20 is the anticipation of getting this technically done.

21 MS. RICCIARDELLA: I should make that clear,  
22 yes. We're having some technical issues.

23 (Pause.)

24 MS. RICCIARDELLA: It appears to be working  
25 on our computer but not on the Court's computers.

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1 MR. MATANOSKI: It appears we're going to  
2 need a minute.

3 SPECIAL MASTER HASTINGS: All right.

4 MR. MATANOSKI: Apologize.

5 SPECIAL MASTER HASTINGS: Let's go off the  
6 record for a minute.

7 (Discussion off the record.)

8 SPECIAL MASTER HASTINGS: Let's go back on  
9 the record.

10 MS. RICCIARDELLA: It plays on our computer  
11 but it's not playing for the Court's computers.

12 BY MS. RICCIARDELLA:

13 Q Would you just please describe, and we'd  
14 just ask that the Court pay particular attention to  
15 this particular designation in the King case, would  
16 you please describe why you selected this to show to  
17 them?

18 A As I said before it's a sequence where he's  
19 playing alone, manipulating objects of different kinds  
20 appropriately. But his mother is filming and trying  
21 to engage him socially by calling him, making  
22 comments. And at no point in time does he orient  
23 towards his mother as you would expect. So is lack of  
24 social response which is a good characteristic and  
25 which in the case of Jordan really emerged at the

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1 beginning of the second year of life.

2 It is true that there has been, when I  
3 reviewed the tapes, as a child when he was like 9  
4 months, 10 months he was really much more socially  
5 engaged, eye contact was much better. But as you move  
6 on in time you see that the eye contact is slowly  
7 disappearing, that he's less responsive, much more  
8 following his own agenda in what he does, and there is  
9 therefore gradual onset of autistic symptoms. But it  
10 doesn't occur like overnight by a loss of skills.

11 And I think the other thing I want to  
12 reemphasize is that both by parental descriptions and  
13 the father's descriptions and by my own observations  
14 of the video he's a remarkably quiet, not using  
15 vocalizations as a normal baby would do, and has no  
16 words at all. So I do not doubt that he had maybe a  
17 few words at one point that he might have used once or  
18 twice in very highly -- in highly contextualized  
19 fashion, but it's not a child who had developed  
20 language properly and for me it's very clear.

21 SPECIAL MASTER HASTINGS: Dr. Fombonne, let  
22 me ask again about the particular segment that you  
23 were just describing and you weren't able to play for  
24 us. But did you say that what time frame you were  
25 assuming this video was taken, what age?

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1 THE WITNESS: I think it's about like 15  
2 months of age, about that. I've been trying to look  
3 at clips which will be occurring before the 18 month  
4 mark.

5 SPECIAL MASTER HASTINGS: Before the 18  
6 month.

7 THE WITNESS: Yes. Because it seems to be.

8 SPECIAL MASTER HASTINGS: But and the reason  
9 you're concluding this was about 15 months is it  
10 because it came off a particular tape?

11 MS. RICCIARDELLA: Yes.

12 THE WITNESS: Oh, yes.

13 SPECIAL MASTER HASTINGS: I mean how do you  
14 know this is at 15 months?

15 THE WITNESS: Let me see. Yeah, it's a  
16 tape, it's a tape which is in 1999 from January to  
17 June.

18 SPECIAL MASTER HASTINGS: Okay.

19 THE WITNESS: And in that case it's in the  
20 early part of the tape.

21 SPECIAL MASTER HASTINGS: Okay.

22 THE WITNESS: So that's, I don't think I  
23 could come up with a precise date otherwise I would  
24 have noted that. But it's probably around that time.

25 SPECIAL MASTER HASTINGS: So you're

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1 surmising from where it is on the tape, it's at the  
2 beginning of that tape?

3 THE WITNESS: Yes. Yes, it's at 20 minutes  
4 after the beginning of the tape.

5 SPECIAL MASTER HASTINGS: And how long is  
6 that tape, is that 4 hours? Did anyone say how long  
7 that tape was?

8 THE WITNESS: It's at least going up to 48  
9 minutes. But probably more than that. It's probably  
10 one hour long, yes.

11 SPECIAL MASTER HASTINGS: All right.

12 MS. RICCIARDELLA: And it's disk one, file  
13 one.

14 SPECIAL MASTER HASTINGS: Right.

15 MS. RICCIARDELLA: Is the designation of the  
16 cue.

17 THE WITNESS: But if I may explain on that,  
18 it's again it's the consistency of observations across  
19 different clips at different ages before that age or  
20 before the 18 month mark which is what I rely upon for  
21 my opinion.

22 BY MS. RICCIARDELLA:

23 Q Doctor, I'd like to turn to the videos of  
24 William Mead. If we could go back to them.

25 SPECIAL MASTER HASTINGS: Before you go on

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1 to William Mead let me ask one more question about  
2 Jordan King. Do you recall how much video of Jordan  
3 King you witnessed, how many hours' worth?

4 THE WITNESS: Probably 10, 12 hours, 15  
5 hours.

6 SPECIAL MASTER HASTINGS: All right.

7 THE WITNESS: Because I did that very long I  
8 sat to review. It's a long, long, long, long time.

9 SPECIAL MASTER HASTINGS: And how much of  
10 that was the pre-18 month period, roughly?

11 THE WITNESS: I couldn't say. I couldn't  
12 say between that, I don't know.

13 SPECIAL MASTER HASTINGS: Half of it, do you  
14 think it was half or at least a substantial portion  
15 or?

16 THE WITNESS: Yes. Oh yes, I would say. I  
17 would say probably, let's say half or it or more or  
18 less. There are long sequences which are also not  
19 informative. You know, there are longer musical  
20 scenes where actually Jordan is not present. But I  
21 had to watch it to be sure he was not there.

22 So anyway, I would say about half of it.

23 SPECIAL MASTER HASTINGS: All right.

24 THE WITNESS: But it's --

25 SPECIAL MASTER HASTINGS: Go ahead.

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1 THE WITNESS: -- half is still lengthy.

2 BY MS. RICCIARDELLA:

3 Q I'd like to turn to the William Mead videos.  
4 Do you have any general comments about the videos that  
5 we saw today with regard to William Mead before we  
6 look at a specific clip?

7 A Yes. Again, all the clips which have been  
8 presented are from birth to 12 months of age. So as  
9 for the reasons I indicated before I don't think this  
10 is very informative for our debate. We will return on  
11 the one taking his bath, for instance, where I could  
12 have a different spin, interpretative spin on what is  
13 presented. But more or less I would think that the  
14 first, this first year of life is not particularly  
15 informative. We will show a clip for which we have a  
16 date, which is 15 months I think exactly, which will  
17 be more informative in terms of showing signs before  
18 the alleged regression at 18 months of age.

19 But before we see it I just want to again  
20 talk about the 18 month time. It's highly  
21 inconsistent in the medical record when the regression  
22 occurred. I know Mr. Mead during his testimony dated  
23 back to 18 months of age the onset of regression or  
24 loss of skills. But it is fair to give some weight to  
25 the medical record evidence because these records are

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1 based on parental reports at the time as well. So  
2 it's parental reports forward. And again the  
3 regression is said to occur in the summer of 2000  
4 several times. And at 18 months of age. And at other  
5 times it's a bit uncertain, again, at what time  
6 exactly the loss of skills occurred. And we will  
7 discuss it by which skills might have been lost in a  
8 minute.

9 So can we just look at --

10 Q The "Bath Time" tape?

11 A Yes.

12 Q It's Number 9, or excuse me, Number 3.

13 A So just to maybe alert you on the types of  
14 behaviors, I think Dr. Mumper used that example to  
15 indicate that William was not hypersensitive to his  
16 head being touched. And, you know, we can discuss  
17 that if we listen to his somewhat negative reaction  
18 with his sister is pulling his hair. But it's not the  
19 point. He does look at the camera with a smile  
20 forward but there is not much of a variation  
21 otherwise.

22 And I want to draw your attention on, again,  
23 the amount of spontaneous vocalization and babble that  
24 William produced during this long scene. And also to  
25 which extent he does or does not relate to his sister.

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1 But relating to him considering his position in the  
2 bath would be to look at his sister and you will see  
3 he doesn't make eye contact really to her at all.

4 (William Mead Video Clip No. 3 played.)

5 You see there is a slight emotion.

6 (Video continues playing.)

7 So he has a, again, there is not much  
8 interaction with his sister using eye contact. He  
9 seems to be responding to his father or engaging him,  
10 and that's fine. But in terms of his spontaneous  
11 vocalization there is not much. I mean there aren't  
12 basically any spontaneous babble coming out of him.  
13 The noise is from his sister and the father but he  
14 doesn't really spontaneous babble.

15 It's, again, it's a kind of observation  
16 which is very technical. Nobody would put too much  
17 weight in the clinical assessment when you observe  
18 that. But it's of note. Also, he had done some  
19 unusual movements in the headline that are generally  
20 brief but they're noticeable.

21 Q We've also selected another clip of William  
22 Mead.

23 A Yes.

24 Q Do you have that ready to go, Brandon?

25 Okay.

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1           A     So this is -- sorry, can you hold onto that?

2                     I think Dr. Mumper presented a clip this  
3 morning where she spoke about social reciprocity and  
4 said that William was playing with his sister. And if  
5 you look again at that clip you will see that William  
6 does not play with his sister. He's there, his sister  
7 is there, and then at one point he goes into the shade  
8 spot alone. She follows him in the shade. He gets  
9 out of the shade but there is no reciprocal play  
10 between the two. It's misconstrued to say that  
11 because there are the two together in the shade at one  
12 point in time that there is reciprocal play between  
13 the two of them.

14                     I will not review that clip but the clip  
15 that you will be presented now is also a clip which  
16 involves him and his sister. As you will see, there  
17 is no reciprocal interactions between the two. When  
18 there are interactions they are initiated by the  
19 sister and William responds but he's not initiating  
20 it.

21                     And secondly, I would like to review it is  
22 18th of July, 1999, so we are there he's 14 months and  
23 a half, sort of, yeah. And again evaluate how much  
24 language he has. Evaluate how much vocalization he  
25 produces, their quality, their communicative function,

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1 and evaluate how he can gesture as well to  
2 communicate. And you will see his father at the very  
3 beginning is asking him to raise his hand. A child  
4 will normally do that, and he just cannot do it. He  
5 doesn't understand then already what is being asked  
6 from him. His sister does it, he doesn't copy her.

7 Q Unfortunately, Dr. Fombonne, I'm getting the  
8 high sign that we do not have that ability to show  
9 that to the Court. However, what we will do is we  
10 will let the Court know what clip and what the time  
11 frame if the Court would like to review what Dr.  
12 Fombonne is talking about. Sorry.

13 Dr. Fombonne --

14 A So --

15 Q Go ahead, if you would like to further  
16 explicate what is on that clip?

17 A So again, it's 14 1/2 months of age.  
18 William has no words. In reviewing all the tapes of  
19 William I heard two 2-word sentences, "Hi, Dad." which  
20 has been shown this morning which is not presented as  
21 a 2-word sentence, "Hi, Dad" is like one word. And  
22 then "mac cheese." "Mac cheese" he says in a sort of  
23 meal that he takes with his sister. This is the only  
24 word utterances which are present on the tapes.

25 Other than that, and this tape at 14 months

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1 and a half of age is clear in showing that at that age  
2 he is not babbling, he is not communicating, he is not  
3 gesturing really, he cannot copy a gesture, he cannot  
4 respond to his dad, he doesn't play reciprocally with  
5 his sister. It would be obvious to every person who  
6 knows a child of 15 months.

7 Q Doctor, there's been a lot of discussion  
8 this morning about the age when Jordan King and  
9 William Mead allegedly went into a regression. Do you  
10 have any further comments about the age of the onset  
11 of the regression in both little boys?

12 A No, as I said, you know, I was hoping we  
13 would not go into the video exercise, but the video  
14 just as clearly shows to me that both boys were  
15 abnormal and that they were abnormal before the  
16 regression or the loss of skills which occurred maybe  
17 at 18 months of age. I think there is an  
18 inconsistency of reports in the case of William Mead  
19 in particular that when exactly he lost his skills. I  
20 don't dispute that he lost skills. That's fair.

21 I want also to say that those children who  
22 do have regression of language usually have reached  
23 the language developmental stage which is not very  
24 advanced. They have usually five words, 20 words  
25 maximum, or 30 words sometimes. It's very rare that

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1 they will have 60 words or that they'll have phrase  
2 speech including a verb and like "I want" something or  
3 "I see the horse." So the kind of language that  
4 William has been presenting as having before the loss  
5 I have some doubts that he had that level of language.

6 So to me the tape of 15 months of age shows  
7 that he had no language of that type at that age. And  
8 for those who are parents, like me, if you have a  
9 child with 60 words and who is speaking 3-word  
10 sentences and you lose that skill you go to the  
11 emergency room and you see a pediatrician right away.  
12 So this kind of loss would be dramatic, observable and  
13 would precipitate an immediate consultation with a  
14 neurologist.

15 Q Did you see anything in the medical records  
16 or the videos that William suffered such a dramatic  
17 observable loss?

18 A No, no. Because as we said previously,  
19 William's pediatricians note a delay, delay lack of  
20 speech at age 2 I think, as I recall. I mean had he  
21 lost 60 words at age 18 months and 3-word sentences  
22 that would have been followed by some kind of medical  
23 consultation.

24 So I think it's not to -- I don't want to  
25 dispute any further what is the true reality; it's

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1 difficult. I think recall by parents is tainted by  
2 too many expanse, it's hard retrospectively to time  
3 with accuracy this phenomenon. It's true in autism,  
4 it's true for regression, it's true for neural  
5 disorders. So we know that. So I think that there is  
6 an area of difficulty in terms of assessing  
7 retrospectively the timing of loss of skills or  
8 emergence of skills as well. It's very hard.

9 But it's very clear for me that William, I  
10 see no evidence that he had normal language  
11 development by 15 months of age and he had no  
12 gesturing and no vocalization of the kind that you  
13 would expect to be for a 15-month old. So I think we  
14 can safely conclude that in his case there was a  
15 progressive, gradual onset of autistic symptoms which  
16 emerged more saliently over a period of time. That's  
17 the experience of parents. That's why it's very hard  
18 to point at a particular date. It doesn't happen  
19 overnight, it's a progressive change in the child.

20 Q And what about Jordan King, would you say  
21 the same for Jordan King?

22 A Yes. Yes, very much so.

23 MS. RICCIARDELLA: Thank you. I have no  
24 further questions.

25 SPECIAL MASTER CAMPBELL-SMITH: Any

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1 questions from Petitioners' counsel?

2 MR. POWERS: Yes. Thank you, Special  
3 Masters.

4 CROSS-EXAMINATION

5 BY MR. POWERS:

6 Q Good afternoon, Dr. Fombonne.

7 A Good afternoon.

8 Q I think I will be brief here, just a few  
9 questions. You mentioned in your earlier testimony  
10 today some questions about the types of treatment, the  
11 medical care and treatment and Jordan King and William  
12 Mead got. There's no evidence in the medical records  
13 and no testimony that you're aware of indicating that  
14 the medical care that these boys received caused them  
15 any harm, is there?

16 A No. No.

17 Q And it's fair to say that the parents and  
18 the treating physician Dr. Green both report  
19 improvements. Now, I understand that I'm not asking  
20 you to attribute it to anything, but the record is  
21 that the parents and the treating doctor both noted  
22 improvements; correct?

23 A Yes. But can I comment on the meaning of  
24 these improvements?

25 Q No, that wasn't my question. My question

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1 was what the parents reported and what the doctor  
2 reported.

3 A Yes.

4 Q I think you've already given your opinion on  
5 the nature of improvement.

6 A Okay.

7 Q Is it your testimony that both boys actually  
8 did regress?

9 A I think they lost skills probably, yes,  
10 absolutely.

11 Q And they lost skills in all three  
12 developmental domains that are relevant to an autism  
13 diagnosis?

14 A I cannot assess that based on the records or  
15 the videos. It's not clear.

16 Q Now, we heard representations this morning  
17 when Dr. Mumper was being cross-examined that Dr. Rust  
18 agreed that both of these boys have been diagnosed  
19 with regressive autism and that he agreed with that  
20 diagnosis. Do you agree or disagree with Dr. Rust  
21 that these boys have regressive autism?

22 A I disagree with the fact that regressive  
23 autism is not a diagnosis.

24 Q Do you agree with Dr. Rust's  
25 characterization of both of these boys having

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1 experienced autistic regression?

2 A That's yes.

3 Q In describing some of your descriptions of  
4 William Mead, and particularly William Mead's video,  
5 you used the term "this is my interpretative spin."  
6 Is that a correct characterization of your analysis as  
7 you testified here today that it's, as I wrote down,  
8 your interpretative spin?

9 A I think for the reasons I mentioned before  
10 the clips in a very young child are very difficult to  
11 interpret because some children, for instance, have  
12 several type movements of the body which are brief, so  
13 it's hard to interpret what we see with some stronger  
14 conclusions particularly. That's why we do not pick  
15 up abnormalities in the siblings of autistic children  
16 before age 12 months. So I'm just presenting that  
17 tape to show where this boy even at that very early  
18 age he's not vocalizing much, he's not directing  
19 babble to anyone, he's not looking at his sister.  
20 These are observations but I'm cautious about what  
21 kind of inferences I would draw from them because of  
22 the narrow behavioral repertoire which exists at a  
23 very young age.

24 Q And would it also be fair to say that any  
25 particular analysis opinion in looking at these tapes

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1 is going to be somewhat subjective in that one  
2 particular reviewer might look at a particular video  
3 and there could be different conclusions that are  
4 reached based on one's interpretative spin; correct?

5 A No. No. It would not be true for video  
6 clips which are when the child is older. When the  
7 child is older and you don't see any communicative  
8 attempt, no gesturing, no response to the name being  
9 called, no vocalizations, no pointing, no copying of  
10 gestures, this is quite robust.j

11 Q At what point in a child's life can video  
12 analysis move from the realm of interpretative spin  
13 into objective analysis in your opinion?

14 A Well, the results show actually that the  
15 analysis of home videos show good prediction of later  
16 diagnosis starting at the age of 10 or 12 months. In  
17 some studies it's earlier but most studies it's about  
18 10, 12 months of age.

19 Q When you say that these boys are abnormal  
20 are you describing that they were in the bottom 2.5  
21 percent of their age cohort at any particular point in  
22 time?

23 A On which domain?

24 Q In any of the domains.

25 A I cannot, I cannot make a comment on that

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1 based on what evidence I have.

2 Q I ask that because in my understanding of  
3 looking at a distribution of development over time  
4 there's a bell curve; is that correct?

5 A Uh-huh. Yes. Sorry.

6 Q Yeah, I knew that you were saying "yes" but  
7 we have to have it out loud.

8 A Yes.

9 Q And there's a median; correct?

10 A Yes.

11 Q And the normalcy or abnormalcy or the  
12 closeness of a child to the median is often measured  
13 in standard deviations; correct?

14 A Yes, correct.

15 Q And two standard deviations is typically  
16 what is used to evaluate abnormal, that is the 2.5  
17 percent at the tail end of both sides of the bell  
18 curve those would be the abnormal numbers; correct?

19 A Correct.

20 Q So that's why I'm asking, can you tell the  
21 Special Masters whether Jordan King or William Mead in  
22 their overall development were in the bottom 2.5  
23 percent of their age cohort?

24 A I would say probably in terms of language.  
25 You would have to refer to a standardized test. You

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1 need to establish that to have standardized tests of  
2 language development. One of them which is used is  
3 called the Communicative Development Inventory, the  
4 CDI, from MacArthur which has norms for language  
5 development which are separate for boys and girls. At  
6 the age of 15 or 18 months these boys should have had  
7 more language than they have in terms of words.

8 Q How about in the other domains, are they in  
9 the bottom 2.5 percent of their age group at any point  
10 in their first year of life in, say, social  
11 reciprocity?

12 A There is no good instrument to evaluate  
13 social reciprocity. We don't have norms for that. So  
14 that's the only instrument which can assess that is  
15 the environment, the scale which have scores which  
16 give you a communications score, social interaction  
17 score. But at that it's apt to be unreliable. And in  
18 order to get a score like that you will have to be  
19 present at the time and to have administered the  
20 instrument at that time. So I cannot --

21 Q And so the answer would be you don't know  
22 whether they were in the bottom 2.5 percent.

23 A Yes, I don't know for the other domains, no.

24 Q Okay.

25 A But for, again for language, yes, and for

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1 gestures as well.

2 Q And then again you do not disagree that both  
3 of these boys experienced an autistic regression;  
4 correct?

5 A No, I don't. No.

6 MR. POWERS: Thank you. No further  
7 questions.

8 SPECIAL MASTER CAMPBELL-SMITH: Any  
9 questions from my colleagues? Nothing?

10 MS. RICCIARDELLA: I just have a couple  
11 more.

12 REDIRECT EXAMINATION

13 BY MS. RICCIARDELLA:

14 Q Dr. Fombonne, Mr. Powers was using the  
15 phrase "interpretative spin" because you used that in  
16 yours, and he kept throwing it back at you. When you  
17 review a videotape, Doctor, what skills do you apply  
18 when you are looking at a videotape?

19 A My observations and my vast clinical  
20 experience and being trained to measure the ADOS,  
21 which is an observational measure. We develop I think  
22 particular accuracy to look at situations there you  
23 have a pressure on the child to communicate or to  
24 gesture or to request something, and we develop this  
25 kind of experience based on our training and number of

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1 ordinary children which we have seen or untypical  
2 children as well.

3 Q Now, Doctor, Mr. Powers also asked you  
4 whether an analysis of a videotape can be somewhat  
5 subjective. Now, Dr. Mumper is using the videotapes  
6 today as saying that she can rely on the videotapes to  
7 show normalcy or typicality during the first 12 to 15  
8 months of these two little boys' lives. Are  
9 videotapes a reliable source to show typical behavior?

10 A Typical? What do you mean typical?

11 Q Do clinicians use videotapes to actually  
12 diagnose autism or as evidence to show that a child is  
13 developing typically?

14 A No. Because they -- first, when we code,  
15 when we use video for research purposes we have coding  
16 schemes which are extremely precise. So we look at  
17 sequences, we rate particular behaviors according to  
18 rules. We take into account the amount of time of the  
19 tape because obviously if you have a tape which is  
20 very long we need to take that into account because it  
21 gives you more opportunity to observe abnormal or  
22 normal behavior. So there are a lot of rules which  
23 are followed in the research that of course we cannot  
24 follow here.

25 I would say my clinical practice I often see

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1 parents come in with films or videos or children.  
2 And, you know, when you see a child who is young and  
3 who is normal it's often not always informative  
4 because there are some clinical deficits which can  
5 occur but in particular situations which have not been  
6 filmed by the parents. So I think if you have on the  
7 contrary a situation which is consistent where you  
8 don't see skills that you would expect to find in the  
9 child, then we can give some credence to these  
10 observations. But you don't diagnose a child based on  
11 retrospective video assessment for clinical reasons.  
12 For research it has been used, not for clinical  
13 reasons.

14 MS. RICCIARDELLA: Thank you.

15 SPECIAL MASTER CAMPBELL-SMITH: Anything  
16 further?

17 MR. POWERS: No, not from us.

18 SPECIAL MASTER CAMPBELL-SMITH: Thank you,  
19 Dr. Fombonne.

20 THE WITNESS: Thank you.

21 (Witness excused.)

22 SPECIAL MASTER CAMPBELL-SMITH: Does  
23 Respondent's counsel have any additional witnesses to  
24 call?

25 MR. MATANOSKI: Yes, ma'am, we do. At this

1 time we call Dr. Jeffrey Johnson.

2 MR. WILLIAMS: While they're setting up I  
3 want to pose just a fairness objection. Last Friday  
4 we discussed that we were going to deal with Dr. Deth  
5 issues on Thursday. And we arranged for Dr. Deth to  
6 be here all day yesterday. And they had no one to  
7 call yesterday in response to Dr. Deth. And now I  
8 guess, I assume that this is what we are going to hear  
9 now.

10 And I just put that on the record as a  
11 fairness objection. And we may want to seek relief  
12 for it depending on what happens.

13 SPECIAL MASTER CAMPBELL-SMITH: Mr.  
14 Matanoski?

15 MR. MATANOSKI: Thank you. Actually, ma'am,  
16 Dr. Johnson is going to be responding to the rebuttal  
17 testimony of Dr. Kinsbourne this morning.

18 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

19 Dr. Johnson, as a preliminary matter, you  
20 are still under the oath that you took earlier in this  
21 proceeding.

22 DR. JOHNSON: Absolutely.

23 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

24 //

25 //

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1                   Whereupon,

2                                   JEFF JOHNSON

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

6                   SPECIAL MASTER CAMPBELL-SMITH: We're having  
7                   a technical adjustment again.

8                   MS. BABCOCK: Seems to be an afternoon of  
9                   technical difficulties.

10                  SPECIAL MASTER HASTINGS: While we're  
11                  waiting for the technical difficulties, a technical  
12                  issue I wanted to address. Last year during the  
13                  Sabile hearing we also heard video reviews. We  
14                  decided as direction for the court reporting service  
15                  that we didn't want them to -- there was no need for  
16                  them to transcribe all the words that were said during  
17                  the video by the parents or just today the parents of  
18                  both children, and we had also William Mead and his  
19                  sister say a few words. I think I hope we are all in  
20                  agreement, they don't need to transcribe that part.

21                  MR. POWERS: Yes, sir.

22                  SPECIAL MASTER HASTINGS: Okay. I just  
23                  wanted to clarify that for the court reporter  
24                  especially.

25                  SPECIAL MASTER CAMPBELL-SMITH: Technical

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1 matter is resolved. Respondent's counsel to proceed.

2 MS. BABCOCK: We'll discover as we go along,  
3 I suppose.

4 DIRECT EXAMINATION

5 BY MS. BABCOCK:

6 Q Could you please state your name for the  
7 record?

8 A Dr. Jeff Johnson.

9 Q And since there may be some confusion, Dr.  
10 Johnson, are you a neurotoxicologist?

11 A Yes.

12 Q Now, Dr. Kinsbourne spent some time this  
13 morning emphasizing that he was putting forth a  
14 hypothesis or model that could explain how TCVs cause  
15 autism. What is the scientific community's  
16 understanding of the terms "hypothesis" or "model"?

17 A Well, in the context that I would put that a  
18 hypothesis is something where you put together certain  
19 aspects and certain ideas that you see in the  
20 literature, put it together and formulate a hypothesis  
21 that you think might be relevant. And, you know, 99  
22 percent of the time it could be completely wrong. And  
23 if you're lucky, I mean very lucky usually in science  
24 you might actually think of something that might be  
25 correct.

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1 Q And does the same apply to a model?

2 A Oh yeah. The model, developing a model, I  
3 mean I could go back to my office and develop ten  
4 models tomorrow, you know, and none of them could be,  
5 you know, right or wrong, depending on the science.  
6 But I mean it's something that you can, anybody can do  
7 that.

8 Q And both a hypothesis and model would  
9 certainly require testing before any real credence  
10 could be given to them; correct?

11 A Oh, absolutely. I mean if all my hypotheses  
12 worked I would have cured Alzheimer's, Parkinson's,  
13 ALS and every other disease I study.

14 Q Now, do you agree that what Dr. Kinsbourne  
15 has put forth would be considered a hypothesis?

16 A Yes.

17 Q And just as a clarification, do you think  
18 this would rise to a level of, say, more than likely  
19 than not true, to be true?

20 A Oh, absolutely not. It's at the lowest  
21 level.

22 Q Now, is neuroinflammation involved in other  
23 neurological diseases?

24 A Yes. It's involved in almost every  
25 neurodegenerative disease that's been looked at at

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1 least to some extent, including Alzheimer's,  
2 Parkinson's, Huntington's, ALS.

3 Q And you study these diseases; correct?

4 A Yes. Yes, I do. Yes.

5 Q Including both you have a laboratory and an  
6 academic practice and research?

7 A Yes, absolutely.

8 Q Does current research indicate that  
9 neuroinflammation is involved as playing a causal role  
10 in these neurodegenerative diseases?

11 A In general the concept in these other  
12 neurodegenerative diseases is that the  
13 neuroinflammatory response in astroglialosis and  
14 microglial activation are part, a progressive part of  
15 the disease, the progression part of the disease as a  
16 result of the pathologic process. I don't think that  
17 anybody at least in the field would argue that they're  
18 a causative factor at this point, it's more an  
19 outcome.

20 Q Now, does treatment of symptoms via drugs or  
21 clinical trials necessarily implicate a cause of the  
22 disease?

23 A Absolutely not. And I think one of the key  
24 examples of that is in Alzheimer's disease. In  
25 Alzheimer's disease the patient manifests a lot of the

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1 symptoms that, if you're familiar with, and a lot of  
2 people have seen Alzheimer's patients, so you see  
3 these symptoms. And a lot of the symptoms in  
4 Alzheimer's disease are due to a loss of a specific  
5 neurotransmitter called a acetylcholine. And so a lot  
6 of the drugs, most of the FDA-approved drugs for  
7 treating Alzheimer's disease actually increase those,  
8 the levels of acetylcholine in the brain. So the  
9 patient cognitively appears to get better. But in the  
10 background the pathologic process and the mechanism  
11 that's killing the cells is continuing on unabated.

12 So treating symptoms is a way to, is a thing  
13 that you can do. Such as if neuroinflammation is part  
14 of the progression if you can treat that then you may  
15 alleviate some of the symptoms. But none, there's  
16 really no causal, direct causal association with  
17 treating symptoms and what's causing the disease.

18 Q Now, Dr. Kinsbourne also discussed a paper  
19 on his rebuttal today by Dr. Lopez-Hurtado which I  
20 believe is PML-446. You were also asked about this  
21 paper during cross-examination. So obviously I assume  
22 you've got some familiarity with it. In your review  
23 did you identify a fairly significant methodological  
24 flaw used by those authors?

25 A Yeah. When I asked about this I said I

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1 hadn't had time to evaluate the data. Now, I have.  
2 And so there is a significant issue that I have with  
3 the paper. And that is simply this, and I want to try  
4 to be very clear on this. They do a lot of  
5 statistical analysis in this paper comparing one  
6 sample to another sample and things like that. But  
7 that cannot be done because really what they've done  
8 in this paper is they've actually counted the density  
9 of neurons in one brain. So just to give you an idea,  
10 so you take pieces, different layers of the brain and  
11 you count, you know, five different parts let's say,  
12 and you get a number from each of those five parts.  
13 And you average that and you get one number.

14 Now, the standard deviation that you  
15 generate from averaging those five numbers is  
16 basically a standard deviation generated by your error  
17 in counting. It has nothing to do with standard  
18 deviation between samples. And so when you finish  
19 this analysis what you end up with is you end up with  
20 a variety of numbers that you pool together to get the  
21 density of cells in the cortex or in a specific region  
22 for one person.

23 So with the one person you cannot generate a  
24 standard deviation or an inter-individual standard  
25 deviation to run statistics on. So this whole paper

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1 basically uses N's of one to run statistics and the  
2 standard deviations are based exclusively on the  
3 reproducibility of their techniques for counting but  
4 nothing to do with, say, the average of four autistic  
5 brains, four different brains. They don't do that.

6 And so the statistical analysis is really  
7 invalid.

8 Q So let me see if I can boil this down. They  
9 use standard deviation with their graphs but they  
10 shouldn't have?

11 A No, no they shouldn't have because there  
12 really is no standard deviation for an N of one.

13 Q And what's the effect if you take out the  
14 standard deviation?

15 A Well, they can't run any of the statistic  
16 inferences that they did. And if you take out, I mean  
17 if you really look at the data, the way that you can  
18 look at the data is to actually look at the rate of  
19 change with age with regard to glial cell number and  
20 neural cell number and micro -- or the lipofuscin  
21 containing cell number. And if you do look at that  
22 you actually see that the rate of change in the  
23 control patients and the rate of change in the  
24 autistic patients is almost exactly the same.

25 So as the autistic patients age the number

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1 of glial cells go up. As the normal patients age the  
2 number of glial cells go up and those lines are  
3 basically exactly parallel. So to me what that says  
4 as an interpretation of that data which wasn't done in  
5 the paper is that the main difference between an  
6 autistic patient and a normal patient is not the  
7 change the differential during the time or the age,  
8 it's actually the baseline where they start.

9 So if the autistic patients starts with a  
10 higher number of glial cells initially then their rate  
11 of change as they age is going to be, it's just going  
12 to be parallel to what you see in the normal patient.  
13 So I don't want to make this complicated. What I am  
14 saying is as you really look at the data as an age-  
15 dependent process there doesn't seem to be any  
16 difference. And it appears to be the baseline,  
17 probably from the developmental standpoint and what  
18 was laid down during development that's giving you  
19 this differential effect as you look across these  
20 patients of aging.

21 Q Now, Dr. Kinsbourne also discussed  
22 astrocytic function this morning. Do you study  
23 astrocytic function in your laboratory?

24 A Yes. A lot.

25 Q And have you published on this topic?

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1 A Yes.

2 Q In the last, about how many papers in the  
3 last two years?

4 A Probably more than ten. I can count them  
5 but I don't want to take the time.

6 Q We certainly understand.

7 If astrocytes are unable to mop up  
8 glutamate, what happens?

9 A Well, the glutamate will interact with the  
10 neurons and cause excitotoxicity.

11 Q So neurons die?

12 A Eventually, yes.

13 Q And Dr. Kinsbourne also discussed the  
14 Purcell article I believe in his rebuttal, which is  
15 PML-567. I'll let you find it.

16 A I got it. Yes, I have it.

17 Q In autistic brains is there evidence for  
18 increased glutamate transporters?

19 A Yes, absolutely. This article, one of the  
20 things that's interesting about this article is it  
21 does microarray analysis. So what it does is it  
22 actually does gene shift and microarray -- are you  
23 familiar with microarray analysis?

24 SPECIAL MASTER HASTINGS: Can you start that  
25 sentence again?

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1 BY MS. BABCOCK:

2 Q Slowly.

3 A This paper does a microarray analysis and  
4 they look at -- what that is is a fancy way of PCR.  
5 So we go back. What they did is they looked at  
6 microarray analysis, they identified genes that were  
7 different.

8 SPECIAL MASTER VOWELL: Doctor, you're going  
9 to have to slow down.

10 THE WITNESS: Right.

11 SPECIAL MASTER VOWELL: It sounds like  
12 you're saying "micro ray" when really you're saying  
13 "microarray."

14 THE WITNESS: Array; right.

15 SPECIAL MASTER VOWELL: Okay.

16 THE WITNESS: And so the gene chip  
17 basically.

18 And so they identified some candidate genes  
19 that were different between autistic brains and normal  
20 brains or control brains. And some of those genes  
21 were these, we've talked about these EAAT1 and EAAT2  
22 transporters, correct, that transport glutamate into  
23 astrocytes. If you look at the paper, not only did  
24 they identify some of these as being changed but in  
25 fact when they did the RT-PCR and --

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1 SPECIAL MASTER VOWELL: By RT-PCR you're  
2 referring to?

3 THE WITNESS: Looking at messengers.  
4 Messengers on A levels in the brains of autistic  
5 patients.

6 SPECIAL MASTER VOWELL: So reverse  
7 transcript.

8 THE WITNESS: The RT-PCR.

9 SPECIAL MASTER VOWELL: Because we have  
10 heard RT-PCR used in a different context as well, real  
11 time.

12 THE WITNESS: Right.

13 SPECIAL MASTER VOWELL: So what are you  
14 referring to?

15 THE WITNESS: Yeah, I think this -- I don't  
16 know what they did on this. What they might have  
17 done, I don't know if they did real time or they just  
18 did RT-PCR. I think this was just regular RT-PCR, not  
19 real time, not quantitative RT-PCR.

20 But what they, and then what they did is in  
21 addition to the RT-PCR they also did Western Blot  
22 analysis. So they looked at the protein levels in  
23 autistic brains. And in both situations the EAAT1 and  
24 the EAAT2, which are the glutamate transporters on the  
25 astrocytes were significantly increased in autistic

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1 patients. Which to me suggests that the autistic  
2 patient actually has a greater capability to handle  
3 glutamate than the normal patient based on these  
4 studies.

5 BY MS. BABCOCK:

6 Q And are you referring to specific charts or  
7 graphs in that paper?

8 A Yes.

9 Q And if so, could you specifically identify  
10 where they are?

11 A Yes.

12 Q I'm not sure if we have it in trial  
13 directory but at least if you could identify the page  
14 number?

15 A Figure 2. Figure 2 on page 1623 and Figure  
16 3 on page 1624.

17 Q Dr. Johnson, if you have continued chronic  
18 glutamate access would you expect the process to  
19 become neurodegenerative?

20 A Yes.

21 Q Now, Dr. Kinsbourne also discussed Dr.  
22 Aschner's papers this morning, which I believe are  
23 PML-568 and 570. Are you familiar with Dr. Aschner's  
24 work?

25 A Yes. I have known Mickey for a long time.

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1 Q And these papers in particular?

2 A These papers I've looked at, yes. I tend to  
3 not like to look at reviews because I like to under --  
4 I mean I will look at the reviews but then I also like  
5 to find the interesting points of the reviews and go  
6 and look at the real manuscripts and the real data  
7 that actually where they're referring to in their  
8 review. So I've seen a lot of Mickey's original work.

9 Q Now, first, what was the dose necessary to  
10 get astrocytic dysfunction?

11 A It's in the micromolar range in almost all  
12 of his work.

13 Q So that's very high?

14 A Yes, very high.

15 Q Certainly much higher than would be  
16 administered via thimerosal-containing vaccines? And  
17 again I should explain, a different type of mercury  
18 also?

19 A Yes.

20 Q Which is methyl mercury?

21 A Yeah. And I'm qualifying, I mean I'm not a  
22 mercury distribution expert, but from what I've heard  
23 from the testimony and listened to this week I would  
24 say, yes, that seems to be. Because we always talk  
25 nanomolar versus micromolar. Here it's micromolar.

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1 Q Yes. Just limiting you to the dose and I'll  
2 leave the rest to the toxicologists.

3 Now, do you agree that once triggered, as  
4 Dr. Aschner says, a vicious cytotoxic cycle ensues?

5 A I completely agree with that. And we saw  
6 the conclusions I think in cross this morning. Those  
7 are valid and solid conclusions based on the data.

8 Q And is the concept that once you trigger  
9 astrocytic dysfunction you do get that vicious  
10 cytotoxic cycle, is this well accepted in the  
11 scientific community?

12 A Yeah, I would say it's very well accepted in  
13 the scientific community that deals with this kind of  
14 process.

15 MS. BABCOCK: I have nothing further.

16 SPECIAL MASTER CAMPBELL-SMITH: Thank you.  
17 Questions from Petitioners' counsel?

18 MR. POWERS: Yes, thank you.

19 CROSS-EXAMINATION

20 BY MR. POWERS:

21 Q So, Dr. Johnson, you were talking about the  
22 Purcell paper. Ultimately the Purcell paper did  
23 conclude that the involvement of glutamate levels in  
24 the brain is something that ought to be investigated  
25 in autism, and this is what they said in 2001;

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1 correct?

2 A Yeah. No, I'm not saying that glutamate  
3 shouldn't be investigated in autism, glutamate should  
4 be investigated in all of these diseases because it's  
5 clearly been implied to be part of the pathogenic  
6 process.

7 Q And that the blockage of glutamate receptors  
8 might actually improve autistic symptoms, that's one  
9 of the conclusions that the Purcell investigators made  
10 in their paper; correct?

11 A you can conclude whatever you want in their  
12 discussions but I don't know that there's been any  
13 evidence showing that in autistic patients. And again  
14 I'm not a clinician but I know there is evidence in  
15 some of the other diseases that glutamate inhibitors  
16 might have some effect, slight.

17 Q Right. And in the discussion of the Pardo  
18 and Vargas work, the Pardo and Vargas papers do report  
19 chronic ongoing neural inflammation in the brains of  
20 autistic patients; correct?

21 A They show inflammation or they show  
22 astroglial activation and microglial activation in  
23 postmortem brains of autistic patients. That doesn't  
24 mean it's ongoing, that means that it's there at the  
25 time that the patient died.

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1 Q And that was a cross a wide range of  
2 subjects; correct?

3 A Yeah, across a wide range of ages. I don't  
4 remember specifically.

5 Q Roughly 7 to 44; does that sound about  
6 right?

7 A Okay, yeah, maybe something like that, yes.

8 Q And in those frames this endpoint of massive  
9 neuronal death had certainly not been reached;  
10 correct?

11 A I don't -- I'd have to go back and look.  
12 There was something with the Purkinje cells I think.  
13 But outside of that I don't think there was massive  
14 neuronal death in the brain, no, at that point.

15 MR. POWERS: No further questions.

16 SPECIAL MASTER CAMPBELL-SMITH: Thank you.  
17 Any further questions from Respondent's counsel?

18 MS. BABCOCK: Nothing, thanks.

19 SPECIAL MASTER CAMPBELL-SMITH: Any  
20 questions from my colleagues?

21 SPECIAL MASTER VOWELL: I have one follow-up  
22 for Dr. Johnson.

23 THE WITNESS: Sure.

24 SPECIAL MASTER VOWELL: You said chronic  
25 glutamate excess would lead to neurodegeneration.

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1 THE WITNESS: To killing of neurons, yes.

2 SPECIAL MASTER VOWELL: Okay. And is there  
3 any particular reason you say that? I mean you stated  
4 it but you didn't give a reason.

5 THE WITNESS: Well, it's been, I mean we use  
6 it to kill cells all the time. In culture we kill  
7 cells *in vivo* with excitatory cytoamino acid toxicity.  
8 And there's also a lot of evidence in Parkinson's and  
9 other diseases that, you know, this kind of a chronic  
10 glutamate factor, specifically astrocyte dysfunction,  
11 and I'm thinking in mind, specifically in mind to ALS,  
12 that astrocytic dysfunction is a key component in the  
13 presumably cytotoxic death of motorneurons in spinal  
14 chord of ALS. And I think that's been shown.

15 So I mean there is evidence out there in  
16 these other disease states where you have an  
17 astrocytic dysfunction you end up in the end with  
18 neurodegenerative disease or kill-offs of neurons in  
19 that region, depending on where that region is, spinal  
20 chord, cortex, hippocampus.

21 SPECIAL MASTER CAMPBELL-SMITH: Any  
22 questions generated by Special Master Vowell's  
23 questions?

24 MR. POWERS: No, Your Honor.

25 SPECIAL MASTER CAMPBELL-SMITH: Thank you,

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1 Dr. Johnson, you are excused.

2 (Witness excused.)

3 SPECIAL MASTER CAMPBELL-SMITH: Any  
4 additional witnesses to be called by Respondent's  
5 counsel?

6 MR. MATANOSKI: Yes, ma'am. At this time  
7 Respondent calls Dr. Jeffrey Brent.

8 SPECIAL MASTER CAMPBELL-SMITH: Thank you.  
9 And I will just take the opportunity to  
10 remind you, Dr. Brent, that you remain under oath.

11 DR. BRENT: Yes, I understand.

12 Whereupon,

13 JEFFREY BRENT

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

17 DIRECT EXAMINATION

18 BY MS. RENZI:

19 Q Good afternoon, Dr. Brent.

20 A Good afternoon, Ms. Renzi.

21 Q Could you please state your name for the  
22 record again?

23 A Sure. Jeffrey Brent, M.D., J-E-F-F-R-E-Y B-  
24 R-E-N-T.

25 Q Thank you. Dr. Brent, you've heard

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1 testimony this morning that an essential part of Dr.  
2 Kinsbourne's model was that methyl mercury decreases  
3 glutamate uptake in astrocytes; is that correct?

4 A I did hear that testimony.

5 Q Can that have any relevance to the effects  
6 of thimerosal-containing vaccines?

7 A Absolutely not.

8 Q And what is the basis for that, please?

9 A Well, that process of glutamate uptake by  
10 astrocytes and effects of mercurial compounds has been  
11 extremely well studied. And Dr. Kinsbourne referred  
12 to the work of Dr. Aschner who has actually  
13 demonstrated that mercurial compounds will indeed at  
14 sufficient dosage inhibit glutamate uptake.

15 Now, when Dr. Kinsbourne presented his  
16 hypothesis he was asked about whether the doses that  
17 would do that have any relevance to the doses of  
18 vaccine. And he said, I think to his credit, that  
19 he's not a toxicologist and would therefore defer to a  
20 toxicologist about this. Because the issue of dose  
21 obviously here is critical.

22 If you look at the work of Aschner. Bring  
23 that up, please.

24 Q And this would be a different one that the  
25 article Dr. Kinsbourne referred to this afternoon or

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1 this morning?

2 A Right. This is the actual --

3 MR. POWERS: Excuse me, Special Masters.

4 I'm going to object because this is a dose discussion  
5 that we just heard was raised not in his rebuttal  
6 today by Dr. Kinsbourne but Dr. Brent just referred to  
7 Dr. Kinsbourne's earlier testimony on direct. So  
8 again this is, the dose issue was not discussed by Dr.  
9 Kinsbourne on rebuttal. And when this did come up  
10 during Dr. Kinsbourne's direct and during the cross of  
11 Dr. Brent, he testified back then he was not a -- Dr.  
12 Brent said that he was not a neuroimmunologist. So  
13 this is outside rebuttal and again going back to Dr.  
14 Kinsbourne's direct testimony and going back to  
15 toxicology issues that Dr. Kinsbourne declined to  
16 offer an opinion on. He did talk about dose this  
17 morning.

18 MS. RENZI: I'll let Mr. Matanoski who is  
19 more familiar with Dr. Kinsbourne's testimony today.

20 MR. MATANOSKI: Actually this morning Dr.  
21 Kinsbourne was speaking at length about astrocytes.  
22 And that's what this testimony is going to, it's going  
23 to his description of astrocyte malfunction in his  
24 response this morning to criticism that was levied,  
25 particularly by Dr. Johnson in the Respondent's case-

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1 in-chief, about Dr. Kinsbourne's reliance on this  
2 astrocyte malfunction as a critical element in his  
3 theory. He came back this morning and he rolled  
4 through his astrocyte malfunction argument again,  
5 including reference specifically to the work of Dr.  
6 Aschner to try to tie that in, astrocyte malfunction,  
7 into mercury. That's the purpose of it in his report,  
8 his written report, that was the purpose of it this  
9 morning.

10 SPECIAL MASTER CAMPBELL-SMITH: Let me just  
11 inquire. Dr. Brent, are you referring to testimony  
12 that you heard earlier this morning?

13 THE WITNESS: Yes. I, after hearing the  
14 testimony of Dr. Kinsbourne this morning I suggested  
15 that we could clarify the issue that he raised about  
16 astrocytes and glutamate in my testimony that relates  
17 to this. So I'm specifically referring to the issue  
18 of glutamate uptake by astrocytes that he was  
19 referring to this morning.

20 SPECIAL MASTER CAMPBELL-SMITH: I will allow  
21 the question.

22 BY MS. RENZI:

23 Q I want to refer you to Petitioners' Master  
24 List article 206. And that's another Dr. Aschner  
25 article; is that correct, Dr. Brent?

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1 A That's correct.

2 Q And we're looking specifically at Figure 2?

3 A That's correct. And this is the actual  
4 data, not the review article but the actual data on  
5 effects of methyl mercury on glutamate uptake. And it  
6 should be noted that ethyl mercury has not been  
7 studied in this regard. So everything that we are  
8 talking about here in terms of inferring effects on  
9 glutamate uptake really is based on data from methyl  
10 mercury.

11 But if we look at the methyl mercury data we  
12 see there are two curves on Figure 2. One which is  
13 the round, where the symbols are round circles, and  
14 one where they are boxes. The round circles represent  
15 the inorganic mercury. And as you can see, inorganic  
16 mercury is a little bit more powerful in reducing  
17 glutamate uptake than is methyl mercury you see  
18 effects of lower concentration, the concentrations  
19 being on the X axis.

20 So if we look just at the inorganic mercury,  
21 the first statistically significant point where there  
22 is a decrease in glutamate uptake is indicated by the  
23 first asterisk that you see. And if you follow that  
24 down it's at approximately 2 micromolar. Two  
25 micromolar. So that is the concentration that is

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1 required of inorganic mercury to reduce glutamate  
2 uptake in the astrocytes.

3 Now, if you will remember from prior  
4 testimony, just to put this value in context, the  
5 normal amount of mercury in the brain is in nanomolar  
6 amounts which is 1,000 times less than micromolar.  
7 Quantitatively, 200 micromolar refer, if you do the  
8 calculation, works out to about 400 parts per billion.  
9 Now, we could put that in context of what we would  
10 normally see in the brain. If we could just go back  
11 to a slide that I showed earlier which is from Lapham.

12 Q And this is Respondent's Master List 294.  
13 And this is toxic levels of mercury in the brain in  
14 development studies. And you referred to this in your  
15 direct testimony?

16 A Yes. Yes, I did. And I just want to put  
17 this 400 parts per billion level that is necessary to  
18 inhibit glutamate uptake in the context of what is  
19 actually seen. And if you will remember, as you can  
20 see in the lower three lines, in the general  
21 population, the background level of population, the  
22 amount of mercury in the brain is in the low parts per  
23 billion. You know, anything from 2 or 3 up to about  
24 40 or so. And if we look at the Seychelles study  
25 population where we know there are no adverse effects

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1 demonstrable from mercury and yet they have large  
2 exposure to mercury through seafood, we see that the  
3 amount in their brain is well over 100, 100 or 200  
4 parts per billion without any adverse effect.

5 So clearly the amount of inorganic mercury  
6 that is necessary to inhibit glutamate uptake in  
7 astrocytes, which as we saw in the Aschner study in  
8 their system was at a minimum of 400 parts per  
9 billion, is far above or significantly above the  
10 amount that people normally have in their brains and,  
11 therefore, could not possibly come from the -- could  
12 not possibly be related to anything you would see, for  
13 example, from a vaccine where, if you will remember,  
14 the extra burden in the brain was 2 or 3 parts per  
15 billion.

16 In addition, the Aschner study, remember, is  
17 an *in vitro* study. So as we talk about, and we'll go  
18 over it again, *in vitro* studies are studies where the  
19 substance being studied, in this case mercury, is  
20 simply incubated with the cells you're studying, the  
21 astrocytes, and that therefore they are exposed to a  
22 relatively high concentration of the astrocytes.  
23 Because in the brain if you have 400 parts per billion  
24 it's not, the mercury would not just be sitting there  
25 interacting with the astrocytes, it would be bound to

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1 all the thiols. And the free concentration of mercury  
2 that would be available therefore to interact with the  
3 cells would be very, very, very small, or as Dr. Deth  
4 puts it, damagingly small.

5 So that shows that the amount of mercury  
6 that is necessary to cause this astrocyte effect is  
7 vastly, vastly greater than what could be generated by  
8 a vaccine and far above anything that would be  
9 expected to be seen in normal human experience.

10 Q Thank you. We heard Dr. Mumper's testimony  
11 today that she wasn't certain that Dr. Rust had  
12 actually, actually interviews and gets histories from  
13 his patients. But I'd like to talk a little bit about  
14 how you as a clinician, as a medical toxicologist see  
15 patients. You do regularly see patients, don't you?

16 A Yes. That's what I primarily do.

17 Q And you practice in a university setting; is  
18 that also correct?

19 A Yes. I practice in a university setting and  
20 I have a private setting as well.

21 Q And in both your private practice and in  
22 your academic practice how are the patients, how do  
23 you take histories in the patients that you see?

24 A Well, thank you for asking that question.  
25 I've heard twice now on two separate occasions Dr.

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1 Mumper's description of academic physicians as  
2 physicians who don't take histories. And I would like  
3 to put that to rest.

4 I take, and I don't think I am any different  
5 than any of my colleagues, I take very extensive  
6 histories. I teach medical students, as we all do,  
7 that 90 percent of what you learn about a patient  
8 comes from the history. The history is an extremely  
9 important component of a patient's assessment. And if  
10 I, for example, have a complex patient that I am going  
11 to see I usually schedule two hours for the initial  
12 consultation, of which probably an hour-and-a-half of  
13 that is taking the history.

14 So I think it's important that we dissuade  
15 the listeners from any misconception that it is only  
16 doctors like Dr. Mumper who take histories. And I  
17 was, frankly, a little offended by that. I think  
18 academic physicians, physicians in private practice do  
19 definitely take histories.

20 Q And you also see autistic children in your  
21 practice; is that correct?

22 A I do.

23 Q And are the histories you take of autistic  
24 children any less thorough or any more thorough than a  
25 regular patient that you would see?

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1           A     No.  Actually they are very thorough because  
2           it's a slow history.  It's usually a history from the  
3           family.  Often the child is there.  It's often a  
4           difficult chore to take a history.  Often they come  
5           with an awful lot of questions about things that they  
6           have learned on the internet and various kinds of sort  
7           of alternative medicine treatments that we've been  
8           hearing about today that have been recommended, and  
9           they'd like advice about that.  So they tend to be  
10          very long discussions.

11          Q     Dr. Mumper also discussed today several  
12          aspects of chelation therapy.  And we heard Dr.  
13          Fombonne discuss the efficacy of that treatment.  But  
14          that aside, as a medical toxicologist do you see any  
15          reason for the chelation to remove mercury from either  
16          Jordan King or William Mead in these cases?

17          A     Absolutely not.  If we could bring up the  
18          slide that I think we showed earlier I just want to  
19          make one point on that slide.  Yeah.

20          Q     This is slide 45 from Dr. Brent's direct  
21          testimony.

22          A     If you will recall, the normal pattern of  
23          what we see for assessing mercury is that if we take a  
24          urine sample of mercury and we simply collect it on a  
25          patient there are validated reference ranges, that's

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1 an unprovoked urine, there's no chelator, there are  
2 validated reference ranges. And under normal people  
3 who are not mercury toxic will have a urine mercury  
4 excretion.

5 If on the other hand we take a normal  
6 person, any one of us here in this courtroom, and we  
7 add a chelator that urinary excretion will be  
8 increased, and often increased out of the normal  
9 reference range for unprovoked urine. So that's  
10 normally what you would expect to see. And, in fact,  
11 our gold standard test for assessing mercury toxicity  
12 is a urine mercury level. There is no test in  
13 medicine except on the cases of very shortly after an  
14 acute exposure where we might look at blood level  
15 there is no test in medicine that is more valid for  
16 assessing mercury toxicity than an unprovoked urine  
17 mercury concentration.

18 Below that you see the results of Jordan  
19 King and William Mead. And here we see that their  
20 unprovoked urine concentration is exactly in the  
21 normal range.

22 On the other hand, they have been chelated.  
23 And the justification for that chelation with regard  
24 to mercury comes from what you see in the righthand  
25 column where in both cases four out of five provoked

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1 urine samples have had increased urine mercury. Well,  
2 you're supposed to have increased urine mercury with  
3 provoked urine -- with provoked samples. Therefore,  
4 there is absolutely no indication based here or  
5 anything else I saw in the medical records that  
6 suggest that there is any mercury effect in these  
7 children and, therefore, there was absolutely no  
8 reason to chelate them for any mercury-related reason.

9 Q Thank you.

10 SPECIAL MASTER HASTINGS: Just for my  
11 benefit when I go back to read this, this is slide 46  
12 not 45; isn't that right?

13 MS. RENZI: I apologize, Special Master.

14 SPECIAL MASTER HASTINGS: Okay.

15 MS. RENZI: Slide 46.

16 BY MS. RENZI:

17 Q Dr. Mumper also testified today to seeing an  
18 increase of lead levels in children and that chelation  
19 may help with the adverse effects from lead. Is there  
20 any scientific or medical basis for that statement?

21 A It is true that chelation therapy is the  
22 appropriate therapy for lead toxicity. However, the  
23 records do not reflect any lead toxicity in the case  
24 of either of the two children at issue here, Mead or  
25 King. Neither of them had had an elevated live lead

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1 level. And a blood-lead level is the gold standard  
2 test for lead toxicity. Because contrary to testimony  
3 that was given earlier today, blood lead remains  
4 elevated and will be elevated for years in children  
5 that have lead toxicity. It equilibrates with tissues  
6 and if there is high tissue burden there's going to be  
7 high blood burden.

8 Q So you disagree with Dr. Mumper that the  
9 blood levels would only test for acute toxicity?

10 A That's absolutely wrong. So there was no  
11 indication, therefore, for treating either of these  
12 two children with a chelator for any lead effect.

13 Q Is there any other accepted test for  
14 measuring lead toxicity other than blood?

15 A Blood lead is the gold standard. And there  
16 are no other accepted tests in medicine now that  
17 routinely give blood levels, lead levels.

18 SPECIAL MASTER CAMPBELL-SMITH: Can I  
19 interrupt for just a moment?

20 MS. RENZI: Sure.

21 SPECIAL MASTER CAMPBELL-SMITH: I'm hearing  
22 a little something in your microphone, Dr. Brent. Can  
23 I encourage everybody to check to make sure you're  
24 turned off. Oh, and the distance from the microphone.

25 THE WITNESS: I understand.

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1 (Pause.)

2 SPECIAL MASTER VOWELL: Dr. Brent, while  
3 we're in a pause may I follow up on your comments  
4 about the lead levels in the --

5 THE WITNESS: Please.

6 SPECIAL MASTER VOWELL: Excuse me, the  
7 mercury levels post-chelation --

8 THE WITNESS: Right.

9 SPECIAL MASTER VOWELL: -- in both the Mead  
10 and the King boys.

11 THE WITNESS: Please.

12 SPECIAL MASTER VOWELL: Was there anything  
13 about the levels you observed in the medical records  
14 post-chelation that would cause you to think that  
15 these were extraordinarily high levels of excretion  
16 upon chelation?

17 THE WITNESS: No. You always expect the  
18 levels in the urine bumped post-chelation. It would  
19 happen to any one of us. There are no validated  
20 reference ranges for post-chelation, that's why  
21 they're not used in medical practice or there is no  
22 valid way of using them. And, in fact, if you look at  
23 these two children they've had mild increases in  
24 urine-lead excretion as I recall, but they were  
25 nothing different than what you would normally expect

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1 to see if you give a chelator to them.

2 SPECIAL MASTER VOWELL: Have you given  
3 chelators to a lot of children?

4 THE WITNESS: I have chelated a number of  
5 children.

6 SPECIAL MASTER VOWELL: So there's nothing  
7 here that would be out of the ordinary from your  
8 experience even in the absence of a standard reference  
9 range?

10 THE WITNESS: Well, I have to -- in truth we  
11 don't follow urine leads because the correct test is  
12 blood leads. So I haven't looked at many blood leads  
13 -- urine leads in children that I have chelated. So I  
14 can't speak to that from my experience. But I have  
15 seen, I have had a number of patients now come to me  
16 because of these Doctor's Data type of laboratories  
17 where which are based on urine, chelated urine, and  
18 they always have high leads in their chelated urine.  
19 And I tell them, well, let's just do the gold standard  
20 test, get a blood lead level, and so far 100 percent  
21 of the time they've been normal.

22 SPECIAL MASTER VOWELL: All right. And  
23 let's go back to mercury though.

24 THE WITNESS: Okay.

25 SPECIAL MASTER VOWELL: Are the post-

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1 chelation mercury levels in either of these two boys  
2 in excess of what you would see or in excess -- I take  
3 it there's no standard reference range post-chelation?

4 THE WITNESS: No standard reference range  
5 there. You do tend to see small increases, they've  
6 had some minor increases in their mercury excretion  
7 over the reference ranges for the non-provoked. It  
8 was not, certainly not very dramatic. And it was  
9 certainly well within the range of what you would  
10 expect to see.

11 For example, if you look at the studies that  
12 I've cited on where they were studying chelators and  
13 they would look at the effect of the chelator on urine  
14 mercury excretion, now that's a valid time to do a  
15 post-chelation mercury if you want to study the effect  
16 of the chelator. And if you look at the normal  
17 controls in those studies when they give them a  
18 chelator you do see some increase in the urine mercury  
19 excretion and it's a moderate increase and it's really  
20 not very different from what you'd see, what we saw in  
21 these children.

22 BY MS. RENZI:

23 Q Dr. Brent, I just want to clarify something.  
24 When you say you've chelated children you've chelated  
25 them for lead toxicity or mercury toxicity?

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1 A Actually both.

2 Q And under what circumstances did you chelate  
3 for mercury toxicity?

4 A I've had a number, but probably the most  
5 common and the most dramatic relates to the fact that  
6 I live in Colorado and in the Rocky Mountain area  
7 there are people that are still out panning for gold.  
8 And the way they do it is they collect gold ore, which  
9 is a mixture of gold and other things, and they take  
10 advantage of the fact that you can extract the gold  
11 from ore using liquid mercury. And so they chop up  
12 the ore, they grind up the ore, they mix it up with  
13 liquid mercury, they extract the gold and they get  
14 into the liquid mercury. They get rid of everything  
15 else. Now they have the gold separated, the only  
16 problem is it's in all this mercury. And what they  
17 will often do to get rid of the mercury is they will  
18 heat it. And they will often heat it in their house,  
19 in their kitchen for example.

20 When you volatilize mercury like that a  
21 tremendous amount will get into the air. And I've had  
22 now a number of families that have become profoundly  
23 mercury poisoned because somebody had heated up the  
24 mercury in an attempt to do this. Patients that were  
25 so sick that they've had to be on -- families that

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1 have had to be so sick that they've had to be on  
2 ventilators, they've had protracted stays in the ICU  
3 for severe inhalational mercury vapor poisoning.

4 Q Thank you. So when Dr. Mumper said that she  
5 saw mobilization of heavy metals by chelation and then  
6 assumed that the chelation was beneficial do you agree  
7 with that statement?

8 A No. That's exactly -- I think what you see  
9 is you give a chelator, you look in the urine and  
10 there is more than the non-chelated reference ranges  
11 for the levels in the urine, and it's what you would  
12 normally expect. It tells you nothing about  
13 mobilizing stores of heavy metals in the body.

14 Q Dr. Mumper also talked about supplements and  
15 those supplements to increase glutathione to treat  
16 mercury toxicity. Do you agree that that therapy is  
17 warranted in cases?

18 A Glutathione, no. Supplemental glutathione  
19 to treat mercury toxicity has no validity at all.

20 Q And why is that?

21 A Well, the reason for that is that we have  
22 very, very, very large amounts of glutathione in our  
23 bodies. WE have huge amounts of glutathione in our  
24 bodies. And glutathione is never limited in terms of  
25 being able to handle heavy metals. It's a defense

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1 that has been put into humans and animals and it works  
2 extremely well. And there is no way that some small  
3 additional amount of glutathione on top of the already  
4 very, very large stores we have, can make the  
5 slightest difference.

6 MS. RENZI: Thank you. I have no further  
7 questions.

8 THE WITNESS: Thank you.

9 SPECIAL MASTER CAMPBELL-SMITH: Thank you.  
10 Any questions from Petitioners' counsel?

11 MR. POWERS: Yes. Thank you, Special  
12 Masters.

13 CROSS-EXAMINATION

14 BY MR. POWERS:

15 Q Dr. Brent, my name is Tom Powers. I didn't  
16 have a chance to talk to you on direct or cross last  
17 time, that was Mr. Williams' privilege, but I have a  
18 couple of questions for you now.

19 A Sure. Please.

20 Q You were talking about Dr. Aschner's paper  
21 on glutamate uptake a little while ago; correct?

22 A Correct.

23 Q This is an experiment that was *in vitro* rat  
24 cells; correct?

25 A Correct.

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1 Q And there is some evidence, we've heard  
2 testimony, that human cells are often more sensitive  
3 than rat cells; is that correct?

4 A I don't know of any data about human cells  
5 being more sensitive to inhibition of glutamate uptake  
6 by mercury than rat cells.

7 Q Can you describe a human model that  
8 parallels what Dr. Aschner did with this rat model?

9 A If there was a very good human model that  
10 could be used then Dr. Aschner would probably be  
11 studying humans and not rat brains. The problem is  
12 that we don't, it's very hard to have cultured human  
13 neurons that are -- that have not been so transformed  
14 that they're highly artifactual. So unfortunately  
15 there's not a really good model for that. And that's  
16 why the rat models are typically used.

17 Q And actually this isn't a rat brain, these  
18 are isolated rat cells in a petri dish; correct?

19 A As I said, it was an *in vitro* culture. It's  
20 an *in vitro* experience, yes.

21 Q And this *in vitro* experiment featured  
22 astrocytes; correct?

23 A Correct.

24 Q And since it's an isolated culture it would  
25 not have microglia; correct?

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1 A Correct.

2 Q And you understand, of course, that Dr.  
3 Kinsbourne's hypothesis is based on the idea that  
4 microglial activation releases proinflammatory  
5 cytokines that harm astrocytes; correct?

6 A Correct.

7 Q So there's nothing about this petri dish  
8 that is absent microglia that is at all relevant to  
9 Dr. Kinsbourne's position that it's the reactive  
10 oxygen species in the proinflammatory cytokines  
11 released by microglia that cause the astrocyte damage;  
12 correct?

13 A Well, in fact this is the data that exists  
14 on mercurial effect on astrocyte glutamate uptake. I  
15 don't know of any data that is specific for this sort  
16 of complex scenario that you are describing in your  
17 question. However, clearly if you look at the data  
18 that was cited by Dr. Kinsbourne for showing that  
19 mercury inhibits astrocyte uptake of glutamate, this  
20 is the data. And that's the date, therefore, I was  
21 referring to.

22 Q Dr. Kinsbourne also was citing the Vargas  
23 and the Pardo papers and the evidence of inflammation  
24 that involved proinflammatory cytokines in the  
25 possible effect on astrocytes; correct?

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1           A     I don't recall him citing anything that  
2           suggested that, that showed that proinflammatory  
3           cytokines altered astrocyte glutamate uptake in  
4           response to mercury.

5           Q     And, in fact, when Mr. Williams cross-  
6           examined you on this issue you testified that you were  
7           not a neuroimmunologist and you didn't comment on  
8           those papers under cross and declined to comment on  
9           those papers under cross; correct?

10          A     Well, mostly correct. I don't think we were  
11          discussing neuroimmunology. This is not an  
12          immunological question.

13          Q     But the inflammatory response --

14          A     Right.

15          Q     -- is an immunological process?

16          A     Right.

17          Q     And Dr. Kinsbourne posits that it's  
18          initiated by microglia; correct? And there is nothing  
19          in the Aschner paper -- I mean it's impossible for it  
20          to address that issue because there were no microglia  
21          in the petri dish with the rat brain cells?

22          A     That's true. That's true.

23                   MR. POWERS: No further questions.

24                   SPECIAL MASTER CAMPBELL-SMITH: Any further  
25                   questions from Respondent's counsel?

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1 MS. RENZI: No, thank you.

2 SPECIAL MASTER CAMPBELL-SMITH: Any  
3 questions from my colleagues?

4 (No response.)

5 SPECIAL MASTER CAMPBELL-SMITH: Thank you,  
6 Dr. Brent.

7 THE WITNESS: Thank you, Special Master.  
8 (Witness excused.)

9 SPECIAL MASTER CAMPBELL-SMITH: Any further  
10 witnesses to be called by Respondent's counsel?

11 MR. MATANOSKI: No, ma'am.

12 SPECIAL MASTER CAMPBELL-SMITH: We are  
13 roughly at 4:00 o'clock. I think this might be a good  
14 moment to take a break and let counsel gather your  
15 thoughts for the brief closing arguments that, or  
16 closing remarks that counsel had indicated that they  
17 were planning to make. So how brief would you like  
18 this recess to be to prepare your brief remarks?

19 MR. POWERS: I'm happy with 10 minutes,  
20 Special Master.

21 MR. MATANOSKI: That would be fine for me as  
22 well.

23 MR. POWERS: Maybe just 10 after the hour.

24 SPECIAL MASTER CAMPBELL-SMITH: Perfect. We  
25 are in a brief recess.

1 (Whereupon, a short recess was taken.)

2 SPECIAL MASTER CAMPBELL-SMITH: We are back  
3 on the record for brief remarks.

4 MR. POWERS: And, Special Masters, I  
5 appreciate a description of this as brief and just  
6 remarks. And I just want to acknowledge that,  
7 particularly for people who might be listening either  
8 live or will download this, what we are going to do  
9 here, it's certainly not what I plan to do, is a  
10 summary of the evidence and argue with the evidence  
11 really not at all, because that's something that in  
12 this program, as the counsel and the Special Masters  
13 know, is something that happens in the months after  
14 the evidence is closed in theses cases and happens  
15 largely on paper through motions and pleadings. But  
16 we do want to take advantage of the opportunity that  
17 you have provided to make some comments about the  
18 proceedings here over the last three weeks and the  
19 proceedings in the Omnibus Autism Proceeding in  
20 general.

21 One issue that I want to talk about is  
22 something that we've heard about and this idea that  
23 the Petitioners somehow are wanting to spring  
24 surprises, whether it's on the Court or on  
25 Respondent's counsel. And I just want to make it

1 clear, particularly to the Special Masters, that that  
2 absolutely is not the Petitioners' intent. We are  
3 responding to a dynamic scientific environment. And  
4 we do everything we can to stay on top of the  
5 literature. We monitor everything we can. And when  
6 we find something new we want to bring it your  
7 attention to inform your decision in these important  
8 cases.

9 We are working hard to do that. And I can  
10 definitely assure you that if we found something  
11 helpful we would want to talk about it early and talk  
12 about it often. So there is no intent here to slip in  
13 a surprise or hide the ball. We want our best and our  
14 strongest case in front of you and in front of the  
15 Respondent as early and often as we can.

16 But as I said, we are in a dynamic  
17 scientific environment. There is new research going  
18 on all the time, some of this during the hearing.  
19 There were abstracts presented on some of these  
20 relevant issues at international conferences. There  
21 were peer-reviewed papers that appeared in journals,  
22 some of them were published during this proceeding,  
23 some of them only became available in our language  
24 during this proceeding.

25 It's also important to understand, I

1 believe, that there's an interesting dynamic at work  
2 in the Vaccine Program that one does not encounter in  
3 traditional civil litigation, and I believe it's  
4 intention, and Congress set it up this way. It's  
5 important to remember that the Respondent here is the  
6 United States Department of Health and Human Services  
7 and its related agencies. They have a charge and a  
8 public mission and a public obligation and a public  
9 duty to stay abreast of the science, to follow the  
10 science and, in a sense, to not be surprised by the  
11 science. And it's important that in these proceedings  
12 the litigation goals of prevailing not be confused  
13 with the client's overarching public policy goal of  
14 staying abreast of the science, interpreting the  
15 science, and getting the word about the science out to  
16 folks, whether it's their attorneys that are here in  
17 these proceedings to the families of the children  
18 here, to the Special Masters, and to the scientific  
19 community at large.

20 I believe that one of the reasons that  
21 discovery is not available as a matter of right in  
22 this program is in a sense to help address the tension  
23 that you see in civil litigation about the interests  
24 of the parties. In civil litigation each party in the  
25 adversarial system has only its own self-interest at

1 mind, that is, the only interest they have in the  
2 adversary system is to prevail in that litigation and  
3 to win in that litigation. But here the Respondent,  
4 the U.S. Department of Health and Human Services, has  
5 that larger obligation to be doing the scientific  
6 research, to fund research, to make data and research  
7 available to the public.

8 So by taking away the contentious  
9 adversarial rules of discovery it seems that it helps  
10 alleviate that tension and doesn't create a conflict  
11 between the litigation defense goals and the public  
12 policy goals of not being surprised by the science.

13 We've heard testimony that a lot of the work  
14 that the Petitioners have introduced in this case is  
15 work that is in fact funded by the NIH, by the CDC,  
16 and by other entities involved with the Respondent,  
17 with HHS. To the extent that the Respondent is  
18 involved in the science, whether it's doing the  
19 science itself, funding the science and monitoring the  
20 science, they ought not to be claiming complete  
21 surprise when new science does come out. Again, we  
22 cannot confuse the litigation goals with the public  
23 policy goal and the institutional goal that HHS has.  
24 And as I said, I believe that is one of the reasons  
25 that Congress wanted this to be a non-adversarial

1 system and to not have those rules of discovery that  
2 for those experienced in civil litigation really turns  
3 it into a fight over sometimes every scrap of paper  
4 that you are trying to pull from the other side.

5 So the program should be less adversarial in  
6 that way. And I think it's important to remember  
7 that. It's also to remember that the program is  
8 designed to be less adversarial in order to provide an  
9 environment for families who believe that they have  
10 legitimate claims to appear and present their case.  
11 And that also includes having experts who are willing  
12 to come in and testify for them. The experts in this  
13 process are obviously critically important because all  
14 of the issues that you all have to decide are often  
15 very complicated issues of fact that require technical  
16 explanation, interpretation, and presentation. And I  
17 just think it's a shame that in these Omnibus Autism  
18 Proceedings we have seen from the Respondent a  
19 regrettable inclination to launch attacks, often  
20 unsubstantiated smear attacks, on some of the  
21 witnesses involved in these cases. And we saw it with  
22 Dr. Kinsbourne in this proceeding.

23 Again, if the Federal Rules of Civil  
24 Procedure were at play none of the issues that  
25 Respondent's counsel attempted to impeach the

1 credibility of Dr. Kinsbourne on would have been  
2 allowed in. This is a 31-one-year-old employment  
3 dispute that was resolved in his favor but they  
4 brought it in. And I argue and Petitioners believe  
5 that the lack of rules of the Federal Rules of Civil  
6 Procedure applying here explicitly was done by  
7 Congress in order to make it less adversarial and to  
8 remove some of those adversarial qualities that one  
9 sees in the civil litigation system. And that's a  
10 system where you commonly do see this type of attack  
11 constrained by the rules. But here the absence of the  
12 rules shouldn't allow people to engage in conduct that  
13 would be barred by the rules in a civil proceeding,  
14 and it's regrettable.

15 Dr. Kinsbourne, obviously, was perfectly  
16 capable of defending himself, and he did, and he's  
17 made that record. But it is just regrettable that in  
18 every one of these hearings, whether it's Dr.  
19 Bradstreet being accused of being an exorcist to Dr.  
20 Kinsbourne being attacked for the issues he was  
21 attacked on here is regrettable and we ought to be  
22 able to avoid that in this congressionally-mandated  
23 non-adversarial study.

24 One of the last things I wanted to conclude  
25 on is addressing a thematic argument that I have heard

1 and I think all the Petitioners have heard from  
2 Respondent's experts, and that is the idea that the  
3 Petitioners' expert witnesses are somehow so fixed on  
4 a conclusion that they leapt to that they are willing  
5 to ignore contrary evidence, that they are staring  
6 through Tycho Brahe's telescope insisting that the  
7 Earth is the center of the universe. I think in the  
8 testimony that you've heard in this proceeding that  
9 absolutely is not the case. And I just want to use  
10 the example of Dr. Mumper.

11 Dr. Mumper is a clinician not a bench  
12 scientists, not somebody that does original research,  
13 but she is a clinician who has responded to the needs  
14 of a significant patient population who weren't being  
15 addressed by other doctors, including Dr. Rust. And  
16 so Dr. Mumper, even if the Respondent's experts  
17 disagree with her conclusions, what you heard from Dr.  
18 Mumper is a doctor who is doing her absolute best to  
19 follow good science, to keep on top of the science.  
20 Here is a pediatrician in Lynchburg, Virginia that is  
21 spending her resources to build bibliographies of  
22 science, to get that information out to other doctors,  
23 to validate her work as scientifically as she can, to  
24 bring in the resources to increase the scientific  
25 rigor and the scientific integrity of the work that

1 she's doing.

2 She is doing that while on the other hand  
3 Dr. Rust is so fixed in his telescope, the Tycho Brahe  
4 telescope, or the *idee fixe* that what you have here,  
5 he testified for at least an hour on Rett syndrome.  
6 And it seemed to be his argument that Rett because  
7 it's congenital and genetic is a model for autism  
8 because if Rett's is genetic and autism shares some of  
9 the symptoms of Rett's then autism itself must be  
10 genetic.

11 That line of argument is a faulty syllogism.  
12 It's sort of another example of the classic false  
13 syllogism that Aristotle is a man, all man are mortal,  
14 therefore all men are Aristotle. It's a flawed logic.  
15 And it just represents how fixed he is on the idea  
16 that this is an inevitable, at inception,  
17 predetermined outcome that he is not willing to  
18 entertain apparently the idea that environmental  
19 factors might be at play, that care and treatment  
20 might alleviate the symptoms, that some care and  
21 treatment somewhere down the road in an investigation  
22 into etiologies that aren't presumed to be genetic are  
23 worthwhile. His mind is closed to that.

24 And those are just two very contrasting and  
25 telling examples of the type of, if we're going to be

1 describing expert advocacy in these cases as the  
2 Respondent's experts have attempted to do, that is an  
3 example of the Respondent's experts who are so focused  
4 on what they think the outcome is that they are  
5 willing to spend 114 pages in a PowerPoint really just  
6 arguing, as I said, the false syllogism that all  
7 autism is like Rett's, and therefore all autism is  
8 congenital. That is not supported by the science.

9 So these are just some observations about  
10 this proceeding as we move forth. Again, this ought  
11 to be a science-based inquiry. This ought to be a  
12 non-adversarial setting. This ought to be the type of  
13 setting where families and their experts can come and  
14 air their meritorious claims. And whether one  
15 disagrees with the conclusion that any particular  
16 witness reaches, the idea that at every single one of  
17 these test cases there is going to be some one of the  
18 Petitioners' experts who is going to be targeted the  
19 way that some of these experts have been in earlier  
20 proceedings is something that we should avoid and  
21 focus on the science, be willing to consider the  
22 science that comes in, understand that the science is  
23 changing, understand that there is a convergence of  
24 science over time, and understand that when we do  
25 close the evidence in these cases there probably will

1 be more information out there in the scientific  
2 literature. And the science will need to speak for  
3 itself at some point. And as we see the science  
4 converge on some of these key issues the Petitioners  
5 will do everything that we can to bring that  
6 information to the Special Masters, to share it with  
7 the Respondent, but ultimately with the idea that  
8 litigation strategy in this program is really not what  
9 should be driving the consideration of the science but  
10 ultimately, again considering the unique position of  
11 the Respondent as a party here, reflecting a  
12 responsible fulfillment of the mission to keep up to  
13 date with the science, protect public health, consider  
14 the science and apply it in a way that's going to  
15 provide the best information ultimately to the three  
16 of you deciding the general issues and the specific  
17 issues in all of these cases.

18 Thank you.

19 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

20 Mr. Matanoski?

21 MR. MATANOSKI: Thank you, ma'am.

22 In putting together my closing remarks,  
23 though the time that we have is brief, I feel I would  
24 be tremendously an error of my part to not acknowledge  
25 the families that were involved here, the King an the

1 Mead family. Probably the most poignant moments  
2 during this trial was hearing their testimony,  
3 testimony of MyLinda King and George Mead discussing  
4 William and Jordan. We thank them for their  
5 participation. Certainly our hearts go out to them  
6 and to all of the families that have autistic  
7 children. We may be litigating one side of this issue  
8 but we certainly have tremendous respect and  
9 admiration for all of them.

10 You have a threshold matter before you  
11 that's a scientific matter, however, which you must  
12 address. And obviously a scientific question  
13 necessarily turns on scientific evidence. And there  
14 are certain legal standards that must be applied in  
15 this courtroom and every courtroom to how you handle  
16 scientific evidence. What, indeed, can even be  
17 considered reliable scientific evidence.

18 The Supreme Court has spoken. It said that  
19 it is evidence that must be tested, it's evidence that  
20 should be subject to publication and peer review, it's  
21 evidence that has general acceptance in the scientific  
22 community. On the PSC side of the ledger of the  
23 evidence you have not heard that yet, you've heard  
24 speculation pure and simple.

25 What you've hard in terms of comments from

1 Mr. Powers this morning suggests that that evidence as  
2 far as the Petitioners are concerned or the PSC is  
3 concerned is still not available. He talks about the  
4 dynamics of science, ongoing studies, which in some  
5 way may imply a lack of evidence, scientific evidence  
6 that is available to the PSC at this point to prevail.

7 Now, the PSC's case started with a curious  
8 approach. Rather than putting on evidence in support  
9 of their claim they put on evidence that was to, or  
10 put on testimony that was designed to undermine  
11 evidence against their claim. That was the testimony  
12 of Dr. Greenland. But Dr. Greenland's testimony and  
13 his whole postulate depended on a supposition. The  
14 supposition was that the Petitioners would prove to  
15 you a case that their mechanism applied to clearly  
16 regressive cases only. Now, you've heard from Dr.  
17 Deth about his hypothesis and he said it did not apply  
18 only to clearly regressive cases. You heard this  
19 morning from Dr. Kinsbourne who said that he hasn't  
20 even looked at whether his hypothesis would have any  
21 application on non-regressive cases so he can't even  
22 address whether his hypothesis is only limited to  
23 clearly regressive cases.

24 All of the abundant epidemiological evidence  
25 that has addressed the precise issue in front of you,

1 that is whether thimerosal-containing vaccines can  
2 cause autism or are associated with autism is back on  
3 the table. It never was off. Dr. Greenland's  
4 supposition is in error.

5 If you follow the mechanisms proposed by the  
6 PSC here to their logical conclusion, they fail to  
7 show that thimerosal-containing vaccines are the  
8 cause. They propose that inorganic mercury is the  
9 causative agent. Inorganic mercury is not specific to  
10 childhood vaccines. It's in what we eat, it's in the  
11 air we breathe, it may be if we have poor dental  
12 health may be in the fillings in our mouth. They have  
13 failed to specify how much inorganic mercury is  
14 necessary to cause autism. Their experts consistently  
15 refused to say. In fact, when they did say they  
16 essentially said any amount. They have pushed the  
17 threshold down so that any exposure to inorganic  
18 mercury could be a potential cause of autism.

19 They have described a causal mechanism or  
20 mechanisms that are so general they apply to virtually  
21 every disease and to every case of autism. Oxidative  
22 stress is seen in conjunction with almost every  
23 disease. You even see it after trotting or jogging,  
24 you even get it after you bang your thumb nailing it,  
25 hammering in a nail. Neuroinflammation is seen in a

1 variety of neurological illnesses, including  
2 Alzheimer's and Parkinson's disease, for example. And  
3 in the Vargas study every single autistic patient in  
4 that study had neuroinflammation, regressive, non-  
5 regressive, young and old alike. These are non-  
6 specific causal mechanisms that are proposed to you.

7 In the end, you could just as easily  
8 conclude that a tuna sandwich or a dental filling  
9 could cause autism as a childhood vaccine. And to  
10 flip it around, you can just as easily consider that  
11 an 80-year-old man who received a flu vaccine would  
12 get Alzheimer's from it.

13 Mr. Powers commented about what I describe I  
14 guess as -- or his description of a smear campaign or  
15 heavy-handed treatment of Petitioners' experts. You  
16 take the witnesses as they come. Now, perhaps there  
17 was an explanation, and you've heard it for the events  
18 that transpired with Dr. Kinsbourne's departure from  
19 the University of Toronto, but again, you take the  
20 witnesses as they come. When Dr. Deth took the  
21 witness stand and said that he's willing to come  
22 before you and say that his hypothesis, you should  
23 rely on that to make a finding of this import even  
24 though he's not willing to go to the scientific  
25 community and say that it's acceptable without further

1 testing, I think that bears consideration.

2 Dr. Kinsbourne when he sat in the witness  
3 chair he put his credibility on the line. If he's  
4 coming before you saying, rely on me, believe me,  
5 trust me as an impartial scientist, because that's how  
6 he's coming to testify to you, you deserve to know  
7 whether he gets that kind of trust. You know he's  
8 known to you, you've seen him appear many times. If  
9 you go back and look at the cases that are currently  
10 active in front of the Special Master's Office you  
11 will find that he's maintained or offered an expert  
12 opinion saying vaccines have done harm in over 30  
13 cases. In the past year he's offered one article in a  
14 medical journal. I think that tells you whether he's  
15 coming to you as a witness who spends his time in the  
16 courtroom or as an impartial scientific expert witness  
17 who is adding some value to what your deliberations  
18 are from a point of view of reliable science.

19 Now good science and reliable science comes  
20 from testing, publication, critical review,  
21 validation, verification of results. It's performed  
22 by those who work in the field, apply scientific  
23 method to their research. The Supreme Court tells us  
24 there can't be untested hypothesis, as Dr. Deth has  
25 essentially described his causal mechanism. And good

1 science won't be first revealed in the courtroom, as  
2 Dr. Kinsbourne's hypothesis is. But it's going to see  
3 the light of day through critical discussions of  
4 research among the scientists themselves. It's not  
5 reliable science, indeed it's not any kind of science  
6 to sit at your computer, take your last litigation-  
7 driven report, run find and replace. Find measles  
8 vaccine and replace with thimerosal-containing  
9 vaccine. A litigation-driven contrivance such as that  
10 has no place in this courtroom; the Supreme Court has  
11 mandated that.

12 Now, when the trial began Mr. Powers  
13 described thimerosal-containing vaccines as a relic of  
14 history. Perhaps that was a reference to allowing  
15 some leeway in what your evidentiary standards would  
16 be to provide some grading on the curve of the science  
17 you'd accept. In fact, they have done everything to  
18 make this anything but a relic of history. The day  
19 that they said that they held a press conference to  
20 discuss the case. Their experts are here telling you  
21 that trace amounts of mercury that are in vaccines,  
22 the flu vaccine, for example, that's still  
23 administered could be enough to cause autism.

24 This, whether we like it or not, this issue  
25 has great importance, the issue before you had great

1 attention drawn to it. Just last week Time Magazine  
2 had vaccines and the safety of vaccines as their cover  
3 issue. Many eyes are going to be turned to this Court  
4 to see how you handle the scientific evidence before  
5 you. What do you make of that evidence? And it's not  
6 just from the parents that are front of you with their  
7 claims, it's from parents who haven't brought claims  
8 who have autistic children and who are wondering if by  
9 getting them vaccinated they are somehow responsible  
10 for that condition. It's from scientists who work in  
11 these relevant fields, it's from those who treat  
12 autism, and it's going to be reviewed by parents who  
13 are wondering whether they should get their children  
14 vaccinated or not.

15 Now, I'm going to be blunt at this very late  
16 hour having brief remarks. Are you going to decide  
17 that question on the say-so of Dr. Deth and Dr.  
18 Kinsbourne? Or are you going to decide that question  
19 on the evidence given to you by witnesses like Dr.  
20 Catherine Lord, Dr. Eric Fombonne, and Professor Sir  
21 Michael Rutter? Are you going to look at and consider  
22 the fact that every reputable medical, independent  
23 medical organization that has considered this issue,  
24 the Institute of Medicine, the American Academy of  
25 Pediatrics, the European Medicine Association, the

1 World Health Organization have all come to the  
2 conclusion that thimerosal-containing vaccines do not  
3 cause autism?

4 Are you going to also consider that every  
5 Court that has had to consider this claim before it,  
6 or you have considered it in fact, has found that the  
7 claim is so lacking in scientific merit that it should  
8 not be even presented to a jury?

9 Reliable scientific evidence at this point  
10 is all on one side of the ledger: vaccines don't cause  
11 autism.

12 Thank you. I have no further remarks at  
13 this time.

14 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

15 At this time we have reached the conclusion  
16 of this portion of the evidentiary hearing in the  
17 Omnibus Autism Proceeding. And on behalf of my  
18 colleagues I am going to make a few brief comments  
19 this afternoon.

20 First, we again thank the members of the  
21 King and the Mead families who came to Washington and  
22 were with us for part of this hearing. We thank them  
23 as well for generously agreeing to have their sons'  
24 cases designated as test cases in the Omnibus Autism  
25 Proceeding.

1           We also wish to thank the counsel for both  
2 sides who have presented their evidence so ably during  
3 this hearing. We know that they have worked  
4 enormously hard in preparing and in conducting this  
5 hearing. And we appreciate that hard work.

6           We also thank the expert witnesses who have  
7 testified before us.

8           We thank the United States Court of Claims  
9 for the Federal Circuit who have allowed us to use  
10 their courtroom. We thank all of the wonderful  
11 employees of both of the courts housed in this  
12 building who assisted so well in preparing for and  
13 conducting this hearing.

14           Next we want to acknowledge once more  
15 certain other people who are also very important to  
16 this proceeding, that is the families of all the other  
17 5,000 Vaccine Act claimants who have been diagnosed  
18 with autism or a similar condition. Some members of  
19 those families have been listening in by means of our  
20 teleconferencing system. Others have followed this  
21 hearing by downloading the audio from the internet.  
22 To all such family members, as to the King and the  
23 Mead families, we three Special Masters pledge to you  
24 again that we will consider very carefully the  
25 evidence put before us at this hearing and give that

1 evidence our very complete and thorough study. We  
2 realize the great importance of the task assigned to  
3 us in deciding these cases. And we will give our  
4 greatest effort in carrying out that heavy  
5 responsibility.

6 Finally, now that this hearing is finished  
7 in this respect some of you may want to know what will  
8 happen in these test cases. The answer is that,  
9 first, in July we will hear from two more expert  
10 witnesses for Respondent who could not be here this  
11 month. At that same time we will hear any rebuttal to  
12 those two witnesses that the Petitioners wish to  
13 present.

14 We will also hear some case-specific  
15 testimony in a third yet-to-be-identified test case  
16 related to the same theory, which case will be decided  
17 by Special Master Vowell.

18 In addition, after the July hearing the  
19 parties will file written briefs summarizing the  
20 testimony in this hearing. That process will likely  
21 take several months. Then once the last of those  
22 briefs are filed I will issue a written ruling in the  
23 William Mead case. Special Master Hastings will issue  
24 a written ruling in the Jordan King case. And Special  
25 Master Vowell will issue a written ruling in the third

1 to-be-identified case.

2 Finally, for updates concerning the progress  
3 of all three cases and concerning the Omnibus Autism  
4 Proceeding in general please do keep checking the  
5 autism proceeding page on the Court's internet  
6 website.

7 With that, I thank everyone involved in this  
8 hearing. I wish you safe travels to your point of  
9 return. We are now adjourned.

10 (Whereupon, at 4:40 p.m., the hearing in the  
11 above-entitled matter was concluded.)

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REPORTER'S CERTIFICATE

DOCKET NO.: 03-584V, 03-215V  
CASE TITLE: In Re: Claims for Vaccine Injuries  
HEARING DATE: May 30, 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 30, 2008

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