

UNITED STATES
COURT OF FEDERAL CLAIMS

IN RE: CLAIMS FOR VACCINE)	
INJURIES RESULTING IN)	
AUTISM SPECTRUM DISORDER, OR)	
A SIMILAR NEURODEVELOPMENTAL)	
DISORDER,)	
)	
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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. MEAN,)	
A MINOR,)	
Petitioners,)	
v.)	Docket No.: 03-215V
SECRETARY OF HEALTH AND)	
HUMAN SERVICES,)	
)	
Respondent.)	

REVISED AND CORRECTED COPY

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Place: Washington, D.C.
Date: May 14, 2008

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IN THE UNITED STATES COURT OF FEDERAL CLAIMS

IN RE: CLAIMS FOR VACCINE)
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Respondent.)

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

Petitioners,)

v.)

Docket No.: 03-215V

SECRETARY OF HEALTH AND)
HUMAN SERVICES,)

Respondent.)

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

Wednesday,
May 14, 2008

The parties met, pursuant to notice of the
Court, at 9:00 a.m.

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

APPEARANCES:

For the Petitioners:

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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>					
Marcel Kinsbourne	769	845	926	944	--
George Mead	950	981	999	--	--

E X H I B I T S

PETITIONERS'

EXHIBITS:

IDENTIFIED

RECEIVED

DESCRIPTION

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Menkes Child Neurology
textbook

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P R O C E E D I N G S

(9:00 a.m.)

SPECIAL MASTER CAMPBELL-SMITH: This is Special Master Campbell-Smith. We are back on the record in this second test case proceeding of the OAP. I understand that there is a brief matter that we need to take up preliminarily. We can do it on the record if counsel are prepared to do that.

MR. POWERS: Yes. Thank you, Special Master. I guess I should stand up here for the intercom. My name is Tom Powers. I'm one of the attorneys for the Petitioners in these two cases and the Petitioners' Steering Committee.

We became aware at 9:30 last night that there was an electronic filing of a new exhibit from Respondent into both of the individual King and Mead claims that are subject to this proceeding.

My understanding from conferring with Respondent's counsel is that this is material that is not anticipated to be introduced into evidence today, there will be no effort to use it from Respondent's perspective in cross-examination, they will not be bringing in any witnesses today to discuss the material. So for today we don't believe that there's any issue that's created by the late-night filing of

1 this new exhibit. This is Exhibit LL, I believe is
2 the designation for the record.

3 We do, however, at some point if Exhibit LL
4 is going to be discussed at trial, brought into
5 evidence, we simply ask that the person who is the
6 apparent author of this material appear for cross-
7 examination so that we have an opportunity to explore
8 the issues that are raised in the correspondence that
9 is enclosed with Exhibit LL.

10 That really is the only issue that we have
11 the Court is to alert the Court that at the point at
12 which Respondent may want to use this, if they're
13 going to be using it, we would want an opportunity to
14 cross-examine the author.

15 SPECIAL MASTER CAMPBELL-SMITH: Thank you.
16 Mr. Matanoski?

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

18 MR. POWERS: And Mr. Williams just reminded
19 me, cross, and to use it in rebuttal, if they're going
20 to be using it in their case.

21 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

22 MR. MATANOSKI: Thank you, ma'am. I have a
23 comment on that. I think it's ironic that Mr. Powers
24 should take this opportunity to talk publicly on the
25 record about a late filing when so far in this case,

1 as discussed off the record, there have been numerous
2 instances of late filing, late opportunities for the
3 Respondent to respond to matters brought up by
4 Petitioners.

5 In fact, as the Court is well aware, the
6 deadline for filing Petitioners' expert reports was in
7 November of last year, and yet, in April of this year
8 we received Dr. Kinsbourne's report for the first time
9 well after that deadline, indeed, well after the
10 Court's set deadline of March for rebuttal
11 information, which of course this expert report did
12 not include.

13 Just last week the Petitioners' Steering
14 Committee filed with the Court 208 new medical
15 articles, many of which were not on any topics
16 previously discussed in this case. It again is ironic
17 that these matters have been discussed. Oh, and
18 yesterday, obviously, and the day before the witnesses
19 were talking about matters that have not been
20 discussed in their expert reports.

21 Indeed, yesterday the witness was discussing
22 studies that he had been conducting and before which
23 he had information back in the summer of 2007 but we
24 were seeing for the first time yesterday.

25 So I just would observe that it's ironic

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1 that after all these things have been transpiring with
2 Petitioners' Steering Committee, and which we've been
3 discussing off the record as to what kinds of
4 different remedies or procedural changes might be
5 occasioned by these surprises, that they should choose
6 to speak on the record today about a one and a half
7 page letter that they received from the author of a
8 study that they primarily seem to be relying on at
9 this time. Thank you.

10 SPECIAL MASTER CAMPBELL-SMITH: Thank you,
11 counsel. And I can assure counsel that we are aware
12 of the issues that both counsel have drawn to our
13 attention.

14 At the appropriate time, as we have
15 addressed during bench conferences with counsel that
16 should counsel wish to make more formal motions
17 addressing these matters, we would be happy to
18 consider more formal objections or concerns that
19 counsel have at that time.

20 Are there any further preliminary matters that we
21 need to address this morning?

22 MR. POWERS: Not from the Petitioners.

23 MR. MATANOSKI: Nor Respondent.

24 SPECIAL MASTER CAMPBELL-SMITH: Thank you.
25 Petitioners' counsel, are you ready to call your next

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1 witness?

2 MR. POWERS: Yes, we are, Special Masters.
3 The Petitioners call Dr. Marcel Kinsbourne.

4 SPECIAL MASTER CAMPBELL-SMITH: Thank you.
5 Dr. Kinsbourne? Dr. Kinsbourne, please be seated.
6 I'll let you get comfortable. If you could, stay to
7 the rightmost of your seating area. It assists in
8 helping all of us to see you.

9 MR. KINSBOURNE: Okay. It's about as far as
10 I dare go.

11 SPECIAL MASTER CAMPBELL-SMITH: Okay. Thank
12 you. Would you raise your right hand, please.

13 MR. KINSBOURNE: Yes, ma'am.

14 Whereupon,

15 MARCEL KINSBOURNE

16 having been duly sworn, was called as a
17 witness and was examined and testified as follows:

18 SPECIAL MASTER CAMPBELL-SMITH: Thank you.
19 To proceed, counsel.

20 MR. POWERS: Thank you, Special Masters.

21 DIRECT EXAMINATION

22 BY MR. POWERS:

23 Q Good morning, Dr. Kinsbourne.

24 A Good morning.

25 Q Are you all situated there and comfortable?

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1 A I believe so.

2 Q Okay. And can you hear me okay across the
3 room?

4 A Yes.

5 Q Excellent. I can hear you. I know that as
6 we go on if this goes on for a bit I'll ask you to
7 speak up so we can make a clear record.

8 A Certainly.

9 Q Great. I would like to start off just with
10 a brief discussion of your qualifications. They're
11 certainly described and summarized in your expert
12 report and there's I believe a CV, but if you could
13 just describe in summary what it is about your
14 experience, training, skills and background that
15 qualifies you to speak about the issues today?

16 A Yes, sir. Well, as you know, I am a
17 pediatric neurologist and it is customary for people
18 in my specialty and in many others to choose a
19 specific focus within that specialty for their
20 particular interest, both clinical and research. From
21 the beginning, even during my training, I chose as
22 focus mental development disorders in children.

23 In fact, while I was still a resident I
24 published two articles on dyslexia. When I came to
25 the U.S. to my associate professorship at Duke, I was

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1 not only chief of the Division of Pediatric Neurology,
2 but also, head of the Developmental Evaluation Clinic
3 where we saw many children with diverse problems of
4 development, including of course many children with
5 various forms of autism and autistic spectrum
6 disorder.

7 I formed a loose friendship with the folks
8 over at UNC in the TEACCH program, T-E-A-C-C-H, which
9 is a well-known program for autistic care and
10 research, and I did co-author one article with them on
11 a topic about it. When I came to Duke I became very
12 interested in attention deficit disorder and saw many
13 people and started an active research program which
14 went on for many years and has not yet concluded.

15 In that practice, again, one saw many
16 children who were perhaps ADHD, perhaps autistic at a
17 high-functioning level, so the importance of these
18 issues remained a focus for me.

19 SPECIAL MASTER CAMPBELL-SMITH: Just one
20 moment, Dr. Kinsbourne.

21 THE WITNESS: Yes, ma'am.

22 SPECIAL MASTER CAMPBELL-SMITH: Intercall
23 operator, are we having any difficulty?

24 (No response.)

25 SPECIAL MASTER CAMPBELL-SMITH: Is the

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1 intercall operator there?

2 (No response.)

3 SPECIAL MASTER CAMPBELL-SMITH: It appears
4 that we are having difficulty. Let's go off the
5 record.

6 (Whereupon, a short recess was taken.)

7 SPECIAL MASTER CAMPBELL-SMITH: Petitioners'
8 counsel to proceed.

9 MR. POWERS: Thank you, Special Master.

10 BY MR. POWERS:

11 Q So, Dr. Kinsbourne, we'll have an exercise
12 in refocusing here after the technical interruption.
13 You were describing your qualifications, and I believe
14 what you were describing was your work at Duke when
15 you were living in North Carolina. Is that where your
16 story was interrupted?

17 A Right. I was actually about to move to
18 Toronto.

19 Q To where?

20 A I was about to move to Toronto when the
21 technical problem arose.

22 Q All right. So let's go ahead, and if you
23 could pick up your testimony at that point?

24 A Yes, sir. So at Duke, as I say, I had
25 particular interest in and experience into mental

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1 disorders, including of course some autistic spectrum
2 disorders.

3 When I moved to Toronto to be a professor of
4 child neurology at University I ran a learning clinic,
5 which was a very busy one in which, again, one saw the
6 same kind of children, including autistic children,
7 and published a lot of article on various issues of
8 developmental disabilities. I then came back to this
9 country as chief of the Division of Behavioral
10 Neurology at the Eunice Kennedy Shriver Center.

11 Now my research and attached clinical work
12 was entirely in developmental disabilities. I saw
13 hundreds, perhaps thousands, of children in the 10, 11
14 years I was there. Also, because the Shriver Center
15 is on the grounds of the Fernald State School for
16 Mental Retardation and Developmental Disabilities, I
17 consulted on those grounds with people and also in the
18 Developmental Disability Clinic at that center.

19 Then during the 1980s I published two
20 articles on autism. One of them was a presentation
21 that I gave to an NIH panel on the topic which then
22 was fashioned into an article. Now, as of the early
23 1970s, I was contributing my chapter to the *Menkes*
24 *Textbook of Child Neurology*, and of course kept track
25 of developments in the various developmental

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1 disabilities for that purpose, and I have continued to
2 do this.

3 In fact, about 10 months from now the
4 chapters of the next edition are due, so I'm revving
5 up to get up to speed for that 8th edition and my
6 chapter in that volume.

7 Q So, Dr. Kinsbourne, when you say revving up,
8 are you referring to going back and rereading all the
9 current literature so that that 8th edition is truly
10 up to date and reflects state of knowledge when it
11 comes out?

12 A Yes. The challenge is to try to find those
13 articles which are both most novel and yet accessible
14 to the general child neurologist because it's
15 impossible to encompass these enormous fields now in a
16 chapter in one book. So it's really the task of the
17 author to give key references which will then lead the
18 interested reader to further information.

19 So it's a matter of selection as much as of
20 simply looking at everything. At any rate, these are
21 important inducements to keep my nose to the
22 grindstone of being up to date in autism and other
23 developmental areas. I also wrote two articles on a
24 disorder that I called overfocusing, which with
25 further study seems to us to be, as it were, at the

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1 top of the autistic spectrum bordering on the normal
2 range.

3 In a recently appeared article with
4 colleagues at University of Connecticut we were able
5 to show that on a questionnaire study there is
6 actually an overfocusing factor which is present in
7 otherwise normally functioning children and yet is
8 present all the way down the parameter of severity in
9 autistic individuals with severe disabilities.

10 So in the well-functioning end of the
11 spectrum it seems like a child with some
12 eccentricities and unusual predispositions, and yet,
13 that same pattern is reflected quite intensely when
14 one goes into undoubtedly autistic individual. That's
15 an area of a special interest of mine.

16 Q And now given the work that you're talking
17 about, doing the research, it sounds that your
18 clinical practice has ended, is that correct?

19 A I say effectively. Effectively it really
20 has. It's been sort of petering out over a while, and
21 I've been getting really very much more busy in the
22 last few years and I haven't really had time.

23 Q So is it fair to say that early in your
24 career you put a large amount of time into clinical
25 practice, had contact with thousands of children and

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1 have now sort of replaced that workload to a large
2 degree with research, book chapters, articles? Is
3 that a fair statement?

4 A It is.

5 Q Okay. Anything else in your background, in
6 your skills, your expertise, that you think can inform
7 the Special Masters on the issues that we're talking
8 about in these cases?

9 A Only the general statement that I've done a
10 lot of behavioral research, research with children,
11 with adults, with old people, with brain-injured
12 adults, with brain-injured children.

13 I'm very accustomed to the methodologies
14 involved, I'm very accustomed to reading the
15 literature on this kind of issue, I have refereed for
16 numerous medical and scientific journals. In my
17 current university teaching I teach methodology and
18 show students the elements of statistics, so I'm
19 capable of reading the literature and understanding it
20 and deploying critical interest to the extent of my
21 domain as a neurologist.

22 Q Okay. So appreciating the opportunity to
23 talk about your qualifications, let's go ahead and
24 start getting into the substance of your testimony
25 here today. I think a good starting point for that

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1 would be your expert report. This is an expert report
2 that was filed in April and shared with the Special
3 Masters and shared with Respondent. Do you have a
4 copy of that in front of you?

5 A I'm just checking. I don't see it here.

6 Q I know you have materials there. Just a
7 moment. We'll get one over to you.

8 A Yes.

9 Q Well, as we look for that report, Dr.
10 Kinsbourne, I think we can talk about the substance of
11 it and get that started because I'm assuming as you
12 sit here you're certainly prepared to testify about
13 the substance of the report without the paper in front
14 of you. Would that be fair?

15 A Yes, although I do have it right over there
16 in my little bag. At any rate, please go ahead, sir.

17 Q Thank you. So, Dr. Kinsbourne, the very
18 first section in your report is something called the
19 scope of your report. I want to note that your report
20 here is being offered for general causation and you're
21 not offering an opinion in either one of the specific
22 cases, either Jordan King's case or William Mead's
23 case, is that correct?

24 A That is correct.

25 Q So when you say you're offering an expert

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1 opinion on general causation could you describe for
2 the Special Masters exactly what you mean by offering
3 a report and an expert opinion on general causation as
4 distinct from case-specific causation here?

5 A My understanding of the category is that one
6 addresses whether a particular agent can inflict a
7 particular injury, and then, as a separate matter, one
8 addresses whether, indeed, it did in the particular
9 case. I am only addressing the first of these issues,
10 whether in principle the agent we're discussing can
11 cause some forms of autism or autistic spectrum
12 disorder.

13 I'm not giving any opinion about any
14 individual, and I have not reviewed files of any
15 individual case for that purpose.

16 Q And when we talk about the subject at issue,
17 as you mentioned, as it says in your report, we're
18 looking at thimerosal-containing vaccines, correct?

19 A Yes, sir.

20 Q And that is the subject of your report?

21 A Yes.

22 Q Can you describe for the Special Masters
23 what exactly you mean by putting TCVs on the list of
24 potential etiologies of autism in a differential
25 diagnosis where other causes have been worked out?

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1 I'm somewhat paraphrasing the expert opinion statement
2 because the Special Masters have read it.

3 I was hoping you could explain exactly what
4 you meant by framing your opinion that way.

5 A Yes. The thimerosal contains the element
6 mercury, and I discuss evidence as to whether mercury
7 in the brain can cause autistic behavior problems. If
8 it is the case that mercury can do that, and I will
9 explain why I think so, then any source of mercury
10 should be on the list of environmental factors that
11 should be considered among whatever other ones exist
12 as potential causes of the autism in a particular
13 child.

14 On that basis, thimerosal, which, as has
15 been amply discussed, contains mercury, should be one
16 of those sources to be considered as to whether it,
17 given all its toxicological properties, would be one
18 of the forms of delivery of mercury which should be
19 considered as potential causes.

20 Q And one of the reasons it would be
21 considered as a potential cause is that you do believe
22 to a reasonable degree of medical certainty that
23 mercury can induce neuroinflammation and result in
24 autistic symptoms, correct?

25 A And that, in summary, is the gist of what I

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1 say in my report, yes.

2 Q Okay. Great. Well, let's talk about the
3 different components of your report. You begin by
4 talking about the autism spectrum disorder and
5 particularly about regressive autism. Could you
6 describe just briefly what you mean when you say
7 autism spectrum disorder and particularly as how you
8 would distinguish regressive autism from the other
9 types of categories of spectrum disorders that would
10 be included? Could you describe that?

11 A Yes. Actually, the autistic spectrum is
12 really defined more along a dimension of severity than
13 it is on a dimension that includes a question of, was
14 the condition one that gradually emerged as the infant
15 got older, which is the majority of cases, or was it
16 one in which the infant apparently developed normally
17 or nearly so and then fairly precipitously over weeks
18 or a few months, typically in the second year of life,
19 became autistic, okay?

20 Now, it is my clinical impression that the
21 regressive type of autism tends to be cases among the
22 more severe type. So, for example, I am not persuaded
23 that Asperger's disease, which is one of the high
24 functioning ones, is a consequence of a regression.
25 If it is, I'm not aware of that. So the spectrum is a

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1 formula of to this difference between, as it were,
2 congenital versus regressive.

3 Q And so the main distinction between what
4 would be called regressive autism and other autism
5 spectrum disorders, it's not the types of symptoms
6 that appear, is that correct?

7 A That's correct. In fact, most studies have
8 not found very important differences in the outcome
9 between children whose autism gradually emerges and
10 becomes more and more clear, which is the usual
11 situation, and children who regress into such a state.

12 Some articles have found that the regressive
13 ones on average end up a bit more lower functioning or
14 severe and others have not found that, but
15 qualitatively, there is no difference in the pattern
16 of disabilities or abnormal behaviors.

17 Q So really the difference is that in
18 regressive autism, what characterizes that is a period
19 of normal, typical development that is followed by the
20 appearance of symptoms, so it's the time course of
21 regression that makes it distinct, is that fair?

22 A Correct, except with the proviso that
23 development doesn't have to be talking normal.
24 Another way of putting it is to say that for the first
25 period of time, say a year or 15 months, there was no

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1 evidence of autistic disorders. Now, a child who is
2 developing slowly isn't invaluable to whatever causes
3 the regression. It isn't that the autism gradually
4 comes to the fore until the second year of life and
5 sometimes later.

6 Q Now, in your paper, and you sort of alluded
7 to it now, the majority of cases begin with symptoms
8 of autism fairly early in life and then progress on a
9 continuum, and regression is a minority of those
10 cases.

11 A Right, and the important point is that it's
12 rather dramatic, that the child has achieved certain
13 milestones and amazingly somehow isn't doing what we
14 knew he or she could do not long before. Now, it's
15 much more common to have some children be slow
16 developers and then one doesn't quite know: Will they
17 catch up? Will they have a mental growth spurt?

18 Actually, to lose skills you already have,
19 that's something that really doesn't happen at all in
20 most other developmental disorders. I mean, children
21 with dyslexia don't begin to be able to read and then
22 stop, for example. So that is, at least from the
23 parents' point of view, a really rather startling and
24 perplexing situation. Certainly draws attention to
25 itself.

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1 Q And not only from the parents' point of
2 view, but presumably from a treating pediatrician's
3 point of view, correct?

4 A Should. It should do, yes.

5 Q Right. Now, in your report in discussing
6 what percentage of autistic children fall into this
7 regressive category on page 5 you describe 20 to 40
8 percent, at another point 20 to 30. We've heard
9 numbers of 10 to 20, numbers as low as 6 percent.
10 What can you tell the Special Masters to help them
11 reconcile these various percentages that are
12 attributed to regressive autism?

13 A Well, I'm not sure that I can persuade the
14 Master how to reconcile when different sources give
15 different figures and the figures are always
16 approximate. I mean, the majority the -- says 32.5,
17 you know? These are ranges. My best understanding of
18 it is that it's not always clear-cut.

19 The criteria for saying this is where
20 congenital finishes and this is where aggressive
21 begins vary. Some studies simply look for a child
22 stopping saying some words that he or she could say.
23 Others have a stricter criteria. They also look for a
24 change in play patterns, a change in socialization,
25 for example.

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1 Actually, Respondent's expert, Sir Michael
2 Rutter, has a very good discussion in his report of
3 the issues in classification of regressive autism.

4 If one were to speak of the really clear-cut
5 cases where you have significant amount of language
6 and it really goes and doesn't come back for many
7 months or years, or the child who has been playing in
8 a normal, active, curious fashion now starts just
9 lining everything up over and over again. A child who
10 is interested when his mother approaches, father comes
11 in through the door, a sibling comes and play sort of
12 ignores and looks by people and keeps doing whatever
13 he or she is doing, if you take as a clear-cut case
14 like that, then it will be a lower percentage.

15 Q And do you have an idea of how low? Twenty
16 percent? Ten percent?

17 A I think it probably would be below 20, but
18 I'm just describing this. What I'm saying is that the
19 reason for the different approximations I believe is
20 really a different subjective impression of what it
21 takes to be classified as regressive. Having said
22 this, when a child is clearly regressive, that's very
23 clear. This is not a challenge to the category, just,
24 like everything else in biology, there is really a
25 continuum.

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1 Q Now, you just mentioned just now that
2 regression can be very clear and you also described
3 earlier that it could be dramatic. I know that
4 there's a discussion in your report about the
5 incidence of autism in the general population, and you
6 describe the role of the regressive subtype of autism
7 within there as telling us something about the
8 incidence of autism. Could you describe that?

9 A Yes. As everybody knows nowadays, the
10 diagnosis of autism is made immeasurably, well, not
11 immeasurably but dramatically, more frequently now.
12 Some say 20 years ago maybe one or two children in
13 10,000. It was thought to be a rare condition. Now,
14 as we've already heard in this hearing, the figure
15 given in this country is one in 150.

16 Now, that is an enormous increase. Now,
17 other diseases have also increased in diagnosis.
18 Asthma has increased, for example, certain connective
19 tissue disorders have increased, ADHD has increased.
20 Nothing like as dramatically as the diagnosis of
21 autism.

22 Now, when that happens it is legitimate to
23 ask are we seeing a biologically-based increase in a
24 disease, or have diagnostic habits changed, have
25 criteria changed, have we become more alert to the

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1 possibility of such children being around, are we
2 finding them more efficiently? These are all very
3 legitimate questions, in my opinion.

4 And there's another suggestion as being
5 what's called diagnostic substitution, that a child
6 that might have been called something else, say
7 mentally retarded but having some autistic features,
8 might not be called autistic but retarded. You know,
9 you could sort of shift the label without changing the
10 case.

11 And I'm sure all of this happens, and I have
12 little doubt that it accounts for some of the
13 increase. I find it hard to believe that it accounts
14 for such an enormous increase, however. In thinking
15 about this it's usual to think of regressive autism as
16 a case in point. When a child loses skills in this
17 rather dramatic way, as we're discussing, it's really
18 hard to think of diagnostic substitution because
19 mentally retarded children don't do that.

20 You can't really confuse it with any --
21 there are one or two rare entities which I mention in
22 my report where you do get some regression but nothing
23 that could account for any of these numbers. If it's
24 a matter of not having noticed or thinking that the
25 child was actually just a bit eccentric, a bit odd,

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1 that can be applied to some children, the milder
2 cases, but this regression is noticeable, in my
3 opinion.

4 If it were just a matter of being more
5 alert, I think that would have less inference of the
6 incidence of regression for reasons I've explained
7 than on classifying children who don't develop quite
8 correctly and behave in certain ways.

9 So thinking of this and yet noting that
10 reports say that the proportion of autistic children
11 of the regressive subtype has more or less stayed the
12 same over decades tells me that the regressive subtype
13 has become enormously more frequent. I don't see how
14 that can be fully accounted for by these other
15 nonbiological factors.

16 Q Now, Dr. Kinsbourne, what you're talking
17 about is a perspective from somebody with clinical
18 practice and research in the field of autism. You're
19 not an epidemiologist?

20 A No, and I'm not making epidemiological
21 statements. I'm making more statements of
22 ascertainment and diagnosis.

23 Q And in fact in your report you specifically
24 leave it to the epidemiologists to discuss population
25 studies that are tracking the incidence of autism

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1 spectrum disorder?

2 A I do so gladly.

3 Q You would have to go back to school and
4 start a new course of study to be able to testify
5 effectively on that issue?

6 A Well, it's an option which I will consider
7 now that you brought it up, yes.

8 Q So I want to move on to talk about the
9 second topic of your report and that's this idea that
10 there are genetic and environmental factors that
11 combine across the population to produce autism
12 spectrum disorder. Do you recall that section of the
13 report?

14 A Yes, I do.

15 Q Now, there's no doubt, is there, Dr.
16 Kinsbourne, that there's a significant genetic
17 contribution to autism spectrum disorder? Is that a
18 fair statement?

19 A No doubt whatever.

20 Q And how do we know that? How do we know
21 there's a significant genetic contribution?

22 A Well, there a variety of sources of
23 evidence. One that's most usually quoted because it's
24 the most clear-cut is the study of twins. As the
25 Court I'm sure knows, there are two types of twins.

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1 They are monozygotic or identical and dizygotic or
2 nonidentical.

3 From the objective point of view, that means
4 that in the first case, the monozygotic, they will
5 actually have the same genome. They will have the
6 same pattern of genes. In the second case, that
7 pattern will only be 50 percent similar. No different
8 from just a brother or a sister not born at the same
9 time.

10 Now, the methodology is that to the extent
11 that the disorder is considered to be genetic, to that
12 extent if one twin has it, the probability the other
13 one also has it will vary such that if one twin of a
14 monozygotic pair has the disorder and it's fully
15 genetic, you would expect the other one to have it,
16 too.

17 Q And is that concordance?

18 A That is concordance. That is a high
19 concordance.

20 Q And there are studies that have been done to
21 look at concordance rates?

22 A Absolutely. However, in the dizygotic case
23 you actually find normal concordance and within
24 brother and sister of different ages, okay? So in the
25 highly genetic disorder there is a big gulf, and in

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1 autism there is a big gulf. There are actually two
2 ways in which it has been looked at.

3 One is looking at the strict occurrence of
4 autism in the second twin if there was autism in the
5 first. There, the concordance was about 60 percent in
6 typical figure.

7 If you admit what's called a broader
8 phenotype or spectrum so that you say one of the
9 siblings of the twins is autistic, the other one isn't
10 exactly autistic but has some other problems, other
11 disorders that have a similar flavor or have some
12 family resemblance, if you look at that, it's 90
13 percent. So there is absolutely a powerful genetic
14 factor involved.

15 Q Even given the strength of the genetic
16 factor and even given concordance studies, you still
17 have a range of say 10 percent to 40 percent of autism
18 cases, even between these twins, that are unexplained
19 by the genetics. There would have to be something
20 else involved, is that correct?

21 A That is true.

22 Q And if it's not genetics that's determining
23 the symptomatic outcome of these children, it would
24 have to almost by definition be something in the
25 environment, correct?

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1 A There are of course two kinds of other
2 things. One would be some damage. You know, maybe
3 one twin had a rougher birth than the other. You
4 know, there are some intercurrent factors to be
5 considered and they do occur. The other is that there
6 may be environmental factors which, as it were,
7 trigger a strong susceptibility into a clinical
8 actuality.

9 Now, the thing that I like to present to the
10 Court is this, that when you have a high concordance
11 between two twins, that doesn't show that genes do the
12 whole job. It may be that the concordance is not for
13 a gene which causes autism just like that but a gene
14 that makes them susceptible to an environmental factor
15 which when encountered will lead to autism.

16 That's called gene environment interaction.
17 So if the twins are identical they will both have this
18 so-called susceptibility gene, and then when they both
19 encounter the same challenge they will both succumb.
20 Whereas, if it's a dizygotic pair, the one individual
21 may, indeed, have the gene and succumb but the other
22 one is quite likely not to, and there you'll see a
23 much lower concordance.

24 Now, this concert of gene environment
25 interaction is now very generally accepted, it's

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1 researched, there are meetings organized by NIH on the
2 question of environmental factors without in the least
3 minimizing the importance of the genetic part of the
4 matter.

5 Q And, Dr. Kinsbourne, do we now know of
6 specific environmental factors that can lead to the
7 appearance of autism in children? Are you aware of
8 any?

9 A Yes. There are a variety of events that can
10 cause autism and it may be that some of them cause
11 autism without susceptibility, but more likely they
12 cause autism in susceptible people.

13 It's well-known that thalidomide, the toxin
14 which was inadvertently given to pregnant women in
15 Europe to control severe vomiting during pregnancy,
16 and did control it, and nobody had the least idea that
17 the children would then be damaged if was given at a
18 certain point in time during the pregnancy. There was
19 a high incidence of autism among those children.

20 Not 100 percent. Even there you have the
21 variability. That's one. The antiepileptic agent
22 dilantin, valproic acid, is known to cause autism at
23 certain times. It's been long known that if the
24 mother has a rubella, a German measles infection,
25 during pregnancy that there is a considerable risk of

1 autism.

2 There are one or two other factors, but
3 those factors exist. It's also the case that less
4 commonly disease happening after birth can cause
5 autism. There have been a number of case reports of
6 children who have had what is called herpes
7 encephalitis, an infection of the brain with the
8 herpes virus, which caused autistic syndromes.

9 Effectively, the child was autistic. There
10 was one well-known study by Bob DeLong who used to be
11 a colleague of mine presenting two children. They
12 were quite young, I forget the age, who had herpes
13 encephalitis, both became autistic. In that case,
14 actually, both then got better. Then Dr. Gilberg from
15 Sweden has published some cases of much older
16 children, I forget the age, age seven, eight and nine,
17 who became autistic.

18 These are rare events, but it simply
19 underlines what everybody agrees now which is that
20 there are diverse causes for autism even when you
21 can't tell that by looking at the child. In other
22 words, this is called a functional convergence. Many
23 different causes may converge to the same clinical
24 appearances.

25 So when you see the child you can't say, ah,

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1 this must be one of those.

2 Q And one quick note. Some of the
3 environmental causes you were describing are prenatal,
4 in utero exposures, correct?

5 A Right.

6 Q And some of them are postnatal exposures?

7 A Correct.

8 Q But whether prenatal or postnatal, they
9 would all fall in the category of a nongenetic
10 contributing factor to autism, correct?

11 A Right.

12 Q We're going to move on and talk about
13 mercury in the brain. Before we even do that just,
14 again, to make clear, the scope of your testimony and
15 expertise here. You're not a toxicologist. We
16 already heard from a heavy metals toxicology expert,
17 correct?

18 A Yes, sir.

19 Q You're here to talk primarily about the
20 childhood neurology and what goes on in the brain once
21 mercury is in the brain. Is that how you understand
22 the scope of your testimony?

23 A Not primarily, exclusively. I mean, I do
24 intend to stay within my domain of expertise.

25 Q Okay. So before we get into the description

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1 of the mechanism again that the Special Masters have
2 read and to talk about neuroinflammation, I would just
3 like you to give a quick description of the type of
4 brain structures we're going to be talking about
5 because there are different cells that we're speaking
6 about that do different things.

7 We've heard a lot of this scattered through
8 the literature so far. If you could sort of sum up
9 for the Special Masters those issues of brain
10 structure and function that are relevant to your
11 opinion?

12 A Yes, sir. Relevant to our discussion are
13 three types of cells in the brain: the neurons that
14 do the actual work, the control functions, of the
15 brain; the astrocytes, star-shaped cells which have a
16 variety of what we might call caretaker functions that
17 I will come back to and explain; then our connective
18 tissue cells in the rest of the body; and then there
19 are the microglia, which are part of what is called
20 the innate immune system and they are like cells
21 called macrophages in the rest of the body.

22 Now, I'd like to first talk about the
23 structure where a neuron communicates to another
24 neuron because that's basically what the brain does is
25 for the brain mostly talks to itself. Neurons talk to

1 other neurons, and at times they get information from
2 the outside world in one or other way or send orders
3 out.

4 As I'm sure the Court knows, when neurons
5 communicate with each other they do so by what is
6 called a chemical messenger, namely a
7 neurotransmitter, because the neurons aren't
8 continuous with each other like an electrical circuit.
9 They have discontinuities which are bridged
10 chemically. They're called synapses.

11 At the presynaptic end, where impulse is
12 coming from, there are structures which release the
13 neurotransmitter in question. That chemical diffuses
14 into the synaptic cleft, that very tiny structure of
15 fluid medium, and then the molecules that impact the
16 postsynaptic surface of the other neuron have receptor
17 sites sensitive to that chemical.

18 So the neurotransmitter leaves the
19 presynaptic end of the transmitting neuron, attaches
20 to the receptor surface of the receiving neuron, and
21 there, in one of several mechanisms, stimulates a
22 further impasse. Now, it is important from the point
23 of view of the survival of the brain not only to send
24 these messages but to control the amount of chemical
25 which actually passes.

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1 So around the synapse there are devices to
2 mop up those chemicals which didn't make it to the
3 receptor surfaces but diffused in various directions.
4 Why is it adaptive? Well, if they diffuse, they might
5 stimulate other synapses uncalled for and blur the
6 message, for example.

7 In one case it's even more important than
8 that because in the case of glutamate, which is my
9 main topic in my discussion, it's actually dangerous.
10 If too much glutamate escapes from synapsis it can
11 actually damage neurons, make them fire too much
12 causing seizures or even kill them. That's call
13 excitotoxicity. Now, I'll come back to that.

14 Now, some synapses have enzymes in the
15 synapse which break down the spare, unused
16 neurotransmitter, but more pertinent to my discussion
17 are other synapses which have what are called receptor
18 -- they have transporters. They have structures which
19 pick up the spare molecules and return them to the
20 neuron. So it's like you're recycling.

21 So the transporter sites are devices which
22 hold down the total amount of neurotransmission.
23 There are transporter sites on many neurons.
24 Interestingly enough, there are also transporter sites
25 for glutamate on the astrocytes. These glial

1 connective tissue cells have a caretaker function.

2 They mop up spare glutamate.

3 As I have described to the Court on another
4 occasion, actually, the astrocytes are wrapped around
5 the synapse almost like a basket, and, as the
6 glutamate diffuses, the reuptake areas on the
7 astrocytes reabsorb the glutamate and ideally then
8 pass it back to the neuron subsequently for later use.

9 So in this way what is maintained is called
10 homeostasis. Just the right balance of enough
11 glutamate to send the message but not so much that it
12 spills and causes mischief. Also, it's an economy to
13 save not having to manufacture more than is necessary.

14 Now, again, just making the salient points
15 for our discussion in among an enormously more complex
16 situation, the microglia are cells which when dormant,
17 when not doing anything, don't do anything. That's
18 different from the neurons and astrocytes that are
19 always doing something.

20 All neurons always fire. It's just a matter
21 of the rate at which they're going to fire. The
22 astrocytes do the work collaterally. The microglia do
23 nothing until there is a challenge, until some
24 invader, some invading agent enters the vicinity, is
25 detected chemically by the astrocyte and classified as

1 a potentially threatening substance.

2 That then causes what is called microglial
3 activation. The microglia undergo chemical changes,
4 they swell. They often manage to engulf the invader.
5 It could be a virus, it could be, pertinent to our
6 discussion, a small amount of a heavy metal. Whatever
7 it is, the microglia isn't specifically sensitive to
8 one kind of invader.

9 If there's something that shouldn't be
10 there, the microglia may internalize it. The
11 microglia also emit a number of chemicals called
12 cytokines, and many of the cytokines are called
13 proinflammatory cytokines, meaning they cause
14 inflammation.

15 What they are doing of course is to squirt
16 defensive chemicals at the invader, which in evolution
17 mostly would be the bacterium, really, to basically
18 kill it. But, as I say, the microglia don't discern
19 what exactly it is, they just fire. Among the agents
20 that the microglia release are what are called
21 reactive oxygen species, forms of oxygen which in fact
22 cause oxidative stress, which was explained in great
23 detail to the Court by Dr. Deth yesterday.

24 As he did explain, and as I have in my
25 report, these are substances that are apt to damage

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1 any cell in the vicinity as well as the invader. So
2 there is an issue of friendly fire. If the invader is
3 quickly dealt with and none is left, well, there's
4 inflammation, as it were, for a while, just if you
5 scratch yourself on the skin, but it goes away.

6 If the invader is still there in spite of
7 what the microglia are doing, then we can get a
8 chronic continuous outpouring of these cytokines and
9 other agents that can now be damaging to astrocytes
10 and damaging to neurons.

11 Q And so, Dr. Kinsbourne, the idea would be is
12 if there's this persistent invader so to speak the
13 microglia continue to recognize it and just keep
14 pounding away with their inflammatory response,
15 correct?

16 A That's correct.

17 Q And so you get a condition where you have an
18 ongoing chronic process, is that right?

19 A Yes.

20 Q And that ongoing chronic process is
21 releasing cytokines and reactive oxygen species?
22 That's ongoing?

23 A Well, that is ongoing in those situations.

24 Q When proinflammatory cytokines and reactive
25 oxygen species are being released in a chronic way,

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1 you're describing that can actually damage the
2 structure as well as the function of neighboring
3 cells?

4 A Yes. It actually particularly apt to damage
5 the astrocytes.

6 Q Why is that?

7 A Ultimately, one would have to ask that
8 question of nature and nature isn't forthcoming, but
9 the fact is that the astrocytes are quite vulnerable
10 to being attacked by this friendly fire, including, in
11 a particular way that's pertinent from my perspective
12 on the matter, which is that the attack from the
13 microglia impair the astrocytes' ability to scavenge
14 glutamate.

15 In other words, they block the transporter
16 on the astrocyte and actually also cause the astrocyte
17 to release more glutamate as opposed to take it out.

18 Q So this is sort of a vicious cycle rather
19 than a virtuous cycle that results?

20 A It sounds vicious, yes. Definitely. So
21 this actually happens even at the stage when the
22 neurons are not yet materially damaged. So the
23 effects of this microglia activation on neurons is
24 often mediated by the effect on the astrocytes. They
25 seem to be first in the firing line.

1 So if the astrocytes are no longer
2 scavenging the glutamate, then the glutamate is able
3 to accumulate and spill over. So what will then
4 happen is that glutamatergic cells will fire more than
5 they could/should, meaning more than is called for by
6 the particular situation, and also that more of them
7 will fire because of the spilling to enabling
8 synapses.

9 So you're going to get a general excitation
10 of the brain, a higher excitation level of many parts
11 of the brain.

12 Q And that higher excitation level is a result
13 of this excess of glutamate, which is an excitatory
14 neurotransmitter?

15 A Right. Now, and then the next step that can
16 happen is that the excitation of certain neurons is so
17 great it kills them, and that's called excitotoxicity.
18 It's called excitotoxicity. So you have the excito,
19 which I've described, and then if it's too intense
20 it's actually toxic to the neuron that receives the
21 glutamate and kills the neuron.

22 It's a very well-known phenomenon and it
23 occurs in numerous disorders. None of this happens in
24 just one disease. This is a rather general mechanism.
25 Now, in the brain, the brain is so organized as to

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1 maintain the level of excitation within boundaries.

2 One of the ways it happens is that there is
3 of course also in the brain inhibition. So there are,
4 whereas the glutamate, which is a very frequently seen
5 neurotransmitter, is excitatory, there are
6 neurotransmitters which are inhibitory. And GABA,
7 G-A-B-A, is the main one that's spoken about.

8 There's a thing called the glutamate/gaba
9 ratio which basically decides the level of general
10 excitation in the brain. So there tends to be a
11 certain amount of homeostasis and it's kept within
12 certain bounds, but, as one perceives, if the
13 glutamate is out of control in the way I've described,
14 then excitation goes up and the excitatory level in
15 the brain increases.

16 Well, how could that be held in balance?
17 Only if the gaba also goes up to sort of track it and
18 that, to some extent, can happen. As Dr. Deth
19 explained yesterday, the mechanism that he was
20 discussing with the D4 receptor sites, if the Court
21 recalls, and the D4 receptor sites, I think he pointed
22 out, project onto GABAergic neurons, neurons that
23 produce GABA, and impair the production of GABA.

24 So the ability of the inhibitory cells to
25 keep up with the increasing level excitation is

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1 limited. So overall, there's going to be an
2 overexcitation, overactivation of the brain.

3 Q And ultimately, Dr. Kinsbourne, is it true
4 that the astrocytes not just being impaired can
5 actually die, is that correct?

6 A Yes. The astrocytes, again -- see what
7 happens typically is that if the immune challenge
8 continues there actually is an increase in the number
9 of microglia.

10 Q So let me interrupt. Now, you've talked
11 about the microglia that are there change their size
12 and get larger, correct?

13 A Right, right. They can actually multiply if
14 the situation continues.

15 Q And is that proliferation?

16 A Yes. They proliferate, there are more of
17 them, because there's more work for them to do.

18 Q So there's actually three things that are
19 going on with the microglia. There's the activation
20 of the microglia, proliferation of microglia and then
21 those new microglia, so to speak, are active, also, is
22 that correct?

23 A Right, and then they will do the same thing.
24 Now, the astrocytes are then bombarded in the way that
25 I've described and over time they will die. They will

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1 be apt to fall out one after the other. When they die
2 they leave a typical appearance called gliosis, which
3 is analogous to a scar on the body.

4 So it's just like some dead material, which
5 is the traces that there were astrocytes which are
6 glial cells, were there, so that over numerous years
7 what one will tend to see would be an increase in
8 microglia, a decrease in astrocytes, the appearance of
9 gliosis and in severe cases also loss of neurons.

10 That's the simple statement of the complete
11 picture. Just to refer you to a source, among the
12 articles submitted was one by Lopez-Hurtado.

13 MR. POWERS: And let's go ahead and put that
14 up, Scott. For the record, also, this is Petitioners'
15 Master Reference List No. 0446. Scott, if you could
16 just blow up the title.

17 BY MR. POWERS:

18 Q Dr. Kinsbourne, is this the article you're
19 talking about? It's entitled *A Microscopic Study of*
20 *Language-Related Cortex in Autism*.

21 A Yes, it is.

22 Q And this is a study that I think has been
23 discussed earlier, but it involves an analysis of
24 autopsied brains of people who had autism, is that
25 correct?

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1 A I actually don't recall that it was
2 discussed earlier, but at any rate, if I may discuss
3 it briefly?

4 Q Yes, please.

5 SPECIAL MASTER CAMPBELL-SMITH: Pardon me,
6 Dr. Kinsbourne. I'm just going to ask Mr. Powers that
7 we would give Dr. Kinsbourne an opportunity first to
8 describe what he recognizes about the article.

9 MR. POWERS: So, Dr. Kinsbourne, you see in
10 front of you a screen and on that screen is an exhibit
11 that's been marked 0446. Could you describe for the
12 Special Masters and for the record what that is?

13 THE WITNESS: You see, I'm in the throes of
14 an approach/withdrawal conflict. I like to look over
15 there to read this. That takes me away from the edge
16 that the Court has told me to occupy. Then I have to
17 move these things. So I'm juggling for a moment.

18 SPECIAL MASTER CAMPBELL-SMITH: Okay.

19 THE WITNESS: This was a study of brains
20 taken from a bank of, you know, an official repository
21 of brains from autistic individuals who died for
22 various reasons. These investigators decided to focus
23 on a particular area of the cerebral hemispheres,
24 namely the area that subserves language.

25 That was a reasonable choice because, as the

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1 Court knows, problems with language are particularly
2 salient in many autistic individual, most of them. So
3 they thought they'd look at language-related cortex,
4 and that is what they did.

5 Now, what they found in brief was the
6 situation I just alluded to, a proliferation of
7 microglia, a diminution of the density of astrocytes,
8 the presence of gliosis and some loss of neurons.
9 Interestingly enough, they looked at an age range
10 between seven and 44 I believe, a wide age range, and
11 they found that the older the age of the person when
12 they died, the more striking those changes were.

13 So it seemed as if an ongoing process over
14 all these years gradually increased the number of
15 microglia, knocked out astrocytes, and finally,
16 knocked out neurons, too. So this is more likely
17 towards the end point of a long process which began
18 apparently by some challenge to the microglia many,
19 many years earlier.

20 MR. POWERS: And, Dr. Kinsbourne, if we
21 could look at page 11 of the exhibit.

22 Scott, we're looking at page 11 of 16 of the
23 exhibit that's still in front of you. On the text of
24 the study it's page 140 but the exhibit number is 11.
25 Scott, if you could blow up the highlight portion at

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1 the bottom of the left column and going up to the top
2 of the right-hand column?

3 BY MR. POWERS:

4 Q Now, Dr. Kinsbourne, take a look at that.
5 I'm not going to ask you to read it, read it out loud
6 that is. Take a look at that and describe for the
7 Special Masters why that's significant to you.

8 A Well, these sentences do refer to the
9 proliferation and this hypertrophy, the swelling, and
10 what's called the reactive gliosis, the gliosis that
11 was caused as a reaction to the death of the
12 astrocytes.

13 Q And so I just wanted to make clear that when
14 they're talking about the gliosis here, they're
15 talking about astrocyte death?

16 A Yes, yes. When neurons die they don't leave
17 gliosis, astrocytes do.

18 Q Anything else in that section, Dr.
19 Kinsbourne, that you find significant that you'd want
20 to share with the Special Masters?

21 A Not really. I mean, the interesting thing
22 is that there are actually more glial, and yet, more
23 glial have died, so it's like sending in more troops.

24 MR. POWERS: All right. So, Scott, if you
25 could pull that down and we're going to look on that

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1 same page, which is page 11 of the exhibit, page 140
2 of the article, there's another section. Thank you.

3 BY MR. POWERS:

4 Q So, Dr. Kinsbourne, take just a second and
5 take a look at that. Again, we're not going to read
6 it into the record since the Special Masters have
7 this, but if you could explain the significance?

8 A Well, the point made here is that what they
9 see would be compatible with the effect of a toxin on
10 the brain.

11 They point out that indeed, particularly
12 metals, as they say, such as lead, iron and mercury,
13 are known specifically to cause glial proliferation,
14 which we discussed and which was seen. And they point
15 out that metals have this toxic effect to increase
16 oxidative stress, and indeed they cite evidence that
17 both increased metals and increased oxidative stress
18 are reported in autism. So they are saying that this
19 is one way of explaining what they saw.

20 Q And then, Dr. Kinsbourne, in the discussions
21 in this paper of glial cells and gliosis, is that
22 consistent with your description in your report and
23 your testimony of the neuroinflammatory process?

24 A Absolutely. Yeah.

25 Q So this is autopsy evidence that supports

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1 not just that it's a conceptual idea but that it
2 actually happens in the human brain?

3 A That is correct.

4 MR. POWERS: Okay. Just pull that down.

5 BY MR. POWERS:

6 Q And we're going to keep talking primarily
7 about neuroinflammation, Dr. Kinsbourne, but as a
8 quick note, in your report you do describe your review
9 of the literature and your citations to the literature
10 that describe how inorganic mercury in the brain is
11 related to this process. Do you recall that
12 discussion in your report?

13 A I do. I'd like to preface that by a more
14 general statement about neuroinflammation. There are
15 numerous different causes that could send up
16 neuroinflammation. The first point is that when you
17 look at the inflammation you can't tell what the cause
18 was. You have to separately look for the causative
19 agent and see if you can find it.

20 Now, the causative agents fall into three
21 categories. One category is viruses, namely
22 neurotropic, neuropathic viruses that have the
23 capability of staying for long periods of time.

24 The second category is toxins, the specific
25 toxins of which heavy metals are mostly mentioned and

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1 that include mercury, although there are other ones,
2 and I think we discussed terbutaline at some point
3 earlier in the hearing which is obviously not a metal
4 and nonetheless is related to neuroinflammation by
5 some research.

6 The third category is neurodegeneration, and
7 I want to explain that for a moment. One of the
8 situations in which microglial cells detect an
9 unfamiliar agent in the vicinity is if cells are
10 actually breaking down and emptying out their
11 contents.

12 Now, for the neuroglia, those contents are
13 new, they haven't previously experienced them, so the
14 neuroglia might react to the contents of breaking up
15 cells as if they were invaders. That is why
16 particularly in the well-known diseases of Alzheimer's
17 disease and Parkinsonism in which neurons break down
18 there is neuroinflammation thought to be secondary to
19 the neurons spilling their contents and the microglia
20 reacting to that spillage.

21 So when one is looking for the causes of
22 neuroinflammation in a given case one of the things to
23 look for is a neurodegenerative disease. That's on
24 the list of differential. You would look for viruses,
25 though of other type described, and you would look for

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1 toxins that are known to potentially have this kind of
2 effect.

3 Q And so what you're talking about here is if
4 you're looking at a child with autism and you're
5 considering thimerosal-containing vaccines and that
6 differential diagnosis you would be looking at the
7 first stage to rule out other causes of
8 neuroinflammation, is that right?

9 A Yes, and there are a number of them.

10 Q So assuming those are then ruled out,
11 thimerosal-containing vaccines, in your expert
12 opinion, ought to be on the list for consideration as
13 to what might be causing the neuroinflammation?

14 A Right. Indeed, any form of mercury, however
15 delivered, should be considered.

16 Q And in these cases, as you describe in your
17 report, we're talking in particular about the
18 inorganic form of mercury? It's been referred to as
19 HG++.

20 A Correct.

21 Q And that's what you're talking about in your
22 report, correct?

23 A Yes, sir.

24 Q And is it your belief to a reasonable degree
25 of medical certainty that thimerosal-containing

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1 vaccines in fact deliver inorganic mercury to the
2 brain?

3 A Yes.

4 Q What's the basis for that opinion,
5 understanding that you're not a metals toxicologist?

6 A Right, I'm not. My understanding is, and I
7 think it was effective in discussions, you know,
8 around the testimony of Dr. Aposhian, but it is that,
9 as is well-known, thimerosal is 49 percent ethyl
10 mercury and ethyl mercury is transported. When it
11 enters the body, some of it does go into the brain.

12 The ethyl part of the molecule makes that
13 possible. However, in the brain the ethyl is broken
14 off gradually from the metal itself, and now the metal
15 has no way to get out again so it stays, and it stays
16 probably for years, for all we know, indefinitely.
17 It's like the roach motel, you know? You check in,
18 you don't check out.

19 (Laughter.)

20 I understand from toxicologists that really
21 all of us have some amount of mercury because there
22 are ambient sources from factory fumes, and from
23 dental amalgams and from fish, you know, there are all
24 these sources of mercury.

25 So the notion that inorganic mercury, once

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1 the mercury is delivered in a vehicle that can bring
2 it into the brain, is apt to accumulate there is not
3 welcome novel or controversial, as far as I can tell.

4 Q And it's your expert testimony to a
5 reasonable degree of medical certainty that the
6 inorganic mercury in the brain that could be delivered
7 by thimerosal-containing vaccines is an agent of
8 neuroinflammation, correct?

9 A It could indeed cause the inflammation, just
10 like mercury of any other kind could.

11 Q Now, in causing neuroinflammation,
12 neuroinflammation is not by definition autism, is it?

13 A Not by definition.

14 Q Neuroinflammation itself doesn't define
15 autism, does it?

16 A No, no.

17 Q So could you explain to the Special Masters
18 then really sort of the, I would describe it as the
19 last component of your model or mechanism of injury
20 here whereby neuroinflammation might express itself as
21 the symptoms of regressive autism?

22 A Well, actually, that's the part I enjoy
23 most, but I'd like to before that perhaps to present
24 to the Special Masters some evidence that mercury in
25 fact has an effect that leads me to believe it can

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1 cause neuroinflammation by the glutamate route.

2 MR. POWERS: Right, we should do that, and I
3 think we're going to refer to a couple of articles
4 here. I'll ask Scott if you could pull them up. The
5 first one that we're going to pull up is, let me make
6 sure I get the exhibit number correct for you, this
7 would be Petitioners' Master Reference List Exhibit
8 0570.

9 BY MR. POWERS:

10 Q Dr. Kinsbourne, do you see that on your
11 screen?

12 A Yes. That's Dr. Aschner's article.

13 Q Okay. Do you need a paper copy, also, or
14 are you comfortable working off the screen there?

15 A I am, actually. Yeah. So far I'm fine.
16 Please go ahead.

17 Q Okay. Great. So you see the exhibit on the
18 screen in front of you. Can you identify for the
19 Special Masters and for the record what that is that
20 you're looking at on that exhibit? It's a medical
21 article.

22 A Yes. The title is *Involvement of Glutamate*
23 *and Reactive Oxygen Species in Methyl Mercury*
24 *Neurotoxicity.*

25 Q Now, in general, before looking at any

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1 passages in that article, can you describe for the
2 Special Masters why this particular article is
3 significant to you and why you're including it in your
4 testimony?

5 A Yes. Well, first of all, Dr. Aschner is a
6 neurotoxicologist and he's particularly well-
7 credentialed and respected one. Secondly, he has
8 taken up the particular question of how it is that
9 mercury becomes neurotoxic. What is the mechanism by
10 which it does that? He's mentioning two things:
11 glutamate and reactive oxygen species.

12 Now, the reactive oxygen species was
13 discussed yesterday by Dr. Deth. I would like to
14 focus on the glutamate part. I think in quotations to
15 come we will see what Dr. Aschner says about the role
16 of glutamate in that process.

17 MR. POWERS: Yes. And let's go ahead and
18 look at the relevant passages here, the ones that you
19 have identified as significant. On page 2 of the
20 exhibit, Scott, there's a highlighted section there
21 that let's go ahead and blow up.

22 BY MR. POWERS:

23 Q If you could take a look at that, Dr.
24 Kinsbourne, and as we've done here not read it aloud
25 but just explain the significance of this section to

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1 your opinion.

2 A Right. The methyl mercury here has in fact
3 the property that I've referred to of inhibiting the
4 uptake of glutamate by the astrocytes thereby
5 increasing glutamate concentrations in the external
6 fluid. The -- glutamate is now bathing the neurons to
7 an excessive extent and sensitizing them to
8 excitotoxic injury, which is specifically what I
9 described.

10 So Dr. Aschner is stressing that this
11 organic mercury compound really is apt to increase
12 glutamate to its toxic effect, which then leads me to
13 think of it in line with other agents, such as
14 viruses, as a realistic cause of overactivation of the
15 brain, to which I will come back in a moment.

16 Q And we should make a note here that Dr.
17 Aschner's paper, this review paper, is talking about
18 methyl mercury in particular, correct?

19 A Yes.

20 Q So in this paper he's not distinguishing
21 between methyl mercury and its inorganic HG++
22 byproduct, is that right?

23 A That's true.

24 Q Does that make a difference in your opinion
25 in terms of the neuroinflammatory process that's

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1 induced?

2 A No, because, as we have said, we're really
3 thinking of the mercury itself.

4 Q Okay. So let's go ahead and look at page 4
5 of the exhibit, please.

6 SPECIAL MASTER HASTINGS: Mr. Powers, before
7 we go on to that, let me just note for the record that
8 the sentence you were referring to in the right-hand
9 column is the sentence that begins with the small
10 letter *c*, the one sentence that begins with methyl
11 mercury. Go ahead now.

12 While I agree we don't want to read a long
13 paragraph, we need to identify it somewhat so when we
14 go back and read the transcript here we know which
15 section of the page he's talking about.

16 MR. POWERS: Okay. I'll sort of try to take
17 the lead with Dr. Kinsbourne and focus him and make
18 the record really clear. I appreciate that.

19 SPECIAL MASTER HASTINGS: That would be
20 wonderful.

21 MR. POWERS: So if we could, Scott, pull
22 down that section and move on to page 4 of the
23 exhibit. Now, there's a very long section, basically
24 the second half of the left column on page 4 of the
25 exhibit, has been highlighted. So if we could just,

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1 Scott, zoom in to the very first half of that. Maybe
2 a little bit less than that. The first full sentence,
3 which would go up to where the footnote that says 46.
4 Yes. Correct.

5 BY MR. POWERS:

6 Q So, Dr. Kinsbourne, we're now looking at the
7 first full sentence of the highlighted section on page
8 4. Take a look at that and again explain the
9 significance of that to the Special Masters.

10 A Yes. It says that the methyl mercury
11 inhibits the astrocyte glutamate transporter, so the
12 astrocytes don't take up the glutamate they should.

13 Therefore, there's more left on the outside,
14 leads to increased glutamate concentrations in the
15 extracellular fluid, the fluid around the cell, and
16 that hyper activates certain glutamate receptors,
17 namely the so-called NMDA receptors, and by
18 hyperactivating them it admits sodium and calcium ions
19 to go into the cell.

20 That's actually of importance because if too
21 much calcium goes in, the cell dies. That's a way of
22 understanding the excitotoxicity aspect. Calcium has
23 a critical role in the transmission of the nerve
24 impulse but not if it's there in excess.

25 MR. POWERS: Okay. Now, we're going to move

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1 on to the next section. Scott, I'll appreciate your
2 skill here. We're going to begin with the word
3 "increase." It's the next full sentence after the one
4 you highlighted. We're going to go down that full
5 sentence to footnote 17 and 18. You got it. Thank
6 you.

7 BY MR. POWERS:

8 Q So, Dr. Kinsbourne, take a look at that.
9 It's the second full sentence in the highlighted
10 section on page 4 of the exhibit.

11 A Yes. It actually takes that point further
12 and says if you increase the calcium levels in the
13 neuron then you are generating reactive oxygen
14 species, those dangerous forms of oxygen that we
15 referred to before.

16 Q Excuse me. When you say referred to before,
17 that was in your testimony?

18 A Well, referred to I think by Dr. Deth, but
19 anyhow, I have also already mentioned them. These are
20 well-known forms of oxygen which cause oxidative
21 stress and injure the metabolic function of cells.

22 MR. POWERS: And in fact Scott, if you could
23 go to the next section, the very next sentence
24 beginning this ROS formation.

25 BY MR. POWERS:

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1 Q Dr. Kinsbourne, does that complete the
2 thought on reactive oxygen species?

3 A Yes. It just goes on to talk about what the
4 ROS then does to the mitochondria, which are in the
5 central part of the metabolic workings of the cells,
6 and it says overproduction of reactive oxygen species
7 mediated, at least in part, by glutamate. So it
8 brings us back to the glutamate as being the mediator
9 of this potentially dangerous situation.

10 Q And when you say glutamate is the mediator,
11 glutamate is the excitotoxic or the excitatory
12 functional neurotransmitter?

13 A Right.

14 Q Okay. So let's go ahead and pull this
15 article down. I know there's another piece of work by
16 Dr. Aschner that we should discuss. This is going to
17 be Petitioners' Master Reference No. 0568. We'll
18 pause here and let that come up on the screen. So,
19 Dr. Kinsbourne, in front of you do you see the exhibit
20 on the screen, and if you do, could you go ahead and
21 describe for the record what that is?

22 A Yes. The article is called *Methyl Mercury*
23 *Alters Glutamate Transport in Astrocytes*, and it's by
24 Dr. Aschner and others.

25 Q Okay. What's the significance of this

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1 particular journal article to you?

2 A Well, actually, this is the same point that
3 we already made in the other one.

4 MR. POWERS: So we'll be brief here. I just
5 want to turn to page 3 of this exhibit. Scott,
6 there's actually a highlight up before. If you look
7 at the top of the right-hand column on page 3, the
8 partial -- yes. Thank you. And then if you could
9 just zoom that in to the word because, beginning the
10 sentence because glutamate.

11 BY MR. POWERS:

12 Q So, again, to make a clean record here,
13 we're on page 3 of the exhibit, it's a continuing
14 paragraph on the top of the right-hand column and it
15 is the second complete sentence beginning there. Do
16 you see what I'm referring to, Dr. Kinsbourne?

17 A Yes. This is actually a bridge to Dr.
18 Deth's testimony yesterday. It says what he pointed
19 out, that the glutamate and cysteine share the same
20 transporter. So if there is more glutamate around, it
21 could compete with cysteine transport into astrocytes,
22 and Dr. Deth explained in detail why that's harmful to
23 the functioning of the astrocytes.

24 So that may be one reason why the astrocytes
25 are under strain and many ultimately die.

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1 Q And that reason being oxidative stress?

2 A Yes.

3 MR. POWERS: Okay. Let's go then, the last
4 thing we'll talk about here is, Scott, at the bottom
5 of the right-hand column of page 3 of the exhibit, the
6 last full paragraph, and we're looking at the first
7 sentence of that paragraph.

8 THE WITNESS: This makes the important point
9 that methyl mercury preferentially accumulates in
10 astrocytes. So I mentioned before that it accumulates
11 in the microglia, which engulf it. It also
12 accumulates in the astrocytes. You will find them in
13 those two cells before you find them in neurons,
14 actually.

15 It says here that this fact is a causal
16 factor in why methyl mercury makes neurons degenerate
17 because I believe the point he is making is that if
18 the astrocyte is disabled, the neuron lacks its
19 support. It becomes less viable.

20 BY MR. POWERS:

21 Q And, again, this is consistent with your
22 theory that you described in your expert report about
23 neuroinflammation and its sequelae in the pediatric
24 brain.

25 A Right. I want to just stress this. I

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1 stress the point that it isn't that everything dies.
2 If everything dies, then nothing would happen in the
3 brain at all and that's not the situation. It's
4 rather that the brain gets into an abnormal state,
5 it's still functioning, and the neurons don't
6 necessarily die, or if they do, some might, but most
7 don't. It is more that the neurons that are there are
8 firing too much.

9 MR. POWERS: So let's go ahead and pull this
10 down, Scott.

11 BY MR. POWERS:

12 Q Dr. Kinsbourne, are you prepared now to talk
13 about the overactivated brain and how it might be
14 expressed as the symptoms of autism?

15 A Yes. So I've mentioned that as the
16 glutamate levels in the brain go out of control they
17 will tend to stimulate more neurons to fire and
18 neurons to fire more frequently so that the excitation
19 inhibition ratio will increase.

20 Now, that is a situation which is consistent
21 with one of the existing models of what brain change
22 it is that underlies autistic behavior and which I
23 called in my report "the overarousal model." Now, I
24 want to preface this by saying that there are several
25 different models of what abnormality it is in the

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1 function of the autistic brain which causes autism.

2 This is not an open-and-shut case. I'm
3 presenting a particular model, and I'm pointing out to
4 the Court that if one adopts that model, then there
5 becomes a clear connection between neuroinflammation
6 and why that brain should manifest autistic behavior.

7 I don't want to imply that therefore
8 everybody should instantly agree that that is the
9 correct model. It is one that I have thought about
10 actually ever since I met its originators back in the
11 1960s, some people called Hutt and Hutt at Oxford who
12 first wrote about overarousal.

13 The model has been around a long time, it's
14 been referred to by some excellent scientists. It's
15 only one of several. Now, there is some evidence that
16 the brain of autistic individuals is overaroused.

17 I submitted an article by a group I think
18 from Rhode Island showing that their autistic subjects
19 had elevated heart rates, that instead of having a
20 flexible heart rate which goes up when one is active
21 and excited and goes down when one is at rest the
22 autistic individuals had a higher heart rate which
23 didn't seem to change.

24 It's like they were in a what's called
25 sympathetic autonomous. The sympathetic nervous

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1 system increases heart rate and if the sympathetic
2 nervous system is functioning at a high level, the
3 heart rate just stays up. You can imagine a person
4 who is very anxious or panicky and they can feel the
5 heartbeat going fast.

6 Well, these children who actually don't show
7 moment to moment distress actually I believe have a
8 fast heart rate and feel the way one does when one
9 does. Then there's evidence from EEG studies and some
10 evidence from neuroimaging also that at least some
11 systems in the brain, such as the amygdala, which has
12 to do with emotion, is overaroused.

13 So, actually, you have an individual, an
14 autistic person, who can't tell you how they feel
15 actually, and you can't tell on their face how they
16 feel, but if you measure them psychophysiologicaly
17 you can infer that they are actually in some turmoil,
18 that they're in an overaroused state.

19 Now, under those circumstances one would
20 expect a variety of consequences. The consequences
21 would be for how well they can do certain kinds of
22 cognitive functions, the kind of environments in which
23 they like to be and what they do to help themselves
24 when they are particularly overaroused. These are
25 three categories of manifestations of autism.

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1 The first one is that as the arousal level
2 of the brain rises, so the focus of one's attention
3 constricts. Now, that's not new. That was first
4 described in 1959, and I have a reference, and not
5 controversial.

6 You can imagine, for example, imagine an
7 expert testifying before a Judge and the expert is
8 understandably nervous and over odd and his attention
9 now is to the point that all he can look at is maybe a
10 part of the Judge's face and all he can think is one
11 thought.

12 As the overarousal goes up, what you can
13 consider in solving a problem diminishes. It's called
14 the narrowing of cue, C-U-E, utilization. Now, we see
15 as a powerful phenomenon in autistic individuals how
16 when they perceive the world it seems as if they're
17 perceiving it in very tiny bits of fragments.

18 Been described for decades that they might
19 look at a person's left ear rather than the person's
20 face, or they might notice a scratch on an object
21 rather than the object. It's a very dramatic focus,
22 narrow focus, and that is something that is known to
23 come with overactivation.

24 Now, I'm not saying that it's the only
25 reason for it, but it's compatible with

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1 overactivation. Now, if you have a very narrow focus
2 then there's a lot of things you won't notice in the
3 world and there's a lot of things you won't learn
4 about the world.

5 If that focus is diverted away from people
6 then you won't learn about people, other people: how
7 they feel, what they intend, what they think, what
8 their reactions are, what they think about you, all
9 the things that most of us are really good at.

10 I mean, we are evolved to be very good at
11 that but we can't get this information if we aren't
12 seeing the face. So the autistic person is
13 notoriously apt to have gaze avoidance. They don't
14 look at you, they look by you. This has been shown
15 elegantly.

16 They'll look at, you know, some furniture in
17 the back or a world, they look by people's faces.
18 That way they're not going to pick up the information
19 they need for social interaction, and of course
20 they're notoriously bad at social interaction. Also,
21 for a brain to function well routinely in simple
22 situations, that's fine.

23 You focus on what it is and you do it.
24 Suppose you have to do a complex task or a novel task?
25 Well, to do that you have to put things together. You

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1 have to look at one thing, consider a second thing,
2 remember a third thing. So you can see different
3 parts of the brain are active.

4 When that happens, if the brain is
5 overaroused, that differential of all these patterns
6 in the brain collapse into one. This incident is the
7 point that Rubinstein and Merzenich make in an article
8 that was submitted. So, as has been stated, autistic
9 people are much better at simple tasks than complex
10 tasks. Well, everybody is, but disproportionately
11 better at simple tasks than complex tasks.

12 My understanding is is for complex tasks you
13 have to hold various parts of the brain together in a
14 very patterned way, and the overactivation really goes
15 against the pattern because to keep a pattern in your
16 mind you have to have inhibition between the activated
17 areas and the inhibition is suppressed, as I've
18 described.

19 Now, if one is in the situation in which one
20 is already overaroused and it doesn't feel good, one
21 is going to try to be in environments that are low
22 arousing, understimulating environments. It's
23 opposite of of course sensation seeking, which is the
24 opposite problem of being underaroused.

25 So a person who is autistic would like

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1 everything to be stable, everything to be the same.
2 It's very upset if you move something that seems
3 inconsequential to anybody else, but why did you shift
4 this? Why did you move my stuff? They like
5 everything the same environment.

6 They are very tentative about change being
7 introduced, even a stranger coming into the house or
8 even not knowing when a familiar person is coming, but
9 when are they coming? So they have these constant
10 questions to assure themselves, that have organized
11 what's going to happen, and nothing surprising
12 happens.

13 That's also why it's thought they don't look
14 at people's faces because people's faces are actually
15 probably the most common source of surprising things,
16 out of control. So they like to remain in a very
17 stable situation, and they like to deal with things
18 that they can control, limited sets.

19 For example, an autistic child might know
20 about every type of shark that exists, and specialize
21 in it and think about it constantly. Well, there are
22 only so many sharks that you can know, none of them
23 are going to come on land to get you. Same as applies
24 to dinosaurs. They liked dinosaurs before anybody
25 else did.

1 There are many examples. They have narrow
2 interests. They don't like to deal with open-ended
3 questions. They like to have well-defined situations
4 that they can organize, arrange. In those situations,
5 they can become even savant.

6 They practice it so much they can tell you
7 all sorts of information in this narrow category that
8 you don't even want to know but they seem to find
9 congenial and comforting to work with. The propensity
10 of autistic individuals to hold sensation down is also
11 shown in how they arrange things.

12 As the child regresses into autism, he or
13 she will stop playing with the train set by going
14 vroom, vroom, vroom, thinking of a little one, and
15 instead line up the cars in a line or go to the
16 supermarket and line up the diapers on the shelves so
17 they all face the same way.

18 These behaviors, which are not particularly
19 adapted, don't seem to serve any particular use, I see
20 as being soothing to the individual, holding down the
21 level of stimulation. Incidentally, being still and
22 addressed is not soothing. What is soothing is
23 behaving repetitively.

24 The Court won't take it amiss if I mention a
25 little poem I wrote in my head for autists. It goes

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1 like this. One potato, one potato, one potato, one.
2 One potato, one potato, one potato, one. That's my
3 tribute to autistic view. So these are preferences
4 which are not just strange or arbitrary, they are
5 adaptive. The child is trying to keep herself in a
6 stable situation where that is threatened constantly.

7 What happens when the child can't control
8 the environment, when the stranger is coming, when her
9 mother takes her to a birthday party? She doesn't
10 want to go, but her mother feels she should be
11 socialized, and she comes into this uncontrollable
12 gang of children, and her mother pushes her in and
13 says go, go talk to everybody.

14 What happens often enough is the child just
15 goes wild, out of control, can't handle it,
16 overstimulated. In a situation short of that the
17 child will go into the very well-known stereotypic
18 behaviors: flapping, whirling, foot tapping, you
19 know, doing repetitive movements with the fingers,
20 making various movements which are called manneristic.

21 All of them have the quality of being
22 nonadaptive. They don't serve an external purpose,
23 they serve an internal purpose. They are dearousing.
24 I've read an article on this which I think I submitted
25 to the Court. So I see the abnormal behaviors of the

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1 autistic children again as dealing with this
2 overarousal state that I've already described.

3 I'm sure there are other examples, too, but
4 I think this gives the Court a flavor of what I take
5 to be the explanatory value of autistic behaviors of
6 the overarousal model and some rationale for why if a
7 child is victim of neuroinflammation, why it should
8 affect the brain in that way, autistic, other than in
9 whatever other way.

10 In other words, I have a continuous
11 mechanism of cause and effect formula. Whatever
12 provokes a microglial activation, including a metal,
13 to the glutamate excess, to the overarousal and to the
14 autistic behavior that results.

15 Q So, Dr. Kinsbourne, then in describing this
16 model it clearly is dependent on the existence of a
17 neuroinflammatory process, correct?

18 A Yes.

19 Q Are you aware of any peer-reviewed published
20 studies that evidence the presence of
21 neuroinflammation in autistic individuals?

22 A Yes.

23 Q Now, would that include the Lopez-Hurtado
24 article we discussed?

25 A Well, first of all, of course the Vargas

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1 article, which is fundamental to, I'm sure the Court
2 is aware of --

3 MR. POWERS: Let's hold on. If you're
4 mentioning that, let's have Scott put that up. It's
5 Petitioners' Master Reference 0069.

6 BY MR. POWERS:

7 Q Dr. Kinsbourne, you've got the exhibit on
8 the screen in front of you. Can you explain to the
9 Special Masters and for the record what you're looking
10 at?

11 A Yes, sir. The article is called *Neuroglial*
12 *Activation and Neuroinflammation in the Brains of*
13 *Patients with Autism*. Now, it actually mentions data
14 in two contexts. One is on autopsied brains of people
15 with autism who died, just like the Lopez-Hurtado
16 article that I already mentioned, and then also on the
17 cerebral spinal fluid of living autistic children.

18 Now, in brief, what they describe is, which
19 really was a surprising finding, a pioneering finding,
20 people haven't realized this, that the brains of
21 autistic individual are not as used to be thought
22 what's called an empty fortress, the shell of some
23 earlier damage and now they're left with a devastated
24 dwelling place, as it were, but rather, there's active
25 disease going on over time.

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1 Now, these were people who had been autistic
2 for a long time. It isn't as if it was just an
3 inflammation to begin, like an encephalitis, which
4 then caused structural problems. They are ongoing
5 process, must be an ongoing process, over years. They
6 found the microglial activation that I mentioned and a
7 certain amount of gliosis.

8 They didn't find much gliosis. It is the
9 other one who found that. They did find some problems
10 with neurons. The important point is they found
11 neuroinflammation in about two-thirds, three-quarters
12 of the small series of brains they looked at, so it
13 wasn't a rare phenomenon, actually, not in their
14 series.

15 MR. POWERS: And, Dr. Kinsbourne, let's
16 focus specifically on page 11 of this exhibit.

17 Scott, if you could pull that up?

18 BY MR. POWERS:

19 Q What I'm going to direct your attention to
20 is on the right-hand column there's a highlighted
21 section, and this is a column that's above a table.
22 In about the middle of that section of text there's a
23 highlighted sentence that begins in these conditions.
24 Could you take a look at that and explain the
25 significance to the Special Masters?

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1 A Yes.

2 MR. POWERS: Excuse me. Scott, I'll need
3 your help here. It actually continues on to the first
4 two lines of page 12.

5 THE WITNESS: Yes. Well, they say more
6 formally what I've just said, which is the microglial
7 activation is chronic, it goes on. It is responsible
8 for a sustained neuroinflammatory response. The
9 neuroinflammation goes on. In the course of that
10 neuroinflammation there's the production of what they
11 call multiple neurotoxic mediators.

12 We've discussed some of those, the cytokines
13 and reactive oxygen species that we've talked about.
14 Then what they say is -- well, you see above there
15 they talk of a number of different disorders,
16 including HIV, and they're saying that
17 neuroinflammatory activation may be a common pathway
18 leading to dysfunction in all these disorders, but we
19 haven't considered those.

20 In the case of autism, the presence of
21 microglial activation supports the view that innate
22 immune responses are present both in the cortex and in
23 the lower levels of the brain and that a state of
24 chronic activation and reactivity may be involved in
25 the mechanism of neuronal and synaptic dysfunction.

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1 I've just been describing to the Court a state of
2 chronic activation and reactivity.

3 BY MR. POWERS:

4 Q So this would actually be pathological
5 evidence that's consistent with your testimony and
6 your theory on causation and a model for affecting
7 regressive autism?

8 A I believe so.

9 MR. POWERS: You mentioned that there were a
10 couple of studies. This is one of them. I'm going to
11 ask Scott, if we could pull this one down, and we'll
12 take a look at what's been marked as Petitioners'
13 Master Reference 0072.

14 BY MR. POWERS:

15 Q That's now up on the screen, Dr. Kinsbourne.
16 Can you identify for the Special Masters and the
17 record what you're looking at?

18 A Yes, sir. The article is called *Immunity,*
19 *Neuroglia and Neuroinflammation in Autism.* The senior
20 author is Dr. Pardo who is the head of the laboratory
21 in which Dr. Vargas works. In other words, this is
22 the Johns Hopkins group that's been very productive in
23 this field.

24 Q And what's the significance of this article
25 to the expert opinion that you're offering in this

1 proceeding?

2 A Well, Dr. Pardo, this is 2005, he was
3 overlooking the findings mostly of his own laboratory.
4 I believe we have a passage marked later in the
5 article that distills the point that one wants to
6 make.

7 Q Well, let's go ahead and just jump to that
8 then. We'll be looking at page 5 of the exhibit, and
9 we're looking at the left-hand column. About a third
10 of the way down there's a highlighted section and the
11 first two words are "in normal." We have that up on
12 the screen now. Take a look at that, Dr. Kinsbourne.

13 A Yes. It starts by saying that normally
14 astrocytes help neurons to survive by certain
15 chemicals and by removing excitotoxic
16 neurotransmitters, namely glutamate as the main one.
17 There are some others, too. However, for an astroglia
18 activated because of injury, they, themselves can
19 produce factors that cause inflammation.

20 Then they list a number of types of
21 chemicals that may magnify immune reactions within the
22 CNS. Now, immune reactions are fine to be magnified
23 if they are contesting an invader, like a bacterium,
24 but these immune reactions are just firing at the
25 local vicinity.

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1 Q And in this firing, as you talked about
2 earlier in your testimony, is it being initiated and
3 sustained by the presence of that, in these cases you
4 believe, inorganic mercury that's in the brain, this
5 pounding away of the inflammatory response?

6 A Well, when you say these cases, I'm not
7 talking about individual cases. I'm saying that there
8 are a variety of persistent stimuli to the microglia,
9 any one type of which can elicit this reaction. Heavy
10 metals, including mercury, are among that list.

11 MR. POWERS: And then the last thing that
12 we'll talk about in this paper is further down, Scott.
13 If you can pull that highlight? It's that last
14 highlighted section, and we're looking at about three-
15 fourths of the way down the page on the left-hand
16 column of page 5.

17 SPECIAL MASTER CAMPBELL-SMITH: Beginning
18 with the words "changes in."

19 MR. POWERS: Yes, beginning with the words
20 "changes in."

21 THE WITNESS: Changes, right. So he's
22 saying that it's changes in the astroglia or microglia
23 that affect how the neurons work. So the attack that
24 they saw wasn't directly on the neurons, it was on
25 these helper cells, but they then secondarily affect

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1 the neurons and what the effect is the control of the
2 neuronal firing.

3 (Away from microphone.)

4 So he says in a more general way that
5 neuronal dysfunction, and I was describing a
6 particular neuronal dysfunction which was firing too
7 much, and that that -- disorganization as those seen
8 in autism may also be responsible for reactive vicious
9 cycle. They may then cause more neuroglial
10 activation.

11 In other words, as you were saying earlier,
12 it could be a vicious cycle.

13 BY MR. POWERS:

14 Q So is this passage also, do you believe,
15 consistent and supportive of your opinion that
16 neuroinflammation can lead to nervous system
17 dysfunction expressed as autism?

18 A Yes.

19 Q The last thing I wanted to discuss, and
20 briefly, Dr. Kinsbourne, is you've made several
21 references to the testimony yesterday of Dr. Richard
22 Deth.

23 A Yes.

24 Q And they've been scattered throughout your
25 testimony today. What I was hoping you might be able

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1 to do is explain to the Special Masters how you see
2 Dr. Deth's testimony about oxidative stress perhaps
3 interacting with the process that you're describing
4 here, which is the neuroinflammatory process in the
5 autistic brain.

6 A Right. In neuroscience, different
7 specialists study the brain at different levels of
8 organization all the way down from behavior, which is
9 the highest level of organization for the individual,
10 to molecular changes, which is the lowest level. High
11 and low, it's just descriptive. It doesn't mean
12 better or worse.

13 Dr. Deth works at the molecular level. At
14 that level, he gave a very complete account of
15 oxidative stress, and how it affects the workings of
16 the cell and what the mediators are because in the
17 nervous system it isn't just a one on one situation,
18 it tends to be a cascade, A causes B, and B causes C
19 and so on.

20 He gave us elegant illustrations of that. I
21 am discussing at the systems level. I'm discussing at
22 the level of the cells in the brain. What he says
23 speaks to particular points in that system, the effect
24 of the oxidative stress that I discussed as being
25 caused by the microglial activation. Well, he really

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1 explains that.

2 I mentioned it to say it damages cells. He
3 tells you how, what goes on. We have in common that
4 both of us are explaining changes to the brain which
5 are not killing cells. We both talk about functional
6 changes. Now, when there is an acute invasion of the
7 brain by viruses or an acute mercury-toxicity, it
8 doesn't stop at changing function. A lot of cells
9 simply die. That's a different situation.

10 Both of us were discussing how cells will
11 keep on working but working inefficiently and in a
12 deviant fashion, and I was discussing the consequences
13 for behavior of them doing that.

14 Q So, Dr. Kinsbourne, then would it be your
15 testimony to a reasonable degree of medical certainty
16 that thimerosal-containing vaccines belong on the list
17 of potential environmental factors to consider if one
18 is looking at what might have caused regressive autism
19 in any particular child? Would that be your
20 testimony?

21 A Yes. To expand, there are two steps to
22 this. One is that mercury certainly should be on that
23 list regardless of where it comes from. The second is
24 that thimerosal is a known quantifiable source of
25 mercury, and clearly, in the differential one has to

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1 consider whether that particular source of mercury is
2 damaging in the particular case.

3 Q And that is testimony that you would leave
4 to somebody who is evaluating that individual case and
5 using the testimony that you've provided here to make
6 that differential diagnosis?

7 A Right. I would see this as a conjoined
8 effort by a neurologist and a toxicologist.

9 Q And perhaps by a treating physician?

10 A Well, necessarily, yes.

11 MR. POWERS: All right. Thank you. No
12 further questions at this point.

13 SPECIAL MASTER CAMPBELL-SMITH: Thank you.
14 We've been going a little over two hours. It looks
15 like an appropriate time to take a break before we get
16 to cross. Just a couple of things to clear up on the
17 record just to make the record clear. I had made
18 notes. Thank you, George, you kind of stopped us.

19 If we could go back and pull back up the
20 Lopez-Hurtado article from PML 446? There were two
21 references that Dr. Kinsbourne was interpreting. The
22 first was at page 11 of that document. I did write
23 this down to memorialize this for the record, and I'd
24 like to do so now.

25 I believe the first one was on the left

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1 column at just nearly the bottom beginning with the
2 words since thickness and extending over to the right-
3 hand column. I believe the second reference was a
4 little further down on that page about halfway through
5 that began with a toxic etiology. Those were the
6 references that I had from the Lopez-Hurtado article.

7 SPECIAL MASTER HASTINGS: And that was the
8 whole paragraph.

9 SPECIAL MASTER CAMPBELL-SMITH: That's
10 correct, and that was that whole paragraph there. The
11 next reference, just for clarification, the article to
12 which Dr. Kinsbourne was referring regarding the heart
13 rate studies was the Goodman article which was
14 referenced in his opinion as PML 496.

15 I believe those were the references that had
16 not been clearly identified in the record, but just to
17 do so, I wanted to take that moment. I think with
18 this being our morning break and for ease of reference
19 if we were to do 15 minutes that would put us at
20 11:25. Will that work with your watch, Mr. Powers, or
21 shall we push a little further?

22 MR. POWERS: That would work just fine.

23 SPECIAL MASTER CAMPBELL-SMITH: Okay. How
24 about you, Mr. Matanoski? Would that give you
25 adequate time to prepare your cross or would you like

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1 the additional five minutes?

2 MR. MATANOSKI: No, ma'am, that should be
3 adequate time.

4 SPECIAL MASTER CAMPBELL-SMITH: Okay. Well,
5 we are in recess for the next 15 minutes. Let's
6 convene again at 11:25. Thank you.

7 (Whereupon, a short recess was taken.)

8 SPECIAL MASTER CAMPBELL-SMITH: We are back
9 on the record. While we're waiting for Respondent's
10 wingman to join him at counsel table, just a reminder
11 that as the CSO security officer indicated, for those
12 of you who are that side of the bench, we're going to
13 have to ask you and remind you to turn off all your
14 devices including your Blackberries, because the
15 click, click that we heard apparently during Dr.
16 Kinsbourne's earlier testimony was related to the
17 interference from the Blackberries.

18 Apparently, it's been a busy morning with
19 your Blackberries. So we are going to have to ask you
20 to turn those off. Dr. Kinsbourne, if you would join
21 us back in the witness stand. Respondent to commence
22 cross.

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

24 CROSS-EXAMINATION

25 BY MR. MATANOSKI:

Heritage Reporting Corporation
(202) 628-4888

1 Q Good morning, Doctor. I'm Vince Matanoski
2 for the United States.

3 A Yes, sir.

4 Q Doctor, Mr. Powers informed us I believe in
5 March that you had come to him to offer your services
6 in rendering an opinion in this case. Does that
7 comport with your recollection?

8 A No. I had come to him to offer my services?

9 Q Yes, sir.

10 A No. I don't go to lawyers and offer my
11 services.

12 Q I see. When did you first become involved
13 in this case?

14 A Probably about in March. I don't remember
15 exactly.

16 Q I'm sorry?

17 A A short time ago. I don't remember exactly.
18 Shortly before I wrote my report, whatever the date of
19 that was. I don't remember.

20 Q I see. What is your definition of
21 regressive autism?

22 A Nobody has a definition of regressive
23 autism. We do have descriptions of what the
24 phenomenon is, and the phenomenon is a child
25 developing at a certain rate, whether fast or slow,

1 and then actually losing skills that he or she had
2 already attained and progressively losing them in such
3 a fashion that they end up in an autistic state.

4 Q That's your definition of regressive autism,
5 is it?

6 A It's my description of what I understand as
7 being regressive autism. There haven't been to my
8 knowledge very consensual formal definitions.

9 Q What are the diagnostic criteria for
10 regressive autism?

11 A They are descriptive. They are that the
12 child having attained a certain observable milestones
13 over a period of weeks or a few months gradually loses
14 them and begins to behave in ways recognized as being
15 autistic.

16 Q And that is the description of regressive
17 autism that I could find in DSM-IV?

18 A I couldn't tell you.

19 Q So you don't know what the description is in
20 DSM-IV?

21 A No.

22 Q Do you know what the description of
23 regressive autism is in ICD-10?

24 A No, I don't.

25 Q Is that because you haven't been treating

1 children in this area for quite some time?

2 A No. It's because I haven't looked it up.

3 Q You said that you were preparing and
4 reviewing the literature to prepare your chapter for
5 *Menkes*, the next edition on developmental disorders,
6 is that correct?

7 A I'm beginning to collect some articles, yes.

8 Q And in that chapter you will be discussing
9 autism?

10 A Yes. It's part of my responsibility to have
11 a section on autism.

12 Q And you haven't reviewed the diagnostic
13 criteria currently for the definition?

14 A My chapter doesn't go into the diagnostic
15 criteria.

16 Q In reviewing this literature and preparation
17 for your chapter have you found the term clearly
18 regressive autism as a distinct disease category
19 within the description of autistic disorders?

20 A Dr. Rutter mentions something along these
21 lines. As I was explaining to the Court, there are
22 some cases that are marginal or subtle and in some
23 cases that are clear.

24 Q In reviewing the literature in preparation
25 have you seen that described, that term --

1 A I don't recall. I may have seen it.

2 Q You don't recall?

3 A No.

4 Q What is a differential diagnosis?

5 A Of regressive autism or what is a
6 differential diagnosis?

7 Q Yes.

8 A Right. When one is confronted by an
9 individual with a particular problem, be it physical,
10 or mental, or both, it is almost always the case that
11 there's more than one possible cause of the
12 appearances that you see.

13 One then lists to the best of one's ability
14 the various disease processes that could lead to that
15 outcome and to the best of available investigative
16 capability tries to find out which one of them it is
17 ruling out the others.

18 Q Would you then proceed from a list of known
19 or suspected causes of a disease and then proceed from
20 that list?

21 A I would look for a list of what I suspect to
22 be possible causes of the disease.

23 Q And in preparing your book chapter in
24 *Menkes*, Volume 6, you prepared such a list of known
25 associations with autism, did you not?

1 A There was quite a long list, yes.

2 Q So if I were a doctor and I was trying to
3 make a differential diagnosis, I would go down that
4 list to try to find a cause, is that right, that I
5 could do that to make a differential diagnosis?

6 A Certainly you could do that. I mean, even
7 my long list isn't exhaustive, but, yes.

8 Q And if I went down that list and I scratched
9 off every single association that's on that list and I
10 had none left, as a matter of differential diagnosis,
11 what would I conclude?

12 A That I didn't know what caused it.

13 Q Will you review your chapter in *Menkes*,
14 Volume 6, the chart of known associations, I believe
15 it is conditions associated with autism, and tell me
16 if thimerosal-containing vaccines are on that list?

17 A Well, no.

18 Q Now, you prepared a similar list when you
19 testified in this Cedillo case, correct?

20 A I think so, yes.

21 Q And it appeared in your expert report in
22 that case?

23 A I'm sure, yeah.

24 Q And when you prepared at that time you added
25 measles virus to the associations with autism.

1 Otherwise, that list was virtually the same as what
2 appeared in the *Menkes* chapter that we've been
3 discussing.

4 A Correct.

5 Q Did that list include thimerosal-containing
6 vaccines?

7 A No.

8 Q To the best of your knowledge, what
9 percentage of cases are there conditions associated
10 with autism?

11 A In what percentage are there known
12 conditions? That's what you're asking me?

13 Q I should rephrase it.

14 A Yeah.

15 Q Amongst cases that are eventually diagnosed
16 as autism, in what percentage of those cases is there
17 an association with some factor?

18 A Is there a known association? About 10 to
19 20 percent.

20 Q So in the remaining 90 to 80 percent, after
21 going through a differential diagnosis, one is left
22 with an unknown cause, is that right?

23 A Yes.

24 Q Now, in your expert report on page 9 at the
25 very bottom you stated in many cases, however, there

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1 is no viable alternative diagnostic option other than
2 the involvement of a postnatal environmental insult or
3 exposure.

4 SPECIAL MASTER CAMPBELL-SMITH: Excuse me,
5 Mr. Matanoski. You need to give us the particular
6 cite reference.

7 MR. MATANOSKI: I'm sorry. I believe the
8 report itself is Petitioners' Exhibit 30.

9 SPECIAL MASTER CAMPBELL-SMITH: Petitioners'
10 Exhibit 30.

11 MR. MATANOSKI: And it was at the bottom of
12 page 9 of that report.

13 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

14 MR. MATANOSKI: Are you suggesting, doctor,
15 that when you exhausted your list of associations that
16 one must then conclude that in cases of regressive
17 autism at least that it must be an postnatal
18 environmental insult or exposure?

19 THE WITNESS: No.

20 BY MR. MATANOSKI:

21 Q Now, in your report you've hypothesized a
22 genetic vulnerability that could lead to regressive
23 autism, is that correct?

24 A Yes.

25 Q And this morning you described it as a

1 susceptibility, but I imagine the terms are meant the
2 same way?

3 A Correct.

4 Q Could you describe what that vulnerability
5 is?

6 A The vulnerability is that under
7 circumstances in which a particular event occurs that
8 is harmless to most people, that event, however, is
9 harmful to a subset of the population who are regarded
10 as susceptible.

11 Q Could you tell me what that susceptibility
12 is to? What event, what particular characteristic in
13 the individual creates the vulnerability?

14 A There are innumerable vulnerabilities that
15 have been presumed to be genetic in origin but most of
16 them have not been identified in terms of particular
17 genes or gene resemblance.

18 Q We've heard Dr. Deth describe it as a
19 lowered state of glutathione, and he has identified
20 that. Can you describe for me what in your hypothesis
21 is the particular susceptibility?

22 A I cannot give you -- the neurobiological
23 basis for why most people under certain circumstances
24 don't become autistic and some do is not known.

25 Q I'm talking about your hypothesis that

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1 exposure to TCVs, thimerosal-containing vaccines, will
2 in a susceptible population lead to regressive autism.
3 I'm wondering what the particular susceptibility is.
4 Is it to an allergic response? Is it that they have a
5 lower threshold? Can you describe what it is in your
6 hypothesis?

7 A I'm afraid I can't help you. The fact is
8 that when the great majority of children received the
9 usual vaccination schedule at a time when thimerosal
10 was used they all received more or less the same
11 amounts and most of them, as far as we can tell, were
12 unscathed and some of them perhaps were not.

13 We know in general that a certain amount of
14 mercury challenge may affect some people much more
15 adversely than other people and that the medicine is
16 pervasive. It happens in all aspects of disease that
17 some people are more susceptible than others to the
18 same challenge.

19 Now, in hardly any of these situations do we
20 actually know the neurobiological basis, and here is
21 one of those cases that we don't know the
22 neurobiological basis.

23 Q Dr. Aposhian hypothesized that it was an
24 efflux disorder is what made these individuals
25 susceptible. Dr. Deth was hypothesizing that it was a

1 glutathione or a sulfur metabolism process, an
2 abnormality in that, that made these individuals
3 susceptible. In your theory, in your hypothesis that
4 you've laid out, you have neuroinflammation, you have
5 glutamate, you have hyperarousal.

6 Can you describe what it is that you think,
7 and I'm not asking you to tell me what genes are
8 involved, but what is the particular susceptibility or
9 vulnerability that these individuals have in your
10 hypothesis?

11 A As I've said, we don't know. I mean, we
12 could ask, for example, whether the innate immune
13 system responds more vigorously in some people than
14 others for reasons that may be genetically determined,
15 but that would be speculation. I don't know.

16 Q So you haven't worked out what it is that
17 you think the vulnerability is?

18 A No, I haven't worked that out.

19 Q Now, I'm going to try to ask a series of
20 questions that will follow through what is happening
21 with the thimerosal that one receives from the
22 vaccination, ultimately where it ends up in the body,
23 brain, at least as far as your hypothesis of what's
24 happening.

25 I'll try to skip us forward because I know

1 we've had testimony already on what happens to
2 thimerosal and its change that occurs and move us
3 right to after a vaccination of a thimerosal-
4 containing vaccine when will the ethyl mercury enter
5 the brain? How long after the vaccination?

6 A It is likely to enter the brain while it is
7 still detectable in the blood, so it will perhaps be a
8 few weeks.

9 Q You believe it's detectable in the blood for
10 a few weeks?

11 A Yeah.

12 Q Do you know with any further precision how
13 long it's detectable in the blood?

14 A No. It doesn't really help me with my
15 hypothesis to know it with further precision.

16 Q How much of the ethyl mercury will enter the
17 brain let's say the first day after vaccination?

18 A That is a question for a toxicologist.

19 Q Is it important to you how much comes into
20 the brain at any given time?

21 A It is important for me to be told by a
22 toxicologist whether enough has entered the brain to
23 elicit the inflammatory action that I am talking
24 about.

25 Q When it first enters the brain, where will

1 it go in the brain?

2 A It's been found in the cerebral cortex, in
3 the cerebellum and in the amygdala. There are a
4 number of structures. It's fairly widespread.

5 Q This is the ethyl mercury now?

6 A I think that whether it's ethyl or methyl
7 doesn't make a great difference in where it goes.

8 Q So you mentioned the cerebral cortex. Where
9 else?

10 A Cerebellum.

11 Q Where else?

12 A And certain brainstem nuclei.

13 Q Anywhere else?

14 A Not to my knowledge.

15 Q And this is both, I should say it's ethyl,
16 methyl, and is it inorganic as well?

17 A Yeah.

18 Q Will the ethyl mercury go to all areas of
19 the brain?

20 A Well, I don't think anybody has ever looked
21 at all areas of the brain. I think that it's fairly
22 widely distributed when we are dealing with low-dose
23 administrations of any mercury-containing product that
24 there's not the same distribution as when one is
25 dealing with an acute mercury overdose.

1 So in an acute mercury overdose one is going
2 to find foci of structural destruction in particular
3 cell populations, but that will not be expected with
4 low-dose mercury burdens.

5 Q Will there be different areas of the brain
6 affected in the acute high-dose exposure versus the
7 chronic low-dose that you were just discussing?

8 A Well, the cerebellum will be one area that
9 is particularly affected with the high dose, but with
10 a high dose the cerebral cortex is affected.

11 Q Do you consider immunization with a
12 thimerosal-containing vaccine to be an acute high dose
13 or a chronic low dose?

14 A A low dose. Absolutely.

15 Q And in what way is it chronic?

16 A In the way discussed by the toxicologists,
17 which is that once the ethyl mercury enters the brain
18 it is degraded to ionic mercury, HD++, which then is
19 chronically present.

20 Q Will methyl mercury go to the same or
21 different areas of the brain?

22 A I didn't see much evidence of difference.

23 Q After it moves to these areas of the brain
24 that you've described, what happens next?

25 A Over a period of time the deethylation or

1 methylation, whatever the agent is, continues until
2 the bulk of the mercury is in metallic form, mercuric
3 form, and then it stays there and may or may not
4 elicit the inflammatory actions that I've discussed.

5 Q How long does that take?

6 A It could be a matter of a few weeks, or
7 months, or longer.

8 Q And for ethyl mercury from the vaccine, how
9 long will it take for it to go through the same
10 process?

11 A For it to?

12 Q For it to go through the same dealkylation
13 process you've described?

14 A It's a progressive matter lasting a number
15 of months.

16 Q A number of months?

17 A Uh-huh.

18 Q For ethyl mercury?

19 A Yes.

20 Q You said that the importance of the timing
21 was that enough ethyl mercury get into the brain to
22 cause the process that you've described.

23 A Yes.

24 Q How much ethyl mercury is necessary for that
25 process?

1 A That you must ask a toxicologist.

2 Q You don't know how much ethyl mercury would
3 be necessary to cause neuroinflammation?

4 A No. That's not in my field of expertise.
5 One would ask a toxicologist to determine that.

6 Q You've rendered an opinion that the amount
7 in childhood-containing vaccines is enough to cause
8 neuroinflammation that will lead to regressive autism,
9 but you don't know how much?

10 A You are misstating my opinion.

11 Q Correct me.

12 A Yes. I've rendered an opinion that mercury,
13 in sufficient amounts, will set up the process of
14 neuroinflammation with its consequences. I've
15 rendered the opinion that it will do that regardless
16 of the vehicle in which it comes, whether it's mercury
17 vapor, or whether it's a vaccination, or whether it's
18 fish, anything.

19 I have not rendered the opinion that the
20 mercury in thimerosal is sufficient to meet such a
21 threshold because it is up to the toxicologist to
22 define that threshold. So my opinion has been that if
23 toxicology tells us that there is enough mercury in
24 the vaccines to cause inflammation in the brains of
25 children, then the process which I have discussed is

1 initiated.

2 Q When you wrote this report what were you
3 relying on then to determine that there would be
4 enough in childhood vaccines to cause
5 neuroinflammation?

6 A Could you point me to the place where I say
7 that there would be enough?

8 Q I'm sorry, sir. The thrust of your report
9 is in fact your conclusion is that childhood
10 thimerosal-containing vaccines will cause regressive
11 autism in a susceptible population, and you said you'd
12 need a toxicologist to tell you how much that is.

13 How can you reach that conclusion without
14 knowing that there's a certain amount that would reach
15 a neuroinflammatory state sufficient to elicit the
16 hyperarousal condition that you've hypothesized?

17 A I'm afraid I'm perplexed by your question.
18 Could you point me to the line in my report at which
19 you're referring?

20 Q Doctor, you've rendered a conclusion, you've
21 rendered an opinion that thimerosal-containing
22 vaccines will cause regressive autism in a susceptible
23 population. I'm trying to find out what you're
24 relying on to say that the amount of mercury in
25 thimerosal-containing vaccines is enough.

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1 You've turned me to toxicologists. When you
2 wrote your report what were you looking at to render
3 that opinion? You've rendered an opinion at that
4 time. What were you looking at?

5 A You keep telling me that I rendered a
6 particular opinion. I would like you to point me to
7 the form of words I used to render that opinion.

8 SPECIAL MASTER HASTINGS: Perhaps I can
9 intervene here. Let's see if I understood what you
10 said, Dr. Kinsbourne, in response to Mr. Matanoski's
11 earlier question. Pardon me with a scratchy throat
12 here. I think if I understood you correctly just now
13 you're saying you haven't given an opinion that the
14 thimerosal component in thimerosal-containing vaccines
15 can cause autism.

16 You're saying your opinion is strictly that
17 mercury in the brain could cause autism by the
18 mechanism that you describe. That's all your opinion
19 is.

20 THE WITNESS: Right, and that if toxicology
21 tells us that there is enough mercury in the vaccines
22 to set up that reaction, then I'll add that to my
23 opinion.

24 SPECIAL MASTER HASTINGS: But you don't
25 claim to know whether the amount in thimerosal-

1 containing vaccine is enough to do that. You don't
2 claim to answer that question.

3 THE WITNESS: Absolutely not. I'm a
4 neurologist. It's not the kind of thing that I would
5 know.

6 SPECIAL MASTER HASTINGS: Okay. I'll turn
7 it back to you, Mr. Matanoski.

8 MR. MATANOSKI: Thank you.

9 BY MR. MATANOSKI:

10 Q How much mercury then is necessary to create
11 astrocytic death and microglial activation?

12 A I don't know.

13 Q So you don't know whether the amount in
14 childhood vaccines would be enough to do that?

15 A As I just said in response of the Court,
16 that I don't know and I don't regard it as in my
17 professional domain to know that. This is something
18 which I consult the appropriate specialist who is a
19 toxicologist.

20 Q And in writing your report you hadn't
21 consulted any such toxicologist, correct?

22 A I have not, and I did not render the
23 opinion.

24 Q Before you wrote your report you were able
25 to read Dr. Aposhian's expert report, correct?

1 A Yes.

2 Q And Dr. Aposhian told us on I believe it was
3 Monday or Tuesday that he had not spoken to you since
4 the Cedillo trial, is that right?

5 A He had not spoken to me?

6 Q Correct.

7 A Yeah, that's correct.

8 Q So in that regard, when you rendered this
9 opinion the toxicological advice that you had been
10 given as to how this mechanism would work was based on
11 your reading of Dr. Aposhian's expert report, is that
12 right?

13 A It was, but I had also read some literature,
14 which I referenced, which made me believe that
15 toxicology can in fact determine whether the amounts
16 in vaccines are enough to put the child at risk for
17 inflammation, and I'm referring particularly to the
18 Burbacher articles which I read independently of Dr.
19 Aposhian's report.

20 Q I'm sorry, which articles?

21 A The work with the macaque monkeys who were
22 administered a series of thimerosal vaccinations
23 stated to be analogous to what human infants receive.

24 SPECIAL MASTER VOWELL: Did you say
25 Burbacher articles?

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1 THE WITNESS: Yes.

2 SPECIAL MASTER VOWELL: Okay.

3 THE WITNESS: Yes.

4 BY MR. MATANOSKI:

5 Q So that would include the Burbacher article
6 and the Charleston articles that were discussed in
7 your report?

8 A Right. Burbacher is the head of the group,
9 but, yes, there were the charts and articles 1994
10 through 1996, and the more recent one was the
11 Burbacher.

12 Q And you read those and interpreted them
13 independently of Dr. Aposhian or the involvement of a
14 toxicologist?

15 A Yes. I have not discussed them with him.

16 Q So what you wrote in your report is your own
17 view of what those articles mean and what you make of
18 them?

19 A It's what I saw written there, yes.

20 Q By written there, you meant in the articles?

21 A What I saw in the article. I read the
22 articles and I commented on them.

23 Q So you don't know whether one vaccination is
24 enough to cause the astrocyte damage that you've
25 described?

1 A No. As I've said, the quantitative aspect
2 -- to restate this, obviously a single atom of mercury
3 is not enough to make a child sick. There must be a
4 certain amount to put at least some children at risk,
5 though probably that amount won't put most at risk and
6 then a greater amount will finally put everybody at
7 risk.

8 Now, as a neurologist, I start at the point
9 the toxicologist leaves off. He or she tells me
10 whether a particular product administration,
11 inhalation, ingestion, is likely to have delivered
12 enough mercury to the brain to set up the process that
13 I talk about, and I pick it up from there and describe
14 the process and its consequences.

15 So what I was expressing in my report was
16 that given that thimerosal contains mercury, we have
17 to consider it as a potential cause of the
18 neuroinflammation I discussed because we know for sure
19 that mercury can cause neuroinflammation.

20 However, it's not possible for me to make a
21 final decision on this because I need to hear from a
22 toxicologist whether the evidence that exists that the
23 amount of mercury in thimerosal is sufficient to cause
24 inflammation is reliable and evidence that I should
25 accept for my own working purposes.

1 Q In your report you stated on page 24 for the
2 reasons summarized in this report, it is my opinion to
3 a reasonable degree of medical probability that a
4 series of TCVs can result in or contribute to an
5 accumulation of HG++ in the brain. The mercury in the
6 brain may trigger an inflammatory response in some
7 children that results in a hyperglutamatergic state,
8 and so on.

9 So are you saying that you don't hold the
10 opinion to a reasonable degree of medical certainty
11 that the series of TCVs can result and accumulate in
12 this --

13 A As I say, I think they can depending on
14 whether there's enough mercury that enters the brain
15 from that vaccination, and I'm not the person to
16 decide, to advise the Court on that point.

17 Q So whether that would or not be enough
18 depends entirely on the testimony of the toxicologist?

19 A Yes. In other words, in my differential
20 diagnosis I would consult a toxicologist. Basically
21 I'm learning that here's a reaction that mercury could
22 cause, I look around for a source of mercury as part
23 of my differential diagnosis, guess what, the child
24 received mercury by a vaccination route.

25 So now I would like to know did that child

1 receive enough mercury to account for this reaction
2 and the toxicologist would tell me what the evidence
3 is that it was or was not enough.

4 Q And you have no idea what that amount is?

5 A I have no idea about what?

6 Q What the amount of mercury is necessary to
7 cause the response that you've hypothesized?

8 A Well, I certainly wouldn't memorize the
9 milligrams or micrograms involved.

10 I have already referred to the Charleston
11 articles which have shown in adult macaque monkeys
12 which were studied as analogous, their brains being
13 analogous to that of humans, that what they say was
14 the amount of, were delivered mercury in amounts
15 comparable to that which children used to get, that
16 those monkeys had measurable mercury in their brains,
17 enough to cause inflammation.

18 Now, that suggests that it's worth taking
19 seriously the question of whether thimerosal-
20 containing vaccines in fact could do this with humans,
21 too. Now, more recently this issue was studied even
22 more directly by doing a similar experiment with
23 infant monkeys.

24 Again, the amount of mercury in the brain
25 was measured. I don't know the amount but I know that

1 it was about one-fifth of the amount that had been
2 found in the adults. Now I understand we're waiting
3 to find out whether in the infant monkeys that amount
4 was sufficient to cause inflammation.

5 Q Now, your understanding that you just were
6 referring to, the 2005 Burbacher article?

7 A Yes.

8 Q And your understanding of the earlier work
9 by that group with the adult macaques, that the amount
10 of mercury used in that experiment simulated that
11 received by childhood vaccinations during the period?

12 A They claim that it did.

13 Q You have no idea how much mercury it will
14 take to cause an astrocyte to die?

15 A It wouldn't help me in my opinion.

16 Q It wouldn't? I thought your opinion was
17 that the mercury was having an affect on astrocytes
18 and microglia. So it doesn't matter how much mercury
19 there is as to whether it could provoke that reaction?

20 A What matters to me is that a toxicologist
21 tells me that there was or wasn't enough to do that.
22 It's not my specialty to figure that out.

23 Q Now, when you wrote this report, what
24 toxicologist had told you that there would be enough
25 for any value to cause the reaction that you've

1 hypothesized?

2 A Could you ask that again, please?

3 Q When you wrote your report, what
4 toxicologist had told you there was any mercury value,
5 any mercury dose, that could cause the condition that
6 you've hypothesized of astrocyte death and microglial
7 activation?

8 A Well, I mean, Dr. Aschner has reported very
9 clearly that mercury in the brain can cause microglial
10 activation, inhibition of glutamate reactate and so
11 on. I think he's a pretty reliable toxicologist.

12 Q Did you consult with him?

13 A Huh? No, I read his article.

14 Q And you were relying on his reports of the
15 toxicology of mercury, correct?

16 A It was his report of the neurobiology.

17 Q Of methyl mercury, is that correct?

18 A In the article that I mentioned this morning
19 it was methyl mercury, yes.

20 Q And you described that as a neurobiological
21 article. Is it a neurotoxicological article?

22 A Well, it's both because he looked at the
23 system, which is, you know, the cell, and he
24 quantitated a toxin. So it really overlaps those two
25 fields.

1 Q And you described Dr. Aschner as a
2 neurotoxicologist?

3 A Yes.

4 Q And as a pediatric neurologist, this is your
5 interpretation of his work?

6 A My interpretation of what he says is what he
7 says. Once I know that mercury at some super
8 threshold amount can unleash a neuroinflammatory
9 process which has much in common with that which has
10 been found in autistic children independently by other
11 research groups, then I have to consider mercury as
12 one of the multiple possible causes of that
13 neuroinflammation.

14 Now, in principle that must be the case, and
15 the inference then is that the various delivery
16 systems for mercury should be scanned for whether any
17 of them might in fact have delivered enough mercury to
18 do this. Now, to answer that question I need a
19 toxicologist. The toxicologist will then refer to
20 evidence, such as the Burbacher papers, in framing his
21 or her response.

22 Q And you felt free within your level of
23 expertise to discuss the importance of the Burbacher
24 papers, the Charleston papers, et cetera, on
25 toxicological issues as to how they impacted on your

1 opinion of a mechanism by which thimerosal-containing
2 vaccines could cause autism?

3 A Well, obviously I will free to discuss any
4 article I want, but what I didn't do, actually, is
5 discuss it. I basically cited their conclusion. Now,
6 a toxicologist might wish to discuss in a more
7 specialized way whether he agrees, or doesn't agree,
8 or what other factors might be considered.

9 I put down conclusions but I leave it to the
10 toxicologist to determine whether this particular
11 source of evidence will enable them to tell me whether
12 the vaccines contain enough mercury. That's not my
13 role.

14 Q Did you or did you not extrapolate from
15 those articles to a conclusion that mercury would
16 create astrocytic death and microglial activation
17 resulting in neuroinflammation and a
18 hypergammatalurgic response?

19 A Well, I concluded that from numerous
20 articles.

21 Q You extrapolated that from numerous
22 articles.

23 A I didn't extrapolate it. There are a whole
24 variety of studies that show that mercury is capable
25 of doing that. I don't think that's really what's

1 contentious. What's contentious is whether a
2 particular form of administering mercury contains
3 enough mercury to do that.

4 Q The Charleston article was about methyl
5 mercury in adult macaques and you extrapolated that to
6 an affect in humans, correct?

7 A Probably, but perhaps you could say it
8 again.

9 Q The Charleston articles were about methyl
10 mercury in adult macaques and you extrapolated that to
11 an affect on humans, correct?

12 A Not entirely. I'll say it again. I'm
13 pointing out that there is evidence available to a
14 toxicologist who can evaluate the usefulness of this
15 particular animal model and determine from that data,
16 and maybe other data, that he has available, that I
17 don't know whether in fact this will help him
18 determine how much mercury is required to cause the
19 neuroinflammation and whether what's in the vaccines
20 is that much.

21 Q And when you wrote your report you did not
22 have that evidence from a toxicologist, correct?

23 A No, I didn't.

24 Q Do you know how methyl mercury enters
25 astrocytes?

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1 A I don't recall.

2 Q Is the same mechanism available to ethyl
3 mercury?

4 A Yes.

5 Q But you don't recall what that mechanism is?

6 A No. Not in my regular detail, no.

7 Q Can you tell me then the detail that you do
8 recall?

9 A No, I'm not able to tell you how methyl
10 mercury enters astrocytes.

11 Q How about inorganic mercury? How would that
12 enter astrocytes?

13 A I would be speculating.

14 Q So you don't know?

15 A What I know is that these substances are
16 found in astrocytes and microglia, which is what I
17 need to know for my analysis of the situation.

18 Q Once mercury kills an astrocyte where does
19 the mercury go?

20 A After it's?

21 Q If it kills an astrocyte where does it go?

22 A Well, it might go into what one might call
23 the debris of the astrocyte. In the Lopez-Hurtado
24 paper they refer to lipofuscin which accumulates in
25 these dying cells. Lipofuscin is a conglomerate of

1 lipids and proteins which are the inactivated products
2 of cell contents and are known to contain metals, if
3 metals were around, so it might be stuck in a cluster
4 of that nature.

5 Q So it may be stuck in a cluster of that
6 nature, is that what you said?

7 A Yes.

8 Q And you were talking about Lopez-Hurtado.
9 Did that article deal with mercury in any way?

10 A It refers to heavy metals, yes.

11 Q What, if anything, happens to neurons during
12 this process?

13 A During this process neurons will, as I
14 explained, be, as it were, flooded with excess
15 glutamate and fire more than is usual for them.

16 Q Will anything else happen to them?

17 A It's a matter of degree. If that excessive
18 firing reaches a certain limit, then the person will
19 show epileptic form or epileptic manifestations. They
20 may have seizures because the neurons were firing
21 excessively.

22 So one of the consequences of an excitotoxic
23 challenge over and above the arousal that I've already
24 mentioned is that seizures be triggered, which of
25 course is a common phenomenon in people with autism.

1 Q Are you implying that seizures in autism are
2 caused by mercury?

3 A I'm trying to think of another way of
4 telling you which works better than the way I've tried
5 to. If you have neuroinflammation for whatever cause
6 and there are markable ones, mercury is just one, then
7 you get into a state in which, among other things, you
8 are more liable than normal to have seizures.

9 Q In this hypothesis that you have of
10 astrocytic death, microglial activation, what would
11 the neuropathological findings be?

12 A The kind of findings reported by the Vargas
13 group which would be sweating of the microglia, and a
14 certain amount of edema and the presence of
15 proinflammatory cytokines, which that group in fact
16 reports. If the process have been of a very long
17 standing you might also find the gliosis that Lopez-
18 Hurtado reports.

19 Q Why would you find gliosis?

20 A Because astrocytes will have died.

21 Q Isn't gliosis an overgrowth of astrocytes in
22 the area of damage to the brain or the spinal cord?

23 A It is an overgrowth which leads to a
24 scarring, just like a scar on the skin is an
25 overgrowth of connective tissue cells which is inert.

1 So is gliosis.

2 Q So if you see gliosis you actually have an
3 excess of astrocytes, correct?

4 A You have an excess of astrocytes in reaction
5 to the challenge, but then those astrocytes can die
6 leaving, as it were, the appearance of scars in the
7 brain.

8 Q Are you saying that gliosis is both an
9 overgrowth and a decrease in astrocytes?

10 A It is like a scar. It is both an overgrowth
11 and a neutralization of function of the structures.
12 In gliosis, astrocytes do not perform their function.
13 It's an abnormal collection due to a disease process.

14 Q And your mechanism relies on astrocytes not
15 being available because they have been knocked off as
16 innocent bystanders, friendly fire, from the microglia
17 so they're not available to mop up excess glutamate,
18 isn't that right?

19 A Not exactly. My model relies on the well-
20 demonstrated effect of microglial activation in
21 inhibiting glutamate transport into the astrocytes and
22 its also documented tendency to have glutamate exit
23 from the astrocytes. In other words, the astrocyte no
24 longer fulfills its function in controlling the flow
25 of glutamates. Doesn't have to be dead.

1 Q Among the various papers you cited, do any
2 show methyl mercury causes Purkinje cell loss?

3 A I think that methyl mercury is likely to
4 cause granular cell loss but that's from situations
5 where the dose was very much higher than we're
6 discussing.

7 Q And ethyl mercury, would that cause Purkinje
8 cell loss?

9 A I know that ethyl mercury does not
10 particularly cause granular cell loss. I believe it
11 may cause some loss of Purkinje cells, yes. But, you
12 see, what's causing it, in my opinion, isn't the
13 ethyl, it's the inorganic mercury.

14 Q So then, I believe you just answered my next
15 question, you'd expect to see Purkinje cell loss with
16 inorganic mercury?

17 A I really can't hear.

18 Q Did you say you expected to see Purkinje
19 cell loss with inorganic mercury?

20 A I would expect inorganic mercury to set up
21 the excitotoxicity potential that I've described, and,
22 if there is excitotoxicity, the Purkinje cells are a
23 very likely target because they're more vulnerable to
24 excitotoxicity than most other cell types in the
25 brain.

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1 Q Did you know that the papers that you cited
2 with respect to Dr. Aschner stated that Purkinje cells
3 were not damaged in methyl mercury poisoning?

4 A I am not discussing what happens in methyl
5 mercury poisoning.

6 Q Do you agree with Dr. Aposhian that methyl
7 mercury will convert to inorganic mercury in the
8 brain?

9 A Absolutely.

10 Q So after exposure to methyl mercury, then
11 inorganic mercury would also accumulate in the brain,
12 is that correct?

13 A Yes.

14 Q And yet, and this is one of the articles you
15 were relying on, Petitioners' Master List No. 568,
16 after exposure to methyl mercury poisoning the
17 Purkinje cells were not damaged?

18 SPECIAL MASTER VOWELL: And that would be
19 page?

20 MR. MATANOSKI: I'm sorry. It's pages 201
21 to 202.

22 THE WITNESS: Actually, let me look at this.

23 SPECIAL MASTER VOWELL: And that would be
24 page 3 and 4 on the Petitioners' Master Reference 568.
25 They're separately numbered.

1 THE WITNESS: Could you continue that
2 paragraph? Right. What I can see here, but perhaps
3 I'm missing your point, is that indeed doses high
4 enough of methyl mercury to kill cells preferentially
5 kill granular cells is what this is telling me.

6 BY MR. MATANOSKI:

7 Q And these were the same doses that you were
8 relying on before for your glutamate proposition, is
9 that right? This was the article you referred to, PML
10 No. 568.

11 A That's the Charleston article.

12 Q No, sir, it's the Aschner article.

13 A All right. Tell me again. And this is the
14 same article I refer to for what proposition?

15 Q Excess glutamate after methyl mercury
16 exposure.

17 A Right.

18 Q You were explaining to the Court how this
19 fits into your theory of mercury creating an excess of
20 glutamate and a hyperarousal state.

21 A I relied upon this article to point out that
22 exposure to mercury products has the effect of
23 inhibiting glutamate reuptake. Now, I wasn't talking
24 about killing neurons, I was talking about a
25 functional state of overactivation. Now, of course

1 it's a matter of degree, and if the dose is large
2 enough the effect is enough to kill neurons, that will
3 also happen.

4 The death of neurons is not a part of my
5 model that I presented to the Court.

6 Q Are you familiar with Bauman and Kemper's
7 work in performing and reviewing autopsy samples of
8 autistic individuals?

9 A I'm aware of their early work which was
10 pioneering in that field.

11 Q Are you aware of what findings they made
12 with respect to Purkinje cells in those individuals?

13 A In autism, yes, there is a smaller number of
14 Purkinje cells than expected in the cerebellum of the
15 autistic people that they autopsied.

16 Q And after exposure to methyl mercury in one
17 of the articles you're relying on for your theory, the
18 Purkinje cells were relatively spared?

19 A My model is one of excitotoxicity, and I
20 have documented for the Court the fact that Purkinje
21 cells are particularly vulnerable to excitotoxicity.

22 Q And if excitotoxicity is a model of autism,
23 then should we see Purkinje cells missing in autism?

24 A If you have excitotoxicity you will see an
25 overexcitation of neurons and in a case like autism

1 that I am talking about it may rise to a sufficient
2 level to kill some cells. If it does, they're likely
3 to be Purkinje cells because they're more vulnerable
4 to excitotoxicity than other brain cells.

5 Q And this paper that you're relying on for
6 part of your process doesn't find that to be happening
7 with respect to the Purkinje cells, correct?

8 A I'd like to tell you again, my overarousal
9 model does not depend on the loss of Purkinje cells.
10 If there are Purkinje cells lost, that's probably
11 because the excitotoxicity rose to that level. If
12 they're not lost, it's probably because they didn't
13 rise to that level.

14 Q Isn't excitotoxicity glutamate excess, which
15 is the proposition that the Aschner paper was being
16 cited for?

17 A I didn't hear what you said.

18 Q Isn't excitotoxicity glutamate excess? Is
19 that correct? You just nodded your head.

20 A Yes, yes, yes.

21 Q And isn't that the proposition that the
22 Aschner paper was being cited for?

23 A Yes, the Aschner paper says that
24 excitotoxicity can result from these mercury products.

25 Q And they didn't see this result with

1 Purkinje cells that one sees in autism, correct?

2 A It's an empirical fact that in methyl
3 mercury poisoning, which we don't have in autism, the
4 granule cells are more vulnerable than the Purkinje
5 cells to being killed. I don't know why that is, and
6 I don't think other people do either.

7 Q You've cited several studies on methyl
8 mercury other than the one we were just discussing to
9 support your claims about thimerosal. What is your
10 relative confidence that ethyl mercury will
11 toxicologically act the same way as methyl mercury in
12 the articles that you've discussed?

13 A Would you refer me to what you're reading
14 here?

15 Q How about we'll start with the Charleston
16 article.

17 A Ethyl mercury and methyl mercury, as we have
18 discussed, both have the same end point of inorganic
19 mercury. I am picking up the story at the inorganic
20 mercury.

21 Q Now, in your report on pages 10 and 11 you
22 cited Charleston, which is PML No. 116, the 1996
23 Charleston article, for the proposition that the
24 astrocyte population in the brain decreased
25 significantly after six months of methyl mercury

1 exposure while microglial cells proliferated.

2 Do you know how much mercury is in one dose
3 of hepatitis B vaccine as preserved with thimerosal?

4 A Do I know how much mercury is in what?

5 Q One dose of hepatitis B vaccine that was
6 preserved with thimerosal.

7 A It may be 12.5 micrograms or something of
8 that order.

9 Q Do you know how much mercury is in one dose
10 of diphtheria, tetanus and acellular pertussis vaccine
11 preserved with thimerosal?

12 A It's off that order, but, as I have said,
13 the exact amounts are matters on which I would consult
14 a toxicologist. I couldn't deduce anything from a
15 figure like that by myself as a neurologist.

16 Q You have an extensive discussion of this
17 Charleston article. Do you know the amount of mercury
18 administered in one day to the macaques in that study
19 was?

20 A I didn't note the amount. I just noted the
21 statement that this was meant to replicate or parallel
22 the human vaccination ranging. That's the part that
23 matters to me as a neurologist. Whether those numbers
24 in fact do that is not something that I can personally
25 judge, but a toxicologist can.

1 Q So you're relying on this article because
2 you believe that the amounts there mimic the amounts
3 used in childhood vaccines?

4 A I'm citing the article because of what they
5 say they did. I'm not the person to appraise it
6 critically, and this is why I say a toxicologist needs
7 to tell me whether indeed this is reliable, this can
8 be upheld and this is helpful in determining whether
9 the amount of mercury in the vaccine rises to the
10 level that is required to cause neuroinflammation in
11 some children.

12 Q So you believe this acts as a roughly
13 comparable amount in childhood vaccines, the amount
14 that is being used in this Charleston study?

15 A There's nothing particularly that I believe.
16 I cited an article that is pertinent to the question
17 of whether it is the case that the amount of mercury
18 acquired through a vaccination with thimerosal could,
19 in some cases, cause neuroinflammation.

20 However, a toxicologist would have to
21 evaluate those articles and the context of other ones
22 available and tell me what his or her conclusion is
23 about that matter. I am not drawing a conclusion
24 about that matter.

25 Q You reached a conclusion in your report

1 without the benefit of the consultation with that
2 toxicologist, correct?

3 A No, I didn't reach a conclusion. I've told
4 you that what I was offering was a mechanism of injury
5 in general causation. I leave it to toxicologists to
6 tell us, tell the Court, whether the amount of mercury
7 received in this way is compatible with the mechanism
8 that I have presented.

9 Q So you're comfortable relying on this report
10 in forming your expert opinion without any notion of
11 whether the amounts of mercury involved in that study
12 in any way mimic the amounts that children are
13 receiving in thimerosal-containing vaccines?

14 A The way that my report is organized is not
15 to say thimerosal is good or bad. I am talking about
16 mercury as a potential cause of neuroinflammation, and
17 mercury is a potential cause of neuroinflammation.
18 Once one has established that, then one must put on
19 one's differential diagnosis: whatever routes exist
20 by which a child might have acquired mercury.

21 Thimerosal-containing vaccines is one such
22 route. At this point, the clinician must find out
23 from a toxicologist whether the mercury delivered by
24 that route was indeed sufficient to explain the
25 disease process that he or she is seeing in their

1 patient.

2 Q So you have no idea what the threshold
3 amount of mercury is to create the neuroinflammation
4 you've hypothesized occurs?

5 A As I've said, I have no idea of the
6 toxicology involved in determining a threshold dose.

7 Q So you have no idea whether the damage that
8 was observed in the Charleston study would at all be
9 related or we could in any way expect that after
10 vaccinations, or I should say doses, of mercury in the
11 amount received in vaccinations?

12 A I'm sorry, I have to keep asking you to
13 repeat yourself.

14 Q I'm sorry. I will repeat that.

15 A I'm not doing this on purpose. I really
16 can't hear.

17 Q No, I understand. You have no way of
18 judging whether the amount of mercury that caused the
19 cell damage that you described on page 10 in this
20 Charleston article would be replicated by the amount
21 in vaccinations? Though you describe it as cell
22 damage, you don't know whether the amount of mercury
23 in thimerosal-containing vaccines would cause a
24 similar amount of cell damage or any.

25 A I understood the Burbacher work as an

1 attempt to check on an animal model the possible toxic
2 affects of a vaccine which incorporates a mercury-
3 containing preservative. They were studying whether
4 that is dangerous or not. You can't study that on
5 children, you use macaques as an animal model, and
6 given the stature of the investigators, I assume it's
7 a valid animal model.

8 Now, for the nontoxicologist it is of
9 interest that they believe they found that in their
10 animal model very low doses of an order comparable to
11 those administered in vaccines at that time in fact
12 caused microglial activation, signs of inflammation.
13 Now, that is of interest to a neurologist.

14 To take it further one needs to know from a
15 toxicologist whether there's any reservations one
16 should have about this finding and whether the analogy
17 can be upheld, and, if it is, whether indeed it does
18 tell us that the small amount of mercury in the
19 vaccine is enough in some individuals to cause the
20 inflammation.

21 Q Would your opinion be any stronger or less
22 strong if the amount of mercury involved in the
23 Charleston study was let's say four times the amount
24 in a vaccine?

25 A Four times the amount of?

1 Q Yes, in a vaccine.

2 A I would not in fact engage myself in these
3 quantitative considerations because they are not in my
4 field. I don't know as a neurologist whether
5 increasing something 50 percent or 400 percent makes
6 this much difference or that much difference. That
7 really is a toxicological question, and toxicologists
8 are involved in this hearing because it is indeed a
9 toxicological question.

10 In cases like this, as you know as well or
11 better than I do, sometimes you need multiple
12 specialties to contribute their specialized knowledge
13 and that be integrated and considered in that sense by
14 the Court. I am contributing what I feel a
15 neurologist can, and I believe that to be helpful to
16 the Court. It doesn't lead me to the final decision
17 with which you credited at me at the onset of your
18 cross-examination.

19 Q So, again, without the benefit of a
20 toxicologist, you have no idea whether childhood
21 vaccines would cause any -- any -- of these changes
22 that were observed in the Charleston study?

23 A Well, you use the phrase "no idea." I have
24 an idea. I've read the Burbacher articles and they
25 are potentially alarming, but I can't take it further

1 as a neurologist. It certainly makes it worth my
2 while to consult a toxicologist as to what's going on
3 here.

4 Q And if these studies that you're relying on
5 or that you discuss actually have far greater amounts
6 -- far greater amounts -- of mercury involved in them
7 that are in childhood vaccines, would you be less
8 confident in your opinion?

9 A If I were to discover that these studies
10 were incompetently done, then I would no longer put
11 any weight in them.

12 Q What do you mean incompetently done?

13 A What I mean is if it were the case, which I
14 don't believe for a moment, that although they
15 purported to be mirroring the amount of mercury given
16 to children in their animal model they weren't doing
17 that at all, then of course I would lose confidence in
18 that.

19 But, in any case, it doesn't matter because
20 a toxicologist is going to make the final decision
21 here. I would defer to that person.

22 Q In the Charleston articles you discussed,
23 where did the researchers look for microglial
24 activation in astrocyte death? Let's start with the
25 1996 Charleston article. Where did they look for it

1 in the brain of the adult macaque?

2 A Well, I can refer to it of course. I know
3 they looked for it in the cerebral cortex and in the
4 cerebellum. If you want more detail, I'll look it up
5 in the article.

6 Q So you believe they looked for it in the
7 cerebral cortex and the cerebellum, is that your
8 answer?

9 A I'm sorry.

10 Q In the 1996 Charleston article, you believe
11 they looked for microglial activation and astrocyte
12 death in the cerebral cortex and the cerebellum?

13 A Well, I don't need to believe. If you refer
14 me to the article and the page I will give you --

15 Q No. I'm asking what you believe they did in
16 that study in rendering your opinion.

17 A Well, I've already answered that question.

18 Q And your answer was the cerebral cortex and
19 the cerebellum?

20 A Yes, but, again, it has no impact on my
21 opinion because my opinion isn't focused on a critical
22 area.

23 Q So if astrocyte death and microglial
24 activation didn't happen in the cerebral cortex and
25 the cerebellum or if you had no evidence for that,

1 your opinion wouldn't change?

2 A If I know that microglial activation has
3 been reported and I know that the glutamate system is
4 all over the cerebrum, then that sets the stage for my
5 model. Whether it is overactivation of the neurons in
6 the cerebrum or whether it is brainstem nuclei that
7 are firing excessively overactivating the cerebrum
8 makes no difference at the level of analysis that I'm
9 discussing.

10 Just to make sure that what I've just said
11 is intelligible to the Court I'd like to say it again.
12 One can get overactivation of the cerebrum either
13 because the neurons in the cerebrum are firing more
14 than they should, which might happen if the
15 precipitant toxin were there, as, for example, the
16 Lopez-Hurtado article implies. Or it might be that
17 systems in the brainstem, lower systems, which send
18 activation up to the cerebral cortex are firing too
19 much and causing overarousal that way.

20 In either of these ways, you would get the
21 overarousal and the consequences that I described.

22 Q So it doesn't matter to you where the
23 astrocyte death or microglial activation occurs?

24 A Well, I'm not sure that you need to rephrase
25 my answer. I gave my answer. I told the Court two

1 different possibilities, either of which would lead to
2 the same outcome. I don't like to say that something
3 just doesn't matter.

4 Q Are there any other areas of the brain
5 involved, other than the two that you've just
6 discussed, that would lead to the effect that you have
7 described as the cause of autism?

8 A Yes, there are. As I just mentioned, nuclei
9 in the brainstem which project to the cerebrum and
10 activate it. If those were working too hard, firing
11 too much, one will get the overarousal that is
12 critical to my opinion on general causation.

13 Q Any other areas of the brain?

14 A I don't recall what they found, and you
15 don't wish me to refer to it. It may be that the
16 hypothalamus was involved, but I'm not sure.

17 Q The hypothalamus, did you say?

18 A It may be that the hypothalamus was
19 involved, but I'm not sure.

20 Q Is that one of the areas that you're opining
21 that if there was astrocyte death and microglial
22 activation it will result in the effect that you've
23 hypothesized is involved in this process?

24 A What I'm opining is in my report, and I
25 don't recall opining on those specific areas.

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1 Q Do you have an answer for my question beyond
2 that, sir?

3 A No. My opinion doesn't critically depend on
4 the particular area that's most affected. I'm talking
5 about a system, the glutamate system, and general
6 overactivation of neurons.

7 Q And it doesn't matter what area of the brain
8 that activation may occur for the effect to be seen?

9 A In my opinion, it's fairly general.

10 Q Were any behavioral disturbances observed in
11 the macaques in the Charleston papers?

12 A I don't recall them.

13 Q You don't know whether there is or you don't
14 recall that there were any?

15 A I don't recall that there were.

16 Q How do the neuropathological findings in the
17 macaques in the Charleston paper compare with the
18 neuropathological findings in autistic patients? Are
19 they similar? Are they different?

20 A I don't recall being struck by any
21 particular similarity but the neuropathological
22 finding in autistic people --

23 SPECIAL MASTER HASTINGS: Both of you
24 gentlemen, and probably for me with my cold, I'm not
25 hearing either one of you very well. If you could

1 both speak up a little bit.

2 THE WITNESS: I don't believe that the
3 neuropathology done in the Burmeister articles in the
4 mid-1990s really examined the brain in the same way,
5 the highly specialized way, in which the brain of
6 autistic children is examined. So it's really hard to
7 make an analogy as to whether when the macaque monkeys
8 were given these low doses of mercury, the particular
9 areas, same areas, were affected in the brief time
10 that they stayed alive as is found at autopsy in
11 autistic individuals many years after their condition
12 began.

13 I haven't seen comparable studies. I
14 haven't seen a group applying the same methods to
15 autistic brains and brains of animals dosed with
16 mercury to make that comparison.

17 BY MR. MATANOSKI:

18 Q So you don't have any information on that?

19 A I don't think the study offers us that kind
20 of information.

21 Q Is neuroinflammation a part of normal brain
22 development?

23 A To a minor extent it can be.

24 Q Does it ever serve a beneficial affect?

25 A Yes. I mean, it wouldn't have evolved if it

1 didn't serve a beneficial affect. The beneficial
2 affect is what I've already mentioned to the Court
3 which is that it is part of the defense of the
4 organism against intruding substances.

5 Q In your hypothesis, how much time elapses
6 between the deposition of mercury in the brain and the
7 start of neuroinflammation?

8 A I don't know that. I don't believe a fixed
9 time is known.

10 Q What time would you expect there to be?
11 What period would you expect?

12 A Well, if the brain is treating an invader,
13 whether it's a virus, or a metal, or another toxicant,
14 as an intruder, it will of course react to that to
15 some extent right away. Now, that reaction, however,
16 as I have testified, may go on for a long time, and it
17 may wax and wane, it may get greater, at which point
18 one could detect it histologically. One would have to
19 ask a neurohistologist.

20 My best answer I can give to your question
21 is that it should begin within a short time.

22 Q And what do you mean by a short time? Can
23 you give us that in hours? Days?

24 A Well, days or a few weeks.

25 Q What's the lower bound of that timeframe?

1 A It's a matter of degree. You might have
2 some response almost immediately when the intruder
3 enters the brain, but that response may gather and
4 become clinically apparent at some later time. I
5 wouldn't say it would take three years.

6 I think that since it is a defense reaction
7 it would have to happen within the timeframe where a
8 useful defense could be mounted.

9 Q When, under your hypothesis, will it become
10 clinically manifest?

11 A Within days or weeks.

12 Q How many days in the lower bound?

13 A No, I can't tell you how many.

14 Q I'm sorry, you cannot tell me? Is that what
15 you said?

16 A I can't tell you how many days.

17 Q And in the upper bound, how many days or
18 weeks will it be?

19 A First of all, we're dealing with
20 probabilities. To expect an answer to a question like
21 that one is regarding the presence of inflammation and
22 its degree as being some simple reaction which is
23 unaffected by anything else. There could be a variety
24 of factors in the brain.

25 Sometimes there are intruders in the brain

1 that stay in the brain for years and don't elicit
2 information, and something happens to perturb the
3 system and the inflammation occurs, so to start giving
4 time limits like this is not helpful. The best I can
5 do to help is to say that it would be quite compatible
6 with the theory for the effects to be seen within days
7 or a few weeks.

8 Q To the extent you're comfortable to 50
9 percent, a little more than 50 percent probability,
10 can you put a range of time on when to expect this
11 that's any more precise than that?

12 A Well, I'd like to explain to the Court why I
13 cannot do that. As the Court knows, the measles virus
14 is capable in some cases of staying in the body for
15 years. Now, here is an intruder, and yet, in the case
16 of SSPE, subacute sclerosing panencephalitis, it's not
17 unusual for six, eight or 10 years to pass and then
18 the inflammation, and the damage, and the necrosis,
19 and finally, the death, occur.

20 So we don't know the factors that permit
21 some reactions to be quick and hold off other
22 reactions for months or years and then they happen
23 anyway with an agent that must have been there all
24 along. So I was just explaining to the Court why I
25 cannot offer you the numbers that you ask me for.

1 Q You used the example of SSPE, when that
2 manifests itself. This was one of your examples in
3 the last time that you were testifying with this
4 hypothesis of neuroinflammation leading to autism.

5 A Yes.

6 Q What is the result of SSPE?

7 A I'm not comparing SSPE to autism, I'm making
8 a particular point. People with SSPE almost always
9 die, people with autism fortunately don't.

10 Q In your theory, does the neuroinflammation
11 continue as long as the inorganic mercury is present?

12 A Yes.

13 Q Will the neuroinflammation subside if the
14 amount of inorganic mercury decreases?

15 A Presumably, although I know of no empirical
16 evidence of that.

17 Q Will the neuroinflammation increase if the
18 amount of inorganic mercury increases?

19 A It sounds logical that it should, but I know
20 of no situation where these various levels have been
21 compared within the same person.

22 Q Do you know of any situation where these
23 various levels have been measured at all, that is,
24 that there's been a neuroinflammatory response to
25 inorganic mercury in humans?

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1 A All I know about are measures at autopsy.

2 Q For inorganic mercury and neuroinflammation
3 or just neuroinflammation?

4 A I'm sorry, I thought you were talking about
5 measuring mercury in the brain, is that right?

6 Q I was trying to find out since you couldn't
7 tell me, you said it sounds logical but you were
8 hedging your answer, about neuroinflammation
9 increasing if the amount of inorganic mercury
10 increased. You said because you didn't have any
11 evidence you were hedging an answer.

12 A I don't quite know how one would find this
13 out experimentally with means available now, so I
14 really can only give you an answer which doesn't tell
15 you more than you already know, which is, it sounds
16 reasonable.

17 Q So logically, though, you'd expect the
18 neuroinflammation to increase if the inorganic mercury
19 is increased?

20 A Well, if it is the case in a particular
21 person that inorganic mercury is causing
22 neuroinflammation, then giving some more would
23 presumably make it worse, but, you know, I can't back
24 that up with numbers or I can't cite an article on
25 that.

1 Q Why would neuroinflammation under your
2 postulate only manifest itself as regressive autism?

3 A You asked me why it only manifests in
4 regressive autism?

5 Q Yes.

6 A I don't know that it only manifests in
7 regressive autism. Did I say in my report that it
8 only does?

9 Q Your report on page 4 begins with an
10 extensive discussion of regressive autism.

11 A That's correct.

12 Q And you talked this morning extensively
13 about regressive autism.

14 A Yes.

15 Q Are you suggesting now that your opinion
16 extends beyond regressive autism?

17 A No. You just asked me, and an only
18 question, and the answer is rarely that something
19 happens only in medicine. Regressive autism has a
20 timing which is consistent with an environmental, as
21 it were, second hit which could have occurred in one
22 or multiple events preceding the age of 15 months or
23 whatever it is.

24 In other words, if you have a child who is
25 slowly becoming autistic over time it is still

1 possible that any of the agents causing
2 neuroinflammation were accumulating in that child, but
3 we don't know. What is very dramatic is that the
4 child was developing normally and then suddenly,
5 fairly suddenly, something really changes and the
6 child gets very, very sick.

7 Now, in such an event, if that happens, an
8 environmental factor of the appropriate timing has to
9 be considered. That is a particularly striking
10 situation calling for explanation. Put it another
11 way, regressive autism is a serious loss of mental
12 skills in young children and requires an explanation.

13 To say, oh, well, this is just the way
14 autism presents sometimes is not an explanation.

15 Q You asked where in your report you had
16 limited your opinion to regressive autism.

17 On page 24 you say it is therefore medically
18 reasonable to consider the involvement of a TCV-
19 induced encephalopathy when one engages in the
20 differential diagnosis of a case of regressive autism,
21 particularly when other known medical causative
22 factors in the differential diagnosis have been ruled
23 out or are not supported by reliable evidence. So
24 your report is limited to cases of regressive autism?

25 A Yes. That doesn't mean I said it could only

1 occur there. I am discussing regressive autism
2 pointing out that whereas there are many causes known
3 or postulated for the classical autism, there really
4 are very few diagnostic alternatives with regressive
5 autism that are known.

6 Q The mechanism that you have laid out applies
7 then equally to regressive autism or nonregressive
8 autism?

9 A No. I'm putting it forward for regressive
10 autism. Whether it extends to any other kind of
11 autism, I haven't considered it in that context.

12 Q Why would neuroinflammation, as you've
13 described it, only manifest itself as regressive
14 autism?

15 A I don't believe for a moment that it only
16 happens in regressive autism. I also believe that it
17 has more than one cause. For example, if one gives a
18 mother terbutaline during pregnancy and then finds
19 neuroinflammation in the autistic child, that would be
20 another cause of neuroinflammation.

21 I wouldn't be surprised if there were other
22 viruses than the measles virus and if there were other
23 toxicants than mercury that could do the same thing.
24 I began by saying that there are multiple potential
25 causes.

1 Q So your hypothesis of neuroinflammation is
2 not restricted to regressive autism?

3 A No. Not at all. I'm sorry if I gave that
4 impression.

5 Q You described Dr. Deth's opinion as an
6 opinion of this process that you've described at a
7 molecular level, is that accurate?

8 A No. What I was expressing was that he was
9 studying at the molecular level a particular component
10 of the broader process that I was invoking.

11 Q I'm sorry, I didn't hear that.

12 A Okay. He was taking one component of the
13 disordered system that I describe, namely the
14 oxidative stress, and explaining it very expertly at
15 the molecular level.

16 Q Is that the operation of neuroinflammation
17 at the molecular level in your view?

18 A It is. When there is neuroinflammation one
19 of the situations that is set up, as I have explained,
20 is oxidative stress. He was explaining to us how that
21 works.

22 Q If his description of oxidative stress were
23 less likely to occur, do you believe that would impact
24 on the likelihood of the mechanism that you've
25 described?

1 A If you mean if there was oxidative stress
2 but explained to us some other way would it make it
3 less likely, is that what you're asking?

4 Q Yes, sir.

5 A I need help here. Could you rephrase?

6 Q Are you trying your neuroinflammation theory
7 at the molecular level to oxidative stress?

8 A No. Oxidative stress is a well-known
9 phenomenon studied by many people. It is known to
10 occur in this situation. The mechanism by which it
11 occurs is not critical to my particular account that
12 I'm offering to the Court.

13 Q You don't know how the neuroinflammation
14 would cause the cell death?

15 A I don't know.

16 Q Do you know how the neuroinflammation would
17 cause the cell death and then thereafter the glutamate
18 excess?

19 SPECIAL MASTER HASTINGS: Can you speak up,
20 Mr. Matanoski?

21 THE WITNESS: Yeah. I'm hoping I'm
22 answering your question because I didn't hear it very
23 well. I know that when microglia are activated they
24 release reactive oxygen species, ROS, which are a
25 known potent cause of oxidative stress. Now, the

1 biochemistry by which they cause oxidative stress in
2 its details I certainly don't know as a neurologist.

3 BY MR. MATANOSKI:

4 Q How much excess glutamate is necessary for
5 astrocyte death?

6 A I'm sorry, I have no idea what you said.

7 Q How much excess glutamate is necessary for
8 astrocyte death?

9 A If you're asking me for quantities, I cannot
10 tell you at all. I don't know whether there is a
11 specialized literature that determines that. It
12 doesn't really impact on my opinion.

13 Q You cited Aschner and Mutkus -- I'm sorry, I
14 do not have the PML for this -- for the proposition
15 that methyl mercury alters glutamate transport.

16 A Yes.

17 Q Do you know how that experiment was
18 conducted?

19 A I don't recall now. I mean, there are so
20 many studies that draw the same conclusion.

21 Q If it was in cell culture, would it affect
22 your level of confidence that the same would be seen
23 in a human?

24 A Not very much.

25 SPECIAL MASTER CAMPBELL-SMITH: Excuse me,

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1 Mr. Matanoski. We've been going for a little while
2 now and just getting an idea of how much longer you
3 anticipate your cross to go.

4 (Away from microphone.)

5 MR. MATANOSKI: I think I could --

6 SPECIAL MASTER CAMPBELL-SMITH: That's fine.
7 In terms of quantifying matters, how much longer do
8 you --

9 MR. MATANOSKI: I think if I could have
10 until 1:30 I could finish up. I'm going to try to cut
11 some of this down.

12 SPECIAL MASTER CAMPBELL-SMITH: Okay.

13 MR. MATANOSKI: I'm sorry, it was PML 571.
14 Thank you.

15 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

16 BY MR. MATANOSKI:

17 Q The amount of methyl mercury used in that
18 experiment, how does it compare with the amount of
19 methyl mercury from a DTaP vaccine? Is it greater or
20 lesser?

21 A I don't recall. It wasn't offered as being
22 at the level of a vaccine.

23 Q Does it matter to you how much, what the
24 relative difference is?

25 A Well, it matters enough for me to want to

1 consult a toxicologist on whether the amount of
2 mercury delivered by the vaccination program is enough
3 to cause a microglial activation and inflammation.
4 It's all the same question back again.

5 Q And in rendering this opinion you had not
6 consulted with such a toxicologist prior to rendering
7 an opinion?

8 A I cannot give you a toxicology opinion, no.

9 Q You had not consulted with one when you
10 wrote this opinion, though, correct?

11 A No. I left that open. As I have said
12 before, I was offering what I take to be a reasonable
13 mechanism of injury.

14 Q Is excess glutamate known to be a cause of
15 regressive autism?

16 A No.

17 Q Is it known to be a cause of autism?

18 A It's not known to be. Once you see the
19 Vargas-type findings one can become quite suspicious
20 of that being the case. The question of the role of
21 neuroinflammation in the causation of autism is being
22 very actively studied, including by the Hopkins group.

23 They certainly know very well that excess
24 glutamate is part of that process. They have enough
25 suspicion of that new information in fact being the

1 cause of the autism that they are now administering an
2 agent that counteracts microglial activation in an
3 attempt to reduce or even cure the autistic
4 manifestations.

5 This is a project using a substance called
6 minocycline which has a specific effect of decreasing
7 microglial activation and for which they got a funding
8 support from the National Institutes of Health.

9 Q The symptoms that you expect after the
10 mechanism that you described, are they any way
11 clinically distinct -- I'm sorry. Are the symptoms
12 that you describe after this, are they clinically
13 distinct in there's a difference between those in
14 regressive autism or in autism?

15 A No. As I pointed out, once the regression
16 has occurred, the person is in an autistic state. I
17 mean, varies in severity just like it does with
18 congenital autism. Any differences are quantitative.
19 There are no really big qualitative differences. For
20 example, there's a greater tendency for regressive
21 autistic people to have seizures but it's a matter of
22 degree. The similarities are more than the
23 differences.

24 Q And you don't belong to any toxicological
25 societies?

1 A No.

2 Q You don't read and review any toxicological
3 publications?

4 A No.

5 Q You haven't been invited to speak on
6 toxicology?

7 A No.

8 Q And you haven't published any papers on
9 toxicology? I'm sorry?

10 A Sorry, I'm thinking. I'm reviewing my CV
11 for a moment. That's why my eyes are watering. No.
12 Not specifically on toxicology, no.

13 Q And you haven't had a clinical practice with
14 children to speak of in about 18 years?

15 A Yeah.

16 Q And in the 1980s you published two articles
17 on autism, and since then you've published a textbook
18 chapter that discussed developmental disabilities and
19 that's been the extent of your publications on autism
20 in the last 20 some years or 30 years?

21 A I published an article probably about two
22 years ago, Liss, et al.

23 Q That was the results of a general survey?

24 A Well, I don't know about general survey. It
25 was a study of certain autistic behaviors performed in

1 collaboration with Dr. Deborah Fein at University of
2 Connecticut, and it utilized my questionnaire for
3 overfocusing that I referred to briefly earlier in the
4 discussion.

5 Q Now, in Cedillo, I asked you a question
6 about the relative strength of your hypothesis, the
7 various parts of your hypothesis, and you went through
8 those for me. Do you recall that?

9 A I do.

10 Q And this mechanism that you've given us
11 today is essentially the same mechanism, right?

12 A Yes. I mean, that is the point I'm making.
13 I'm not presenting it as specific to mercury, let
14 alone thimerosal.

15 Q And I asked you how confident you were that
16 this innate immune response, which is the
17 neuroinflammation we're talking about, correct?

18 A Yes.

19 Q I asked you how confident you were that the
20 innate immune response at that time to measles vaccine
21 or measles virus was having, correct?

22 A Yes.

23 Q And we can substitute in for our purposes
24 now TCVs, or I should say mercury, correct?

25 A It's mercury.

1 Q And at that time you stated that your
2 confidence hovered at about the 50-percent mark?

3 A It was over, yeah.

4 Q Just, yes, about 50 percent I think is what
5 you said. Is it the same today?

6 A The situation is slightly different, so let
7 me talk about it for a moment while I consider my
8 answer. I am very confident that mercury can cause
9 neuroinflammation and that is well-documented in the
10 literature. I don't think it's at all a matter of
11 controversy.

12 I think there are good arguments, good
13 reasons for supposing that when you get
14 neuroinflammation that the kind of consequences that I
15 describe, manifesting autism, in fact occur, but, you
16 know, I mean, there are other theories, as I've
17 pointed out.

18 I am not at all confident whether the amount
19 of mercury in the vaccines is at the level to elicit
20 this inflammation because that is something that, as
21 I've explained, for which I need assistance from a
22 toxicologist. Now, I see enough evidence from the
23 macaque work to believe that it is really important to
24 find from a toxicologist what that answer is.

25 Q I asked you to describe the weakest part of

1 your hypothesis at that time, and in answer to that
2 question you said that it was your particular
3 mechanism of injury. Let's call it the glutamate
4 excitation hypothesis. Is that still the weakest
5 part?

6 A Okay. Again, if you could just repeat what
7 I said? Could you do that again?

8 Q It was essentially that the weakest part of
9 your hypothesis was the glutamate excitation. Is that
10 still the weakest part of your hypothesis?

11 A Well, it's gotten stronger, but let me
12 answer it this way. For most of what I've been
13 testifying I'm relying on published literature by
14 people other than myself and literature which I
15 believe is of high quality.

16 My analysis of the neuroinflammation as
17 having the outcomes that I explained to the Court I am
18 fairly confident of; however, that is my personal
19 opinion appreciation analysis. I do have some
20 confidence in myself, yes.

21 Q In terms of this overall theory, is it about
22 just over the 50-percent mark, or are you very
23 confident in this hypothesis you've given us, is it
24 somewhat lesser than that?

25 A If I were very confident, then I would claim

1 that this was at the scientific level, way above the
2 level called for by these proceedings. All I can
3 really say is that I believe it to be above the
4 threshold set by the Claims Court for a reasonable
5 mechanism of medical injury.

6 Q Now, you testified in Cedillo and Snyder,
7 and in each of those cases the child had also received
8 thimerosal-containing vaccines, and in each instance
9 you didn't opine that the thimerosal-containing
10 vaccines were more likely or even a potential cause of
11 the autism in those instances, is that right?

12 A Did you ask me that about the thimerosal?

13 Q You reviewed those cases and were offering
14 an opinion in each of those cases. Each of those
15 cases had thimerosal-containing vaccines, and you
16 offered the opinion in those cases that it was the
17 measles vaccine, is that right?

18 A I did.

19 Q And you didn't offer the thimerosal-
20 containing vaccines as even a potential cause of the
21 autism, correct?

22 A No, I didn't at all. I hadn't studied the
23 matter, and I hadn't formed any opinion whatever on
24 the rights or wrongs of the thimerosal theory at that
25 time, nor had I been asked to.

1 Q In the Cedillo case you had written a 27
2 page report discussing that case, and, again, in the
3 Cedillo case the child had also received thimerosal-
4 containing vaccine, and that report didn't contain a
5 suggestion that thimerosal-containing vaccines were a
6 cause of that child's autism, correct?

7 A No, I hadn't studied or been asked to study
8 that possibility at all, nor had I much reason to
9 because when you actually find a neurotropic virus in
10 the body of a child, that's a very potent phenomenon
11 in the differential diagnosis. As I was telling you
12 earlier, I had really only begun to consider the
13 question of mercury very recently.

14 Q And in that instance the Court was
15 considering whether the combination of thimerosal-
16 containing vaccines and the measles vaccine caused
17 autism, and yet, you still didn't render this opinion
18 that thimerosal vaccines could have any role.

19 A At the time I left that discussion, as is
20 proper, to the toxicologist.

21 Q In Snyder, you also presented a 27 page
22 report, and in Snyder there also was thimerosal-
23 containing vaccines, and in that instance you also did
24 not opine or even suggest that the thimerosal-
25 containing vaccines could be a potential cause of the

1 child's regressive autism, isn't that correct?

2 A That's correct.

3 Q And while the vaccine at issue is different
4 in these cases, significant portions of your causation
5 mechanism are precisely the same, correct?

6 A Could you say it again?

7 Q While the vaccine at issue is different in
8 this instance, in this case, or at least your
9 causation opinion is to a different vaccine --

10 A So is there a particular vaccine at instance
11 in this case?

12 Q Thimerosal-containing vaccine versus measles
13 vaccine.

14 A You mean the range of them, yes. Yes. If
15 you're saying that MMR does not have thimerosal, that
16 is correct.

17 Q Significant portions of your causation
18 mechanism are exactly the same, correct?

19 A Oh, yes. Absolutely.

20 Q In fact, if we compare passages in the
21 Cedillo trial on several key points, they're virtually
22 identical with your report here, is that right?

23 A Absolutely. When I realized that mercury
24 can set up essentially the same neuroinflammation that
25 viruses can, then I began to take seriously the

1 question of the possible intervention of mercury.
2 That's not something that I had worked on until quite
3 recently.

4 Q In fact, they're virtually identical except
5 that you essentially replaced the word "measles virus"
6 with "thimerosal-containing vaccines," correct?

7 A Well, it took me an awful lot of hours to be
8 identical. No, I don't agree with that, but that's a
9 matter of detail which I can analyze if you want me
10 to.

11 Q I think we'll leave this to our closing
12 briefs. Actually, now we do have these passages up.
13 Can you review that? This is your report in the
14 Cedillo case I believe, is that right? In the Snyder
15 case.

16 SPECIAL MASTER VOWELL: And that would refer
17 to page 16 of what's labeled Petitioners' Exhibit 29
18 on the left of the screen?

19 MR. MATANOSKI: Correct. I'm sorry, ma'am.

20 SPECIAL MASTER VOWELL: There are two things
21 up there and if you say this is, we don't know which
22 one when we read it later.

23 SPECIAL MASTER CAMPBELL-SMITH: And you're
24 comparing that with --

25 MR. MATANOSKI: With page 13 of the report

1 that you submitted in this matter.

2 THE WITNESS: Uh-huh.

3 BY MR. MATANOSKI:

4 Q And the changes that are seen are
5 highlighted, and those are the only changes, correct?

6 A As I've testified, when I'm describing
7 neuroinflammation I'm describing the same thing
8 because there are multiple causes for that thing.

9 Q You served as an expert witness in 130 cases
10 for petitioners in this program, or approximately 130
11 cases, is that right?

12 A I haven't counted them. I would rely on you
13 to tell me how many.

14 Q Does that sound about right in terms of your
15 recollection?

16 A Yes, it does.

17 Q How many vaccine cases are you retained in
18 currently?

19 A How many?

20 Q Are you retained as the expert in currently?

21 A How many cases am I active at the moment?

22 Q Yes.

23 A I'm sorry. I'm visualizing my study and my
24 visual memory isn't that great. I don't know, 20, 30.

25 Q I'm sorry, did you say 20 or 30?

1 A See, as you know, vaccine cases take
2 forever, you know, many, many years, and some of them
3 are getting quite dusty on the shelves and some are
4 active. You know, right now I guess I have like half
5 a dozen cases that are active but others are going to
6 be, you understand?

7 Q When will you testify next in a vaccine
8 case?

9 A The next one, there is one hearing before
10 Special Master Golkiewicz in June and one before
11 Special Master Edwards in November. Those are the
12 ones that I'm aware of currently.

13 Q Could you list some of the conditions you've
14 claimed are related to vaccines?

15 A The conditions I --

16 Q You have claimed are related to vaccines.

17 A Conditions that I have claimed relate to
18 vaccines. Encephalopathies, seizure disorders,
19 Guillain-Barré syndrome, transverse myelitis, ADEM,
20 asepticemia caused by a contaminated vaccination
21 needle in a baby causing abscesses. If you had a
22 list, that would really help me.

23 Q There may be more?

24 A Huh?

25 Q Are there more? That's all you can recall

1 at this time?

2 A I expect so.

3 Q Have you ever previously rendered an opinion
4 involving heavy metals?

5 A I don't think I have previously offered any
6 such opinion.

7 Q Now, in Cedillo you discussed your career,
8 as you did here today, and you mentioned a change in
9 your career plans or career path. You said you made a
10 decision to no longer work in conventional faculty
11 fashion in terms of service, teaching and research but
12 to concentrate on your research program.

13 What prompted your decision to change your
14 career path at that time?

15 A I found that service and teaching, and my
16 interest in research, as my CV reflects, became
17 increasingly intense and my group published more and
18 more articles. And there came a point when I felt
19 that I really couldn't sustain effectively all my
20 teaching service responsibilities and yet without
21 losing momentum in research. So I went instead to a
22 position in which was basically a research institute
23 where I would still see many children but see them on
24 a selected basis. At the time it was mostly children
25 with attention deficit disorder, so I could study them

1 intensely and perform the kinds of experiments that I
2 wanted to do.

3 It was the kind of midcareer decision that
4 quite a few medical specialists make.

5 Q So you made that decision after spending six
6 years in Canada at the University of Toronto?

7 A Yes.

8 Q And what were the circumstances that
9 prompted your departure from the University of
10 Toronto?

11 A There was a disagreement with the new
12 chairman, the details of which I'm not at liberty to
13 disclose.

14 Q You were terminated, weren't you?

15 A Actually, I wasn't. Actually, I resigned.
16 I certainly didn't want to stay there.

17 Q And the grounds the university was using in
18 their termination of your employment was that you had
19 committed unethical billing practices and that you
20 billed for services you hadn't performed, correct?

21 A No, none of that.

22 Q And another ground was that you falsified
23 information on your curriculum vitae, information that
24 they relied on in appointing you to the faculty,
25 correct?

1 A No.

2 Q This is an excerpt from --

3 MR. POWERS: Excuse me. Could we have a
4 copy at counsel table? I can't read anything that far
5 away at this point. Do you have a copy that we could
6 take a look at?

7 MR. MATANOSKI: I didn't think we were going
8 to have to go through this, frankly.

9 MR. POWERS: Well, I'm just asking for a
10 copy that I can look at.

11 BY MR. MATANOSKI:

12 Q This was taken from a grievance panel
13 proceeding where they were working through some
14 procedural aspects of a case where you were contesting
15 your termination from the University of Toronto and
16 the grounds the university had notified you of, at
17 least as found by this grievance panel, that you're
18 aware of were these grounds for their action, correct?

19 A I'm not sure I heard everything you said,
20 but I can tell you what is correct. These were
21 accusations made against me, I filed a grievance, the
22 grievance committee found in my favor, I decided to
23 leave anyhow, and I was paid a sum of money, some
24 compensation, but I was perfectly prepared to leave.
25 The fact is that these accusations were dismissed at

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1 the hearing by the committee.

2 MR. MATANOSKI: I have no further questions.

3 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

4 We are in a position now to take a lunch recess. My

5 thought would be, and we'll probably give some

6 discussion and thought to where we anticipate going

7 this afternoon. Petitioners' counsel, how much

8 redirect for Dr. Kinsbourne?

9 MR. POWERS: I anticipate 20 to 30 minutes

10 of redirect, Special Masters. To be on the safe side,

11 I would say the half hour in terms of scheduling my

12 end of that.

13 SPECIAL MASTER CAMPBELL-SMITH: Okay. Do

14 you have some thought just looking forward to the

15 extent of our day? I know that we have as well time

16 allotted to hear from Mr. Mead and Ms. King. Do you

17 anticipate that will require our moving into a late

18 day or is there a reasonable breaking point, if you

19 will, that we could perhaps reserve some testimony to

20 commence tomorrow?

21 MR. POWERS: I would propose the latter,

22 Special Master, and I'm sort of low blood sugar enough

23 I can't even do the clock math. But assuming Mr. Mead

24 is able to take the stand and testify until relatively

25 close to the end of the day, rather than extend late

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1 we would propose having Ms. King come in tomorrow
2 morning and testify.

3 They're both available tomorrow and they'd
4 be ready, willing and able to do that. So I would
5 much rather do that, and even if it meant ending
6 technically a little bit earlier rather than trying to
7 get both folks in. That's what we propose.

8 SPECIAL MASTER CAMPBELL-SMITH: Just a
9 thought so that as people go into lunch and have
10 thoughts about a working lunch you know what work
11 might be anticipated not only during lunch but later
12 in the day. Is there any concern that you have about
13 that, Mr. Matanoski?

14 MR. MATANOSKI: No. No, ma'am.

15 SPECIAL MASTER CAMPBELL-SMITH: Thank you.
16 Well, so advised. We'll take an hour. I'm looking
17 for a timepiece that will give me some sense of time.
18 I have here, which is purportedly chronologically
19 correct, 1:38, which we'll round to 1:40, so we'll
20 look to see you at 2:40 this afternoon to restart.

21 ALL: Thank you, Special Master.

22 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

23 (Whereupon, at 1:40 p.m., the hearing in the
24 above-entitled matter was recessed, to reconvene at
25 2:40 p.m. this same day, Wednesday, May 14, 2008.)

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1 SPECIAL MASTER CAMPBELL-SMITH: Okay. All
2 right. Are there any other matters before we return
3 to redirect?

4 MR. POWERS: No, Special Master. We're
5 ready for redirect.

6 SPECIAL MASTER CAMPBELL-SMITH: Please. To
7 proceed, counsel. Dr. Kinsbourne, you continue under
8 oath.

9 Whereupon,

10 MARCEL KINSBOURNE

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

14 REDIRECT EXAMINATION

15 BY MR. POWERS:

16 Q Good afternoon again, Dr. Kinsbourne.

17 A Yes, Mr. Powers.

18 Q And since sound, volume and logistics are an
19 issue I just want to make sure that you can hear me
20 across the room here.

21 A Very well, thank you.

22 Q Great. I can hear you. I do want to
23 address some issues that were raised by Respondent's
24 counsel in their cross-examination. Now, do you
25 recall a series of questions relating to Volume 6 of

1 the child neurology textbook in which you wrote a
2 chapter?

3 A Yes.

4 Q And those questions were directed towards
5 Volume 6, as I recall. Is that what you specifically
6 remember, too?

7 A What was the date of that volume now?

8 Q Well, how about if I put it this way. The
9 questions that were asked were in relationship to some
10 material that appeared in the 6th Edition of the
11 *Menkes* textbook.

12 A Yes, that's right. That's right, yeah.

13 Q Okay. And the current edition is the 7th
14 Edition, is that correct?

15 A Yes, that's correct.

16 MR. POWERS: Okay. And, Special Masters,
17 I'm going to discuss a couple of things about this
18 textbook so I think we have to mark it as an exhibit,
19 although physically it might be a little hard to
20 reproduce it and pass it around, but I think the
21 questions won't actually necessarily need reference to
22 the material in the book.

23 SPECIAL MASTER CAMPBELL-SMITH: Are you
24 limiting it to the particular chapter that Dr.
25 Kinsbourne has authored?

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1 MR. POWERS: Yes, I am, Special Master.

2 This would be Chapter 18. What trial exhibit would we
3 be up to, madam reporter?

4 THE REPORTER: Four?

5 SPECIAL MASTER CAMPBELL-SMITH: Four is what
6 I have.

7 (The document referred to was
8 marked for identification as
9 Petitioners' Exhibit No. 4.)

10 BY MR. POWERS:

11 Q So for the record, and for you, Dr.
12 Kinsbourne, we'll call this Petitioners' Trial Exhibit
13 4, and by that I'm referring to the child neurology
14 textbook, 7th Edition.

15 A Thank you.

16 Q And since people listening in can't see what
17 we're doing, I'm holding up so you can look at it. Do
18 you see it?

19 A Yes, sir.

20 MR. POWERS: When I open this book I notice
21 that there is a Library of Congress catalog and
22 publication date of 2005. With the Special Masters'
23 indulgence, I'd like to just show that page to
24 opposing counsel and to the witness.

25 SPECIAL MASTER CAMPBELL-SMITH: Please.

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1 SPECIAL MASTER HASTINGS: Well, we'll take
2 your word for it.

3 MR. POWERS: Okay.

4 SPECIAL MASTER HASTINGS: I mean, I think
5 maybe what you could do is whatever pages are
6 appropriate you could file those after you go through
7 it.

8 MR. POWERS: I'd be happy. To the extent
9 that we do need to follow some of the foundational
10 introduction rules of evidence I wanted to do that,
11 but if we can just make it shorter, which we can, I
12 will represent to you that the publication date was
13 2005. Does that seem right for that book?

14 THE WITNESS: Yes.

15 BY MR. POWERS:

16 Q So if the publication date was 2005, do you
17 have a rough idea of when you would have had to have
18 completed the materials for your chapter?

19 A Probably a year and a bit before.

20 Q So at some point in 2004, perhaps earlier,
21 is when you would have finished your review of the
22 literature that would have appeared in this book?

23 A Right. At the latest.

24 Q So between the time you did that and the
25 submission of the report here today you have developed

1 an opinion that you have since expressed about the
2 potential role for mercury in causing
3 neuroinflammation, is that correct?

4 A Yes.

5 Q And that's an opinion that has developed
6 from the time that the book was published until you
7 presented your expert report here?

8 A Absolutely.

9 Q In fact, if one looks at the references that
10 you cite in the attachment to your expert report here,
11 and, again, I'm not going to ask you to count them but
12 I will represent to you that there are 22 articles
13 cited there that were published in 2005 or later.
14 Does that sound about right to you?

15 A Yes, it does.

16 Q And this would include articles that you
17 rely on in your report and in your testimony including
18 one of Dr. Aschner's studies, the Burbacher infant
19 monkey study, Vargas and Pardo. Does that all sound
20 correct? These would be after 2004?

21 A Yes, indeed.

22 Q So would it be fair to say then that from
23 the time this book was published until offering your
24 opinion today there's been, to the extent that it
25 informs your opinion, a significant body of new

1 scientific literature upon which your opinion is
2 based?

3 A Yes, it is.

4 Q Do you also recall a line of questioning
5 relating to your expert report and your testimony in
6 the Cedillo matter and the Snyder matter?

7 A There was such a line, yes.

8 Q And that was a line during cross-examination
9 with Mr. Matanoski, correct?

10 A Yes, sir.

11 Q You were asked why you did not include any
12 reference to your current opinion regarding mercury as
13 a potential cause of regressive autism. You were
14 asked why you didn't express that opinion in June of
15 2007. Do you recall having that question?

16 A Yes, I do.

17 Q Why didn't you express the opinion back in
18 June 2007?

19 A I hadn't formed it at that time.

20 Q You were asked again by Mr. Matanoski why
21 you didn't proffer this opinion in the Snyder matter
22 which was heard in November of 2007, and I can't
23 recall if you answered it specifically so I'll ask you
24 now. Why didn't you proffer today's opinion back in
25 November of 2007?

1 A I had not formed it at that time.

2 Q What did you rely on between November of
3 2007 and April 2008 when your expert report was filed
4 to develop the opinion that you express today?

5 A At least some of what made me think harder
6 about the matter were some articles by Aschner, one of
7 which was filed, and the Lopez-Hurtado article, which
8 came out I think just a very short time ago.

9 Q And, excuse me, the Lopez-Hurtado article,
10 was that the autopsy study?

11 A That was the autopsy study with the striking
12 loss of astrocytes in the language area, yes.

13 Q So would it be accurate to say that the
14 opinion that you have ultimately come to here today
15 did not become final until some time in the period
16 between January and early April 2008?

17 A Absolutely.

18 Q I want to now talk about some of the
19 substance of the science that came up on cross-
20 examination. Do you recall a line of questions from
21 Mr. Matanoski asking you about astrocyte death? Do
22 you recall those questions?

23 A Yes.

24 Q Do you recall questions specifically asking
25 about what dose of mercury it takes to kill

1 astrocytes? Do you recall that?

2 A Yes.

3 Q Now, in looking at your neuroinflammatory
4 model, is it essential to the model that you have
5 astrocytes dying in large numbers? Is that an
6 essential part of your model?

7 A Not at all. As I explained to the Court,
8 the critical point of information is that the
9 astrocytes under attack by microglia suspend their
10 ability to reuptake glutamate and scavenge it away
11 from the synapse so that they no longer can exert
12 their regulatory function the cause of which the
13 glutamate goes out of control and then the
14 consequences I mentioned of overexcitation follow.

15 So whether they, astrocytes, actually die
16 subsequently would not affect this particular
17 function. Makes no difference.

18 Q And you were also asked if it was the
19 mercury itself that was directly toxic to the
20 astrocytes to kill them. Do you remember those
21 questions?

22 A Yes.

23 Q In your theory or your model of causation
24 here, the inflammatory model, is it necessary that the
25 mercury itself directly kills or impairs the function

1 of the astrocytes to create the sequelae you describe?

2 A No. The mercury in the astrocyte could
3 attract the immune attack but the microglia trying to
4 eliminate it and result in the death of the astrocyte.

5 Q And not only in the death of the astrocyte,
6 but would it be accurate to say the cytokine
7 environment could impair the function of the
8 astrocytes without killing them?

9 A Right, and, as I mentioned before, my model
10 really does not rely on cells actually dying, although
11 some do, but rather on cells suspending certain vital
12 functions.

13 Q So this is a functional issue, not a cell
14 count issue necessarily?

15 A Absolutely.

16 Q And, again, the function that you're
17 describing here with whether it's the death of
18 astrocytes or the functional impairment of astrocytes
19 is the glutamate uptake, is that right?

20 A Yes.

21 MR. POWERS: Now, I am going to show a
22 couple of articles up on the screen for you and ask
23 some questions about those. Scott, if you could put
24 up, I should have it memorized by now, I think it's
25 116, the 1996 Charleston article. And if you could

1 zoom in to the title of that, please, Scott.

2 BY MR. POWERS:

3 Q Dr. Kinsbourne, can you describe what you
4 see on your computer monitor there?

5 A Well, the title of the article is *Changes in*
6 *the Number of Astrocytes and Microglia in the Thalamus*
7 *of the Monkey Macaca Fascicularis Following Long-Term*
8 *Subclinical Methyl Mercury Exposure*, and it's by
9 Charleston and others.

10 Q Now, do you recall a line of questions when
11 you were on the witness stand under cross that focused
12 on the Charleston article that you see in front of you
13 now?

14 A Yes, there was such a line of questioning.

15 Q During that line of questioning, were you
16 provided with a copy of the article to refer to in
17 response to specific questions?

18 A No.

19 Q Do you recall being asked what part of the
20 brain was being examined in this paper?

21 A Right, I was asked that.

22 Q Do you recall what your answer was?

23 A My answer was cerebrum and cerebellum.

24 Q Looking at this exhibit in front of you now
25 would your answer be any different now than it was

1 earlier?

2 A Yes.

3 Q What would your answer be now?

4 A Thalamus.

5 Q And that's just clearly in the text of the
6 article, correct?

7 A Yeah.

8 MR. POWERS: Scott, if you could turn to
9 page 2 of the exhibit. Again, for the record, this is
10 Petitioners' Exhibit 116, page 2.

11 BY MR. POWERS:

12 Q And, Dr. Kinsbourne, we're going to draw
13 your attention to the right-hand column. You see a
14 section that's labeled methods. Do you see that?

15 A Yes.

16 Q And about halfway or about a third of the
17 way into that specific section there's a sentence
18 that's highlighted that begins four groups were. Do
19 you see that?

20 A Yes.

21 Q Take a look at that sentence and tell me
22 what your understanding is of what's going on in this
23 study.

24 A Well, the animals were given methyl mercury
25 on a daily basis, a low dose, but a high-ish low dose,

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1 not an extremely low dose.

2 Q So these were adult monkeys being given
3 methyl mercury orally, correct?

4 A Right, and being given on a daily basis.

5 Q And during your cross-examination do you
6 recall referring to this adult monkey study as having
7 involved the administration of thimerosal vaccines?

8 A Actually, I don't, so I hope I didn't.

9 Q Okay. Because it's your understanding of
10 this article that these were methyl mercury exposures
11 involving oral administration, correct?

12 A Right. No, the thimerosal came up later.

13 MR. POWERS: You can pull that down, Scott.

14 BY MR. POWERS:

15 Q And when you say the thimerosal came up
16 later, what are you referring to?

17 A Well, the infant study that I mentioned. It
18 was to the infant macaques that the vaccine schedule
19 and thimerosal was relevant. That's where they tried
20 to establish some parallel in the dose so as to elicit
21 some hopefully parallel changes if they occur in the
22 brain.

23 MR. POWERS: Okay. Let's go ahead and put
24 that on the screen, Scott. That would be Petitioners'
25 Exhibit 26. And, again, Scott, if you could zoom in

1 on the title there.

2 BY MR. POWERS:

3 Q Dr. Kinsbourne, now that it's up, could you
4 describe what you see on the screen?

5 A Title is *Comparison of Blood and Brain*
6 *Mercury Levels in Infant Monkeys Exposed to Methyl*
7 *Mercury or Vaccines Containing Thimerosal*, by
8 Burbacher and others.

9 Q Okay. So what of these two articles that
10 I've just shown you, the Clarkson article and the
11 Burbacher article, which of these two articles were
12 you referring to when you described it as an
13 experiment that administered doses of thimerosal in a
14 way meant to mimic the human thimerosal exposure?

15 A Well, the 2005 article by Burbacher and
16 others which was about infants.

17 MR. POWERS: And in fact let's just make
18 sure of that. Scott, if you could turn to --, and in
19 the left-hand column under materials and methods the
20 heading that begins mercury dosing schedule, if you
21 could zoom in on that, please?

22 Actually, Scott, if you could extend down to
23 the full length of that paragraph?

24 BY MR. POWERS:

25 Q Dr. Kinsbourne, could you read that

1 highlighted section?

2 A Yes, sir. Seventeen infant monkeys assigned
3 to the thimerosal group were given the typical
4 schedule of vaccines to human infants.

5 Q If you could then look down, it's not
6 highlighted but it's the very last sentence in that
7 paragraph and it begins with the words "a dose of."

8 A A dose of 20 micrograms per kilogram was
9 chosen based on the range of estimated doses received
10 by human infants receiving vaccines during the first
11 six months of life.

12 Q So whenever you were referring then to a
13 monkey study involving sort of a schedule designed to
14 simulate human exposure it would be this 2005
15 Burbacher paper, is that correct?

16 A That's correct.

17 MR. POWERS: And, Scott, you could pull that
18 down. Thanks.

19 BY MR. POWERS:

20 Q Now, Dr. Kinsbourne, is it your
21 understanding that Dr. Burbacher has a second portion
22 of the 2005 study that's underway?

23 A Yes.

24 Q What is your understanding of what that
25 study seeks to determine?

1 A My understanding is that that study seeks to
2 determine whether there is neuroinflammation in the
3 brains of these infant monkeys.

4 Q And is this the type of evidence you would
5 be looking for to provide an answer to the question of
6 what dose of thimerosal in vaccines might reasonably
7 be thought to trigger neuroinflammation? Is this the
8 sort of evidence you would be looking for?

9 A Very much so, yes.

10 Q This would be evidence that you would rely
11 on in forming an opinion about whether the specific
12 dose of thimerosal could trigger your inflammatory
13 model, is that correct?

14 A That's correct.

15 Q I also want to give you a hypothetical.
16 Let's assume that a reliable, credible toxicologist
17 testified that if one compared between the 2005 infant
18 monkey study, if you looked at the highest doses of
19 inorganic mercury remaining in the brain, it's the
20 highest doses among those monkeys, and you then looked
21 at the 1996 data for the adults where they actually
22 found inflammation, if that range of exposures
23 overlaps, would that be evidence to you suggesting a
24 threshold dose of thimerosal that might cause
25 inflammation?

1 A To make sure that I understand this, we
2 start with the fact that the adult monkeys were given
3 bigger doses than the baby monkeys in the later study,
4 right?

5 Q Correct.

6 A Now, we know that on principle infant
7 monkeys, humans, other creatures are more susceptible
8 to mercury. However, what you are suggesting is that
9 the highest doses given to some of the infant monkeys
10 might have overlapped the lower doses given to the
11 adult monkeys, is that correct?

12 Q I'm sorry? Let me rephrase the hypothetical
13 for you if need be because if you have this many
14 questions about the hypothetical it's apparently not a
15 clear hypothetical.

16 A Okay.

17 Q The hypothetical was this. You have one
18 study that shows that with a certain amount of
19 inorganic mercury that ends up in the brain you have
20 neuroinflammation. So that's a given, that's the
21 Charleston paper. You then have another paper that
22 looks specifically at thimerosal-containing vaccines.

23 That paper doesn't show any inflammation
24 yet, but it does show how much mercury actually got
25 into those parts of the brain.

1 A Right.

2 Q If there was an equivalence between that
3 infant dose that we know is in the brains and the
4 adult dose that we know is in the brains and causes
5 neuroinflammation, would that increase your ability to
6 answer the question of what the threshold dose would
7 be, assuming that, as I said before, a reliable
8 toxicologist explained that?

9 A Right, and assuming that it was
10 systematically true of the group of monkeys, that
11 would. It would, indeed, because there's no reason to
12 suppose that a given dose, a given amount of a heavy
13 metal is less effective in eliciting neuroinflammation
14 in infants than it is in adults.

15 Q Is there any reason to believe that it might
16 be more effective at causing neuroinflammation in
17 infants under the age of two than it would be in
18 adults?

19 A Yes.

20 Q What's the basis for that?

21 A Just experience that infants are more
22 susceptible to these anomalous reactions.

23 Q Another question. The Burbacher study, the
24 2005, because I don't want to create any more
25 confusion since Burbacher is on many of these studies,

1 the 2005 infant schedule, if you recall the excerpt up
2 there, it mimicked the vaccine schedule for the first
3 six months of life, correct?

4 A Yes.

5 Q So it didn't mimic the schedule for the
6 first full year of a human's life or the first two
7 years of a human's life?

8 A Right.

9 Q Do you believe that repeated exposures to
10 ethyl mercury via thimerosal can lead to an
11 accumulation of inorganic mercury in the brain?

12 A Well, there are some mercuries delivered
13 every time, and since it is broken down to inorganic
14 mercury, and since that can now no longer leave the
15 cells, it must accumulate to some extent.

16 Q So one would expect then if an animal, and
17 let's talk specifically if a human being, if a human
18 child received not just the six months of thimerosal-
19 containing vaccines but the full-year and the full
20 two-year schedules, you, based on your review of the
21 literature, believe that additional inorganic mercury
22 would be deposited in the brain, is that fair?

23 A Yes, I do.

24 Q And if additional inorganic mercury would be
25 deposited in the brain would it increase the

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1 likelihood that the mercury put into the brain by
2 thimerosal-containing vaccines might be at a dose
3 sufficient to trigger inflammation?

4 A Perhaps you would repeat that last bit.

5 Q I should make it even simpler. If you get
6 extra inorganic mercury in the brain does it increase
7 the likelihood that in fact it will lead to an
8 inflammatory dose?

9 A It must surely do that, yes.

10 MR. POWERS: I have no further questions.

11 SPECIAL MASTER CAMPBELL-SMITH: Mr.

12 Matanoski?

13 MR. MATANOSKI: Thank you.

14 RE-CROSS-EXAMINATION

15 BY MR. MATANOSKI:

16 Q Doctor, what's the dose necessary to elicit
17 neuroinflammation? What dose of mercury is necessary?

18 A You've asked me this before and I told you
19 that I didn't know.

20 Q And what else did you say? Did you say you
21 needed a toxicologist to explain that?

22 A Well, there's nothing to explain exactly to
23 determine that.

24 Q Mr. Powers just asked you a series of
25 questions about what you'd expect from a series of

1 doses and what the expectation would be after the
2 receipt of inorganic mercury and you answered those
3 questions.

4 A Yes, because they were different from the
5 ones that you asked me. You asked me for doses. He
6 asked me whether if a certain amount of a substance
7 elicits neuroinflammation, would adding to it elicit
8 more neuroinflammation? That I'm competent to answer.
9 Yes, it would.

10 Q And what's the lowest threshold that you can
11 think of before neuroinflammation would start --

12 A That is what I would like a hypothetical
13 toxicologist to tell me.

14 Q And if inorganic mercury comes from methyl
15 mercury, then that would increase in the brain over
16 time, too, correct?

17 A Certainly.

18 Q And I believe in your report you said it
19 stays in there for years?

20 A Absolutely.

21 Q And in your testimony this morning you said
22 maybe for life, correct?

23 A I did.

24 Q And if one takes in methyl mercury with this
25 inorganic component in it and one gets it from fish,

1 from dental amalgams, in utero from the maternal
2 sharing of the blood, correct?

3 A Yeah.

4 Q From breast-milk-feeding, from tunafish
5 sandwiches, from eating chicken, according to Dr.
6 Aposhian. All those methyl mercury exposures would
7 continue to build up inorganic mercury in the
8 recipient's brain, correct?

9 A Correct, which is why I kept telling you
10 that I talk about mercury from whatever source.

11 Q Okay. And if, as you said, it could stay in
12 there for years, maybe even for life, then the
13 neuroinflammation affect that you were talking about,
14 which would increase under the hypothetical that you
15 were given by Mr. Powers, would continue to increase
16 over time, correct?

17 A It sounds logical that it would. Now here's
18 some immunology I'm not sure about, whether it is such
19 a simple relationship, but it could potentially do
20 that. In fact, neuroinflammation has been found in
21 autopsies of people in adult life and in midadult life
22 at the very least, so it certainly can continue for a
23 long, long time.

24 Q Okay. And when Mr. Powers asked you that,
25 you did think it would increase with increasing

1 inorganic mercury in the brain?

2 A I think that's a reasonable thing to
3 suppose.

4 Q And you said you thought it would be likely
5 to happen, correct?

6 A What was the last thing I said?

7 Q You believe that would be likely to happen,
8 correct?

9 A Yes, I do.

10 MR. MATANOSKI: Thank you.

11 SPECIAL MASTER CAMPBELL-SMITH: Any
12 additional questions?

13 MR. POWERS: Not for the Petitioner, no.

14 SPECIAL MASTER CAMPBELL-SMITH: Dr.
15 Kinsbourne, with neuroinflammation your theory is this
16 is acute or chronic neuroinflammation?

17 THE WITNESS: It would be chronic. It would
18 build up and simmer, as it were, over a long period of
19 time.

20 SPECIAL MASTER CAMPBELL-SMITH: Chronic
21 neuroinflammation, does that lead to
22 neurodegeneration?

23 THE WITNESS: It potentially does. It
24 depends on the level of the inflammation. If it is at
25 a low level, which we believe it is because the doses

1 are really very low doses, as we're discussing, then
2 it would in my opinion lead to certain distortions of
3 the functions of cells but by no means necessarily
4 kill them.

5 SPECIAL MASTER CAMPBELL-SMITH: I guess what
6 I'm trying to sort out is your opinion is a
7 qualitative opinion. You've made it very clear that
8 it's not based on a particular quantity. Yet, there
9 is a response to, as you indicated, low level episodic
10 exposures to mercury of whatever sort that permit a
11 deposition of organic mercury in the brain.

12 THE WITNESS: Right.

13 SPECIAL MASTER CAMPBELL-SMITH: And it is
14 your theory that it is the presence of that inorganic
15 mercury, from whatever source, that can lead to this
16 neuronal dysfunction through a process of continued
17 neuroinflammation that is somehow below the level,
18 whatever that level is, that would lead to
19 neurodegeneration.

20 THE WITNESS: Correct.

21 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

22 THE WITNESS: Thank you.

23 SPECIAL MASTER CAMPBELL-SMITH: Have my
24 questions triggered any questions by counsel?

25 MR. POWERS: No, they have not, Special

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1 Master.

2 MR. MATANOSKI: Not from the government.

3 SPECIAL MASTER CAMPBELL-SMITH: Any further
4 questions?

5 (No response.)

6 SPECIAL MASTER CAMPBELL-SMITH: Thank you,
7 Dr. Kinsbourne. You're excused.

8 THE WITNESS: Thank you, Special Master.

9 (Witness excused.)

10 SPECIAL MASTER CAMPBELL-SMITH: Petitioners'
11 counsel, are you prepared to call your next witness?

12 MR. POWERS: Yes, we are. Petitioners would
13 like to call George Mead.

14 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

15 MR. POWERS: Counsel was ready. I just
16 wanted to make sure Mr. Mead was ready.

17 SPECIAL MASTER CAMPBELL-SMITH: Good
18 afternoon, Mr. Mead.

19 MR. MEAD: Good afternoon.

20 SPECIAL MASTER CAMPBELL-SMITH: Would you
21 please raise your right hand?

22 Whereupon,

23 GEORGE MEAD

24 having been duly sworn, was called as a
25 witness and was examined and testified as follows:

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1 SPECIAL MASTER CAMPBELL-SMITH: Thank you.
2 To proceed.

3 MR. POWERS: Thank you.

4 DIRECT EXAMINATION

5 BY MR. POWERS:

6 Q Good afternoon, Mr. Mead.

7 A Good afternoon.

8 Q Just so that we get a good, clean record,
9 could you state your name and spell it for the court
10 reporter, please?

11 A My name is George Winslow Mead, M-E-A-D.

12 Q And, Mr. Mead, where do you live?

13 A I live in Portland, Oregon. West Linn,
14 Oregon, to be more precise.

15 Q West Linn is a suburb of Portland?

16 A It is.

17 Q How long have you lived there?

18 A Three and a half years. I've lived in
19 metropolitan Portland since 1992.

20 Q Now, you're William Mead's father.

21 A I am.

22 Q Is William Mead's mother here in the
23 courtroom today?

24 A She is. Victoria Shirley is his mom.

25 Q Okay. And with different last names,

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1 clearly you're not married right now. You and
2 William's mother are not married?

3 A Regrettably not.

4 Q Okay. What do you do for a living?

5 A I'm an attorney.

6 Q What sort of law do you practice?

7 A Construction, real estate and real estate
8 litigation. There was a time about 10 years ago when
9 I, well, for a period of about 10 years I did medical
10 malpractice defense and I stopped doing that, so for
11 the last 10 years I've been doing real estate,
12 construction.

13 Q Okay. So, Mr. Mead, what I want to do is
14 have you fill the Special Masters in on your narrative
15 description of your son, William's, life, particularly
16 in those first couple of years. Obviously the claim
17 here is that there's a regressive autism case at
18 issue, and I'd like to explore with you for the
19 benefit of the Special Masters the facts about
20 William's condition.

21 A Certainly.

22 Q So let's just start from the beginning.
23 When was William born?

24 A May 5, 1998. He was born at St. Vincent's
25 Hospital. My memory is that I think he was nine

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1 pounds, 12 ounces. He was a large baby. Very
2 healthy.

3 Q I'm assuming that his mother's memory is a
4 little more distinct on that than yours.

5 A I think hers is slightly different than
6 mine, yeah, but it definitely was a healthy delivery.
7 It was a great day.

8 Q And an uneventful pregnancy?

9 A Yes. Yes.

10 Q Now, one thing that I had wanted to ask is
11 did William's mom while she was carrying William to
12 term -- and he was full-term?

13 A William was a full-term baby, yeah. I think
14 he may have been two days early, but I think he went
15 all the way.

16 Q So during the time that William's mom was
17 carrying William to term did she have any dental
18 fillings, any amalgam fillings?

19 A To the best of my knowledge, and this may be
20 true to this day, Tori has never had any fillings at
21 all.

22 Q Okay. You've already described just that it
23 was an uneventful pregnancy, at least as uneventful as
24 pregnancies can be, and it was a normal labor and
25 birth, is that right?

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1 A Yeah. I would like to say apropos of
2 something that the Respondent's counsel said about
3 William's before being born.

4 I can tell you that Tori went out of her way
5 not to eat any fish and not to eat any tuna or
6 anything like that, not simply as a matter because we
7 were conscious of that, but also because she simply
8 detested it at the time. And so up to the point that
9 he had been born there had been no tuna or any kind of
10 fish exposure.

11 Q Okay. Now, after he was born he was in the
12 hospital for a couple of days. During his stay in the
13 hospital was he immunized?

14 A Yeah. I don't have the immunization records
15 in front of me but I've looked at them, and I think he
16 was immunized within the first day with the hepatitis
17 B.

18 Q Hepatitis B?

19 A Right, but we didn't know about that at the
20 time.

21 Q Right. So now he was discharged from the
22 hospital. Can you go ahead and just describe for the
23 Special Masters the first few months of his life?
24 Rather than just have it be a totally open-ended
25 sentence, let's look at from the time he came home

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1 from the hospital to, say, six months of age. So look
2 at that window. If you could describe his health in
3 general and his developmental progress in particular.

4 A Well, from a general health standpoint
5 William had some bronchial issues and some ear issues
6 fairly quickly on. He seemed to have croup, and we
7 were in there fairly regularly in addition to getting
8 followup for him and in terms of well baby visits and
9 all of that, but he did have croup and had some ear
10 infections. In terms of his personality and his
11 verbal development, he was a great kid. He was
12 verbal, he was interactive.

13 Q And let me interrupt here. In the first six
14 months we're talking about, so are you saying he was
15 verbal by age six months?

16 A Well, verbal to the degree kids can be at
17 that age. He wasn't saying please pass me the paper,
18 Dad. He was basically, you know, ma, da, kind of
19 interacting very, very actively with us. Obviously he
20 was not having full words at that time.

21 You know, we had read what you can expect
22 and we had kind of educated ourselves about that, and
23 so, I mean, he rolled over appropriately, he started
24 to sit up when he was supposed to, and he was right on
25 track except for this persistent bronchial issue that

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1 we were dealing with, but globally, he was in really
2 good shape.

3 Q Now, he has an older sister.

4 A He does.

5 Q How much older than William is his sister,
6 and what's her name?

7 A Eleanor is his older sister, and she's,
8 let's say 14 months older than he is.

9 Q So she was 14 months old when he was born?

10 A Right.

11 Q So you had had experience with a first
12 child. William was not the first child.

13 A Right. I mean, we had been through it and
14 as Eleanor was developing we were kind of doing it
15 followup, so we were kind of knowing what to look for.

16 Q And I think a minute ago you mentioned you
17 were reading the *What to Expect* book?

18 A Well, we had the whole series thing, you
19 know, *What to Expect When You're Expecting*, *What to*
20 *Expect in Your First Year*, *What to Expect in Your*
21 *Second Year*. I'm not making any kind of plug for it
22 but they were useful books and we read them a lot.

23 Q And based on the *What You Expect* book said,
24 let's expand it all the way up to that first full
25 year. Were you seeing in William what you had seen in

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1 Eleanor's development and what you were led to expect
2 by the baby books you were reading?

3 A Yeah. In that first year we were seeing eye
4 contact, we were seeing verbalization. By the time
5 William was one year old, he was using rudimentary
6 language, he was getting all of his needs met, you
7 know, ma, you know, hi, ma, hi, dad.

8 There's a video bouncing around here
9 somewhere of the summer when William, I'm trying to
10 think, would have been about just after his first
11 birthday, and he was sitting, and we took a visit to
12 Sun River, and, you know, I'm hungry. He was
13 basically getting his needs met using two to three
14 word sentences by the time he was one year old.

15 Q During that first year of life did he play?
16 I mean, did he play with toys and that sort of thing?

17 A Absolutely.

18 Q Did he have favorite toys or favorite things
19 he liked to do with his toys?

20 A Well, he had a normal, you know, he had
21 stuffed toys, he had, you know, a variety of different
22 Thomas and that kind of thing that he'd play with, and
23 he had playmates.

24 He played a lot with his sister, he was part
25 of a mommy and me group and the kids would come over

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1 or he would go over, and he was fully interactive with
2 the other children at that point, and, again, kind of
3 having his language evolve during that time. You
4 know, in terms of his ability to identify things, you
5 know, I want that or I'm hungry, I hungry and pointing
6 at things.

7 His eye contact was very, very good. He
8 looked directly into your eyes. And in terms of
9 interaction, you know, you'd play with him and it
10 would be like, you know, is this the doggy? He's
11 like, no, that's not the doggy. And you kind of look
12 and be able to interact with him and play. He did all
13 of that right. I mean, he was developing perfectly
14 normally that way.

15 Q And was that with both you and his mom as
16 well as with other kids and other adults?

17 A Absolutely. And the thing I think also in
18 that time period is, you know, he could identify me by
19 name, he could identify his mom by name. We had a
20 dog, Baxter, he could identify Baxter by name, his
21 sister by name.

22 I can't remember if exactly at that point up
23 to a year he was identifying body parts, but I know
24 that he was getting to the point where, you know,
25 where's your tummy, and where's your face and all of

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1 that and he was able to do all of that stuff.

2 Q You described that he had some bronchial
3 issues and some ear infections starting some point in
4 that first six months.

5 A It was within the first three months I
6 think, yeah.

7 Q Okay. Did that recur up into the first year
8 that you're talking about?

9 A Right. That was a recurring problem for
10 William. We were, you know, consistently in seeing
11 the doc. And, you know, my memory is that at no point
12 did the doctor or anybody remark during these visits
13 that there was anything that William wasn't meeting
14 his milestones.

15 In fact, my memory is that at one point the
16 doctor even noted that, you know, he's very engaged,
17 very alert and very plugged in. This is right up
18 until he was one year old.

19 Q And during that first year of life, and,
20 again, we're not going through the medical records
21 here, they're on file with the Court, would it be
22 accurate in your recollection to say that he received
23 the normal course of childhood immunizations at two,
24 four and six months?

25 A Right. Well, obviously I've had seven years

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1 to kind of look at this issue, and I did go back and
2 look at his immunization schedule and I can say with
3 certainty that he got every vaccine under the APA
4 schedule that he was supposed to and I think maybe
5 even one more in May, but he got everything that he
6 was supposed to.

7 One other thing that was kind of an
8 important social milestone for William during the
9 first year of his life was because a friend of Tori's
10 was the art director at Williams Sonoma, was not
11 something we sought out to do because we were not, you
12 know, we don't stage kids, but we got this opportunity
13 where William got an opportunity basically to be in
14 Pottery Barn.

15 We took him down as the Pottery Barn kid,
16 and he was I think all of eight months at the time.
17 The picture, again, is floating around here somewhere.
18 But, I mean, we took him down to San Francisco, he
19 went through a photo shoot, it was a beautiful photo
20 shoot, it's a beautiful picture and he was, you know,
21 a happy, engaged kid going through that.

22 So, I mean, that to me is kind of a really
23 pretty good testament of how he was functioning at
24 that time, notwithstanding the issues he may have been
25 having with his bronchial stuff.

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1 Q Now, let's go ahead and if we could pick up
2 William's history at the end of his first year going
3 into his second year and sort of lay that out for the
4 Special Masters, if you would?

5 A Right. Well, by the time he reaches the end
6 of his first year, again, my memory is that he was
7 embarking on getting his needs met with more complex
8 language. I want down, I want go out. Do you want to
9 do this? No. Yes. I mean, kind of really starting
10 to I guess master his universe.

11 It was kind of, you know, what time is it?
12 It's bath time, you know, it's bed time. We're kind
13 of embarking into that. Also, as far as his ability
14 to navigate, he was furniture surfing and kind of
15 taking his first steps and doing that really well. He
16 had an active assistant in that process with his
17 sister, Eleanor, who was kind of, you know, right
18 there next to him.

19 They spent a lot of time together. He was,
20 again, very engaged. Actually, after his one year
21 birthday he was very verbal. I remember, you know,
22 again, that trip to Sun River stands out in my mind as
23 being something where he was really, we had a good
24 time, that was a really great trip and he was really
25 plugged in. He wasn't really suffering. I don't

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1 remember. He may have had some bronchial stuff but
2 not that much at that point. He was really doing
3 well.

4 Q Okay. And this trip to Sun River you just
5 referred to, he was about a year old?

6 A That would have been in July of 1999. He
7 was about 14 to 16 months old at that point.

8 Q Okay. And then how did things progress
9 after that, so say 14 to 16 months? You're not
10 noticing anything, but obviously you're here because
11 you started at some point to notice something.

12 A Well, right.

13 Q When did that happen?

14 A Up until he was about 18 months we continued
15 to wrestle with the ear infections and with the
16 bronchiolitis, and we were in there a lot. I do know
17 there's notes in the medical records when we took a
18 trip I think to Astoria at one point up to Ocean
19 Beach.

20 It was during the summer, and he had had
21 some coughing and stuff, and we continued to wrestle
22 with that. But, again, the whole time he's doing
23 really well in terms of, he's just kind of a sick
24 little kid in the sense that it's like can't you get
25 better as far as the bronchiolitis stuff?

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1 Then at about 18 months, you know, in
2 retrospect what we started noticing, we started
3 calling him our little engineer, and I would say about
4 18 months he started spending more time playing with
5 his toys. He wasn't becoming less verbal in the sense
6 that he was becoming quiet.

7 Of course, you know, with the benefit of
8 hindsight going back and looking at that stuff, there
9 are ominous signs that I wish I could have seen better
10 now, I mean then, than I saw. What he basically did
11 is he started playing by himself and started playing
12 with his toys.

13 He'd still play with his sister and he'd
14 still interact with us. His language, it's not that
15 he stopped suddenly one day talking. What he would do
16 is he would say, you know, I hungry or hungry, but he
17 started saying these little phrases or all I can
18 describe it as is ta-ka-ta-ka-ta-ka-ta-ka-ta-ka.

19 So he'd say something like ah, hungry, ta-
20 ka-ta-ka-ta-ka-ta-ka-ta-ka. So there was kind of
21 verbalization going on but there wasn't any, no
22 forward movement in terms of his development of
23 language. It raised red flags over time. We became
24 more concerned about it such that, you know, by the
25 time he was two years of age we were worried that he

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1 was, you know, having hearing problems.

2 There are some people who had noticed that
3 he was having difficulty navigating with other kids.
4 A couple of people had made a comment in his mommy and
5 me group that William seemed to be spending a lot of
6 time by himself. This is when he's up to about two
7 years of age.

8 Q And so just to get the timeframe correct,
9 now, you're talking about this 18 month 24 month
10 window?

11 A That's right. And that's significant in
12 retrospect because to my way of thinking kind of
13 looking back at the video and the stuff, that's when
14 we began the process of losing him. We didn't know it
15 at the time because we were simply chalking it up that
16 he was a kid who enjoyed playing with his stuff.

17 We also, you know, in that six month period,
18 probably by the time he's 20 months, his sister is
19 very gregarious and we were wondering whether she was
20 bossing him around a bunch and stuff like that. So I
21 guess in retrospect I would say that's when we were
22 beginning to lose him.

23 We thought there were a number of other
24 things that were going on with him. He was also still
25 not kicking the bronchiolitis and still having

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1 problems with the ear infections, and, you know, I'd
2 characterize him at his two-year birthday as being a
3 kid that, you know, we thought, well, you know,
4 William might be having some issues with his sister,
5 he might be having some minor hearing issues, and we
6 were prepared to look into that.

7 We took him in for his two-year shots I
8 think on May, I want to say it's like May 15.

9 Q Or, actually, and tell me if this is correct
10 or not, you actually went in a little early, in April
11 of 2000. Do you recall? We can pull the medical
12 record if you need it, but does -- a little bit before
13 his second birthday you went into the doctor's office?

14 A I remember that he went in in April, I
15 remember that he was supposed to have a well baby
16 visit and then there was also the bronchiolitis issue.
17 I can't remember as I sit here what caused it, whether
18 the well baby visit occurred in April and the
19 bronchiolitis issue was in May.

20 What I remember is he got vaccinated I think
21 in April and he got vaccinated within six weeks later
22 at the second visit. I think the second visit was the
23 one for the bronchiolitis. So after May of 2000
24 things changed for us extremely dramatically.

25 Within a matter of a few weeks William lost

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1 all the language he had, he didn't recognize us, his
2 eye contact diminished, he really kind of looked
3 through us, and one of the things that I remember as
4 kind of a milestone for me on this is, and the ta-ka-
5 ta-ka-ta-ka increased more and more.

6 He kept doing the verbal stimming during the
7 first part of that summer. And, again, we were
8 concerned that it was a hearing issue, and we finally
9 got a referral from his pediatrician for an audiology
10 clinic in September of that year.

11 Over that summer he began to have explosive
12 diarrhea, vomiting, he had unexplained welts. We took
13 him in several times to the emergency room during that
14 summer visit, again, on the coast once, and also took
15 him in to see his pediatrician. The nearer they could
16 explain was, you know, basically he might have some
17 kind of virus and we're trying to get on top of this
18 bronchiolitis.

19 William was taking albuterol for that
20 because he was getting very, very sick over that
21 summer. My parents came from New York to stay with us
22 in mid-July, and they, you know, as family will often
23 do in a loving way but in a very direct way, said this
24 kid has something very wrong with him and you need to
25 do something about it.

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1 We were, it was like we're taking him in to
2 the audiology clinic, we're getting it looked at, and
3 we agree. From that point on really, from mid-July,
4 William's condition began to deteriorate and his
5 behaviors got worse and worse. He started spinning,
6 teeth-grinding, squinting.

7 He would look out of the side of his head
8 like this and kind of look this way at you and turn
9 his head over. He started to hand-flap, he started I
10 said teeth-grinding, toe walking, jumping in place,
11 you know, like without a skip rope, just kind of a
12 skip rope skipping in place, and he would not interact
13 at all.

14 It was like he was on another planet. He
15 didn't recognize anything that he had once had,
16 frankly. I remember coming home in early September
17 and I heard William laughing downstairs. I said to
18 Tori at the time, boy, there's a sound I haven't heard
19 in a while, sounds like William's laughing.

20 I went to the top of the stairs and I looked
21 down into the, we have a finished basement, and there
22 was William at the bottom of the stairs facing the
23 corner with his arms like this just rocking back and
24 forward and laughing at nothing in particular.

25 It was one of the most shocking things I had

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1 ever seen. It was heartbreaking. So we knew that we
2 had to get him in to audiology clinic.

3 Q Excuse me. Let me interrupt. Just to keep
4 this on a timeline because timing is important in all
5 of this, this is after he's 24 months old. You're
6 working into about November into his third year at
7 this point? The incident you just described.

8 A That was early September, so William would
9 be 27 or 28 months of age at that point. His physical
10 condition was not improving at all. His stomach was
11 bloating. He looked, if you've seen the pictures of
12 the children in Darfur with the enormous belly and the
13 very, very, there's no meat up top.

14 William had massive diarrhea for weeks on
15 end that was -- and he had an insatiable appetite for
16 things like the fruit salad bowl. We'd go to
17 Albertson's and we'd get the fruit salad bowl because
18 he would eat it and it was something that he craved.
19 He would go through one of those in two sittings and
20 it would just come -- and it wasn't just fruit salad.

21 He would eat meat and stuff like that and it
22 would just go absolutely straight through him. He was
23 losing weight and he would get these unexplained welts
24 on his body. He would cover his ears. That's another
25 thing. He would kind of go like this and cover his

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1 ears back and forth.

2 We ended up at the audiology clinic in mid-
3 September and they took him through the paces and they
4 said well, the good news is that he doesn't have a
5 hearing problem, the bad news is that we're going to
6 have to set you up for Dr. Stubbs' autism clinic and
7 the first available time we have for you is mid-
8 December because we have so many people.

9 In passing one of the therapists, and I
10 don't know who she was, said, I mean, we were
11 absolutely dismayed at the time and one of the
12 therapists said, you know, some of these kids respond
13 pretty well to a casein-free, gluten-free diet.

14 Having never heard of any wheat-free, dairy-
15 free diet, and it seemed kind of like small measure at
16 the time, but we went home, and we took everything out
17 of our pantry, and put it in boxes and I gave it away
18 or through it away.

19 Starting in early October we became complete
20 casein-free and gluten-free, and what happened over
21 that point was William's diarrhea stopped, his stomach
22 bloating went down, his eye contact improved.
23 Regrettably, none of his language, it didn't
24 spontaneously return or anything like that, but we
25 started to feel like we were kind of getting control

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1 of a lot of the other stuff.

2 Right about in there there was a seminar
3 that was sponsored in Portland called Oasis that Tori
4 signed up to. She said well, I'm going to go to this,
5 it's for biomedical treatment of autism. I basically
6 said, you know, he doesn't have autism, so why would
7 you want to do that, or something constructive like
8 that, and she said well, I'm going to go anyway.

9 She came back and, you know, a box full of
10 stuff, and she said, you know, basically, sit down, be
11 quiet and start reading because this thing, we have
12 all this stuff that we have to learn about, it's
13 amazing. There was a series of presentations
14 obviously about the diet and all the stuff that had
15 been happening.

16 Meanwhile, as far as William is concerned,
17 you know, he's still continuing to spin and do all of
18 this stuff. Some of it got better after we started
19 the diet. I'd say the eye contact improved, the
20 verbalizations improved and things of that nature, but
21 we were still very concerned and very aware of the
22 fact at that point that we were losing him as we sat
23 there.

24 Q And now at this point you hadn't actually
25 been able to get in to the autism clinic yet.

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1 A No.

2 Q So it was a suggestion at that point from
3 the audiology people that autism was something you had
4 to look at, but he had not yet been diagnosed?

5 A Where we were was we were told get an
6 appointment as soon as you can, which we did, we were
7 told get into early intervention as soon as you can
8 because you're going to need to go and see them, and
9 we were told try this diet. We did all of those
10 things immediately.

11 Early intervention signed us up for a
12 meeting for what's called an IFSP, you know, basically
13 an infant plan to get your kid into special education.
14 At that point, which was early November, we went in to
15 see them and William had lost all of his gross motor.
16 He was unable to walk on a balance beam, he was
17 nonverbal.

18 Their IQ testing my memory is it was at 55,
19 which was five points above being so substantially
20 retarded that any type of early intervention isn't
21 warranted basically. And they told us that, excuse
22 me, they told us that he was going to be
23 institutionalized and that basically to expect that
24 he'd never talk again.

25 They put him on a program which they offered

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1 us and which we ended up working with them and
2 ultimately went into private because they wouldn't
3 offer him any real intervention. Anyway, at that
4 point William had lost everything that he once had.
5 Tori turned to me in early November and said to me
6 that we need to videotape William as much as we
7 possibly can.

8 I said well, why is that? And she said
9 because we need to have something on him on videotape
10 so that we remember what he was like.

11 Q If you need to take a minute.

12 A I'm okay. I hadn't really thought that much
13 about this in a little bit. Okay. I'm fine. So we
14 ended up on December 12, 2000, we went to OHSU and had
15 an appointment with Dr. Gene Stubbs, and we told him
16 basically what had happened with William, and we told
17 him about the diet and how the diet seemed to be
18 helping him.

19 He said that he had heard, he was the
20 pediatric neurologist up at OHSU and the guy who runs
21 the CDRC, the Child Development Research Center, and
22 basically, William's official diagnosis was moderate
23 autism, improving, which I thought was interesting at
24 the time because, you know, autism, at least what they
25 were telling me at that point, was that it was a

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1 lifelong disease from which there is no recovery.

2 Q And excuse me. When you say they were
3 telling you, the doctors there were telling you that
4 there's no --

5 A Right. The doctors were telling us, the
6 school district was telling us, our pediatrician who
7 we no longer obviously saw after that point, was
8 telling us that basically this was a neuro, this was
9 an under, it was nobody understood this, that it was a
10 mysterious condition for which there was no cure and
11 that there was nothing that really could be done about
12 it, and pay no attention to the fact that the diet
13 seems to improve all of the symptoms.

14 That was a dialogue that I had with Dr.
15 Stubbs. You know, it's kind of a where there's smoke
16 there's fire, and that why is this kid's symptoms
17 improving if he's on a diet and we're doing these
18 interventions? He said well, I don't really know, I
19 can't tell you that. But that, combined with what we
20 were learning through Oasis, kind of caused us to move
21 into looking into different things.

22 Q And what did you look at, and what did you
23 find?

24 A Well, we looked at a number of things. The
25 first thing that we did is there's this kind of

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1 calculus that you go through in terms of, you know,
2 Down syndrome, or it's like is this a genetic thing?
3 Is this something where there's something in Tori's
4 family or my family where, you know, my great uncle
5 Everett, you know, he had?

6 And the answer is, you know, and I can say
7 this after seven years of kind of looking at this,
8 there has been nobody, to the best of my knowledge, in
9 either side of our family that's ever had anything
10 remotely like this. So we got rid of that idea.

11 Q Now, his older sister, Eleanor, does she
12 have any developmental delays?

13 A No.

14 Q Anything? Speech delay? Anything at all
15 that's ever --

16 A No. She's a very gregarious 11, soon to be
17 12 year old, and she's had a neurotypical development.
18 She's done very well and is soon to be, you know,
19 going into middle school, so she's doing great. So we
20 kind of went through that. As part of this process,
21 we ended up with Dr. John Green, who was then in
22 Canby.

23 Q And when you say Dr. John Green, is he an
24 M.D.?

25 A Oh, M.D., yeah. Tori used to refer to this

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1 as falling down the rabbit hole and I think it's a
2 really good description because everything that you
3 kind of assume or thought you knew about a lot of
4 stuff kind of just, you fall down the rabbit hole. So
5 you start meeting new people, you start talking to
6 people on the phone who are in the community.

7 At the Oasis conference Dr. Green had done a
8 presentation. So we had the letter of introduction,
9 we found a new pediatrician, Dr. Pang, and it was a
10 big deal, and we finally got in to see Dr. Green.
11 That was in January of 2001. What he did was he
12 subjected William to a battery of tests: stool sample
13 tests, blood tests, urine tests, provoked heavy metal
14 challenge tests, metabolism tests.

15 What came out of this was shocking, frankly.
16 What the tests, and you have them in front of you, but
17 they showed that he had myelin basic protein
18 antibodies, which means that his body was dissolving
19 his own brain tissue, he had no IgM or IgA, he had no
20 immune response. He was like an AIDS patient.

21 Basically, the first thing that came along
22 could have killed him. He had massive yeast
23 overgrowth from candida albicans, he had metabolic
24 dysfunction and he had leaky gut syndrome, which means
25 that his intestinal permeability was -- and this is

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1 all in the tests there.

2 This was a kid who, in addition to all the
3 neurological stuff that we were seeing outside, was an
4 extraordinarily sick little boy. We started at that
5 point in January to embark on the journey as far as
6 the therapies that we've done for five and a half or
7 six years. We started cautiously, cautiously, with
8 chelation.

9 Chelation has a lot of spin on it. We
10 started pulling the mercury out of William, and as I
11 indicated, the first provoke challenge tests showed
12 that William had seven times the reference range. A
13 severely mercury-toxic kid. The thing that was really
14 surprising to us is that Tori never had any fillings,
15 we didn't live near a coal fire plant, we weren't, you
16 know, smelting automobiles in the back of the
17 backyard.

18 I mean, I'm trying to be a little levity,
19 but it was a shocker to us. Absolutely shocking.

20 Q And, now, obviously, Mr. Mead, not as a
21 doctor, but it sounds like you've done a lot of
22 reading, sort of the educated layperson, the
23 conclusions and the description of the test results,
24 were those provided to you by Dr. Green?

25 A Absolutely.

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1 Q I just want to make sure that what you're
2 telling the Special Masters is what a medical
3 professional has passed on to you.

4 A Dr. Green provided us copies of the tests.
5 I'm a lawyer, not a doctor. I'm a dad. I'm obviously
6 a little emotionally involved in this. Dr. Green was
7 very good about sending the tests on, and after the
8 first panel of tests came in he sat us down and it was
9 kind of like what's this all about?

10 And so we did a number of things with Dr.
11 Green's oversight, his treatment protocol, and that
12 included chelation, it included transfer factor, it
13 included zinc supplementation, we gave William a
14 number of supplements, and ultimately, because William
15 had what we suspected was gut disease, we were able
16 fortuitously to secure an appointment to see Dr. Tim
17 Buie at Harvard, at MGH.

18 Q Excuse me. MGH is?

19 A Massachusetts General Hospital. We made a
20 trip from hell with William cross-country.

21 Q When you say a trip from hell, I'm assuming
22 that's because William was not particularly able to
23 travel well?

24 A Well, at that point I think he was three and
25 a half. At that point he was severely affected. A

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1 lot of his diarrhea and stuff had subsided because we
2 were doing the casein- and gluten-free diet. But to
3 sit on an airplane going cross-country is challenging,
4 as I can tell you, for anyone, but to do it with an
5 autistic child was very difficult.

6 Then we were in the motel for two and a half
7 days waiting for this appointment. The most difficult
8 part of it was that William needed to be thoroughly
9 cleaned before they would do the endoscopy and in
10 order to do that he had to take, it was calcium
11 citrate or some awful blue stuff. And so for the last
12 day that we were waiting there William was having
13 diarrhea and just kind of clearing everything out.

14 Finally, he went in to see Dr. Buie and the
15 results of that study were very important for us. The
16 first is William came back again with lymphoid
17 hyperplasia. There was clinical. We had the
18 kodachrome color picture of William's lymphoid
19 hyperplasia.

20 Dr. Buie had said that basically as long as
21 he had the hood up, I think was the phrase that he
22 used, would it be okay if we allowed him to do a
23 pancreatic sufficiency study, meaning what are
24 William's gut enzymes looking like? We said okay.

25 What it showed, because we got those tests

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1 as well, is that basically before he provoked it
2 William had almost no -- there were three enzymes that
3 he didn't have.

4 Q And, again, Mr. Mead, I'm not going to ask
5 you to get into the details of the medicine unless you
6 know from the records, but the Special Masters will be
7 hearing from Dr. Mumper and they've seen the medical
8 records.

9 A Right. Okay. So bottom line is had no gut
10 enzymes and then did a provoke test and they got
11 pancreatic sufficiency, and it was a really kind of a
12 dramatic thing for us as well. So we continued with
13 that basically and have continued for the last five
14 and a half years with chelation, supplementation.

15 We've used a variety of different
16 antiinflammatories, including GABA, omega-3 fatty
17 acids and low-dose naltrexone, which, again, Dr. Green
18 prescribed. All of this is prescribed and it's part
19 of his treatment protocol.

20 Q And during this course of time Dr. Green was
21 providing the care for William?

22 A The whole time.

23 Q And to this day is he still providing
24 medical care to William?

25 A The whole time. And, you know, kind of

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1 where is William now?

2 Q Okay. That was going to be my next
3 question. If you could describe, again, without
4 breaking it down into exquisite detail, just describe
5 William's general course moving forward to today?

6 A All right. Well, I will try and keep away
7 from the exquisite detail. There was one thing I was
8 going to say. We did have William taken in to have an
9 MRI done because we were concerned that he had some
10 kind of lesions or anything, and that was done by Dr.
11 George Young and that came back clean as well.

12 So where is William now? Well, William
13 talks, William plays with his sister, his language is
14 emerging. He's 10 years old. He just celebrated his
15 10th birthday last week, and he went on a horse trip.
16 He plays with his siblings. He's in second grade with
17 an aid, he's reading at a first grade level, he's
18 doing math at a first grade level.

19 His language, which is kind of the most
20 noticeable thing if he were here, is utilitarian.
21 It's about getting his needs met. But, he can tell
22 you I'm happy; I'm sad; I'm hungry; I want an apple;
23 no, I don't want orange, I want an apple.

24 He is emerging to the place where, you know,
25 why did you do that, you know, I told you please don't

1 bounce the basketball. He loves to play basketball.
2 It's like why did you do that? Because. So we're
3 kind of at that point in his development where he's
4 starting to have, you know, which is huge.

5 I think probably one of the things you could
6 have knocked me over with a feather about over the
7 course of the last five weeks, when I've been getting
8 ready to go to bed and Willie's, you know, in the
9 house, you know, the door will fly open and Willie
10 will bring two books and we read together.

11 He reads and I read together. I think we're
12 making some progress there.

13 Q Now, you mentioned like when he plays
14 basketball.

15 A Right.

16 Q Apparently he's regained some of his gross
17 motor skills.

18 A Thank you for asking about that. Over time,
19 in terms of what we've done, we've chelated
20 consistently, and it's important, the mercury has
21 dropped to barely detectable levels. With that, a lot
22 of the kind of stuff, William's healthy as a horse
23 now. He doesn't get sick in the wintertime which is
24 really interesting.

25 The rest of us get the flu, William doesn't

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1 get the flu, except rarely. I won't say never, but
2 rarely. With that, his gross motor has really
3 returned. He runs, he plays basketball, he swims, he
4 rides horses, he rides, you know, a jet Ski. We took
5 him last summer. This is William in his jet Ski
6 outfit. So he's definitely doing well. He's showing
7 a lot of stuff that surprises us every day.

8 MR. POWERS: I have nothing further.

9 SPECIAL MASTER CAMPBELL-SMITH: Thank you.
10 Mr. Matanoski?

11 MR. MATANOSKI: Thank you. Ms. Esposito
12 will be doing the examination.

13 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

14 CROSS-EXAMINATION

15 BY MS. ESPOSITO:

16 Q Good afternoon, Mr. Mead. My name is
17 Katherine Esposito. I represent the government.

18 A Uh-huh.

19 Q I'm not sure if you heard my colleague for
20 her opening statement the other day. She and I both
21 share the sentiments that we would like to acknowledge
22 the journey that you've been on.

23 We may disagree as to the cause of the
24 autism, but we certainly have seen through the records
25 and the videos that you have submitted that both you,

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1 and William's mother and other family members as well
2 are very concerned about him and love him very much.

3 A We do. Thank you.

4 Q Now, when you were speaking with Mr. Powers
5 you started going through when you noticed some
6 concerns with William's development and behavior. Can
7 you walk me through some of those dates again?

8 A Well, can you be more specific about when
9 you'd like me to start?

10 Q What was the very first thing that tipped
11 you off as to William having some type of
12 developmental problem?

13 A Developmental, as opposed to hearing
14 problem?

15 Q Okay.

16 A Okay. I mean, I want to make the
17 distinction, and I'm not being coy, that there's a big
18 difference. For a long period of time we thought that
19 what William had was a hearing problem, and until
20 September of 2000 we were operating at least under the
21 fact that if he was losing language and doing this
22 stuff it might be because he had a hearing problem and
23 we were taking steps to get that addressed.

24 So I wasn't concerned at that point, I don't
25 think either one of us, that we were dealing with a

1 kid that was having a, you know, like Down syndrome,
2 or autism, or something like that.

3 I think when I first really tumbled to the
4 fact that he might be having a neurodevelopmental
5 problem was probably at the audiology clinic, was when
6 both of us knew that it was pretty serious, and that
7 would have been September.

8 Q Right. So was there a speech delay that
9 kind of went hand in hand with your noticing that or
10 your suspicion that there was a hearing problem?

11 A Yeah. Again, speech delay, he didn't stop
12 speaking. What he started doing was he started
13 speaking less and then he started this verbal tick
14 that I've described, and what I would say is that
15 occurred some time around April or March of, I want to
16 make sure I get the dates right, it would have been
17 March of 2000.

18 Q March of 2000, okay. The records state, and
19 you said, that William had up to 60 words at one
20 point. You mentioned that he was saying mom, dad,
21 Baxter, hungry.

22 A Uh-huh. Yeah.

23 Q Can you name some of the other words that he
24 might have said, if you can recall?

25 A It's been a long time. I can't remember

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1 specific. What I can tell you is things like bath
2 time, bedtime, pajamas, breakfast, hungry. Stuff
3 having to do with around the house. Dog, cat, farm,
4 horse. All of that stuff was stuff that he had
5 mastered and was actually evolving into, you know,
6 look at the horsy. That kind of stuff was where he
7 was.

8 Q And the first time that the concerns that
9 you had about William's development were raised with
10 the pediatrician was when?

11 A I don't recall that they were raised with
12 the pediatrician. I recall that there was a concern
13 that was raised -- there were two things that were
14 going on at once. William, after May of 2000, got
15 very, very sick and we were wrestling with that almost
16 on a daily basis.

17 At the same time, there was this issue of
18 his not talking, and I think his pediatrician even
19 remarked about that at some point in her notes, but
20 not directly to me at least.

21 So over that summer we're dealing with the
22 getting William better in terms of his stomach and
23 stuff and at the same time, and I think it's by
24 midsummer, I don't remember the exact date, but
25 somebody, Tori or I, had a conference and said you

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1 need to make an appointment with OHSU, and we did.

2 Q And this is you were with the family in mid-
3 July in the summer of 200 and someone in your family
4 had mentioned something?

5 A My parents. My parents had come out from
6 New York and we were at Gearhart, which is on the
7 Oregon coast. They were with us for a week and they
8 mentioned that, you know, something's not right, you
9 need to take this and get this looked at. We knew,
10 you know? It wasn't like I don't know what you're
11 talking about.

12 It's like, yeah, we have some concerns as
13 well, he's also been really sick, so we're going to
14 get him in to OHSU. If this is July, the first time
15 they could see us was in early September. So we got
16 him in as soon as we could.

17 Q When did you first think that William's MMR
18 vaccine might have caused his autism?

19 A I'm not sure that I ever developed an
20 opinion medically about MMR. I know that William had
21 an elevated MMR titer. One of the things, and I
22 didn't mention it, was that when we took the tests,
23 that William's titer showed that he was elevated, but
24 I never developed an opinion.

25 I'm not a doctor. I didn't develop an

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1 opinion about that. I think we gave William vitamin A
2 in case he had the measles virus, but that wasn't
3 something that we, you know, would say, wow, we think
4 it's his MMR. We asked for his complete vaccine
5 history and we wanted to know what he had gotten.

6 Q And at what point did you begin to think it
7 was the thimerosal in his vaccines?

8 A Well, here's how that process went. We
9 didn't identify thimerosal as the immediate villain
10 because first of all, to be candid with you, we were
11 being told that we were nuts for even thinking that
12 there was some kind of a biomedical thing for doing
13 this, so other than Dr. Green, we weren't getting a
14 lot of medical information other than what we could
15 do.

16 So we didn't automatically say well, this is
17 it and this is why it's happening. When I knew that
18 mercury was a problem for William was in January of
19 2001 because a kid who's two and a half years old who
20 has seven times the reference range for the second
21 most toxic substance on the planet, after plutonium,
22 something's wrong.

23 I didn't say, well, you know, it's
24 thimerosal, I said he's got mercury. The first order
25 of business became at that point getting the mercury

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1 out and that's what we started doing. The process of
2 elimination, which is, you know, kind of going through
3 where did it come from, we went through that as a
4 family almost immediately.

5 As I've indicated, you know, his mom doesn't
6 have any fillings. Again, there was no place for the
7 mercury to come from other than the thimerosal, so it
8 was a process of elimination for us.

9 Q Now, you said there were these lab results
10 in January of 2001 with the seven times the mercury
11 level?

12 A That's right.

13 Q Do you remember what lab that was from?

14 A I don't.

15 Q You don't know. Okay.

16 A There were actually two different labs I
17 think taken. There were two different studies that
18 were done and I don't remember who did them but they
19 both showed extraordinarily high elevated levels of
20 mercury.

21 Q I'd like to ask you a little bit about how
22 William came under Dr. Green's care. Did you first
23 find out about Dr. Green from the conference that
24 William's mother attended?

25 A About his human existence, I think that was

1 the first time that we had learned that there was a
2 man named Dr. Green.

3 No, Dr. Green came as a direct reference
4 from Dr. Alvin Pang who was William's pediatrician of
5 the Olson Pediatric Clinic who is one of the more
6 mainstream pediatricians in the Portland area who
7 actually had heard of Dr. Green's work and was not
8 prepared to undertake it himself, not because he
9 wouldn't, he said because he didn't feel qualified to,
10 and he gave us a reference to Dr. Green.

11 Q And under Dr. Green's care William has been
12 exposed to a number of different supplements? That's
13 probably putting it mildly.

14 A Well, I wouldn't say exposed. He has taken
15 a number of different supplements, all of which have
16 been indicated -- one of the things we've done is we
17 have kept track of William. I think you have a copy
18 of William's chart.

19 Over time, William has shown metabolism
20 imbalances, as I understand it, and as a result of
21 that, Dr. Green has made recommendations like he needs
22 more zinc, he needs less zinc, he's having trouble
23 keeping zinc in, he has too much, he has high copper.
24 And as we've gone through that process, and with the
25 yeast, if you go through his chart, we've battled

1 yeast for years because William had trouble kicking
2 it.

3 So we not only did casein-free and gluten-
4 free diet, but we also did the low carb diet and we
5 were hauling Atkins for a while, which was good for
6 some of us. That had the effect of helping Will kind
7 of lose some of the yeast. So he has had a number of
8 supplements, which is the short answer to your
9 question.

10 Q In December of 2000, is it correct that
11 William was undergoing 30 hours a week of ABA therapy?

12 A In the summer?

13 Q December 2000.

14 A No, I couldn't say. The short answer is I
15 know that he got ABA therapy almost immediately, and
16 if that was December when we started with Building
17 Bridges, then, yeah. I don't have the record in front
18 of me. Intensive ABA he got almost immediately.

19 Q Okay. And is it correct that Dr. Green
20 performed intravenous immunoglobulin therapy, IVIg, on
21 William?

22 A He did, and he did it once, well, for two
23 reasons. One, the IVIg was extraordinarily expensive.
24 For one course of IVIg I want to say it was close to
25 \$500 and it wasn't covered. Two, getting an IV into a

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1 three-and-a-half-year-old autistic boy is like trying
2 to cannulate a Chinook salmon. It was a horrific
3 experience.

4 So we all agreed that one course of IVIg and
5 we'd do some other transfer factor and things and see
6 if we could do it because we didn't want to put him --
7 the alternative to that particular thing would have
8 been sedation, and we just talked about that. We said
9 no, we didn't want to do that.

10 Q So he was never sedated for the IVIg?

11 A I can't remember the amount. He may have
12 received some sedation but I don't remember how much
13 he got. I know that it was a very, very awful, awful,
14 awful thing that particular day.

15 Q And this was under Dr. Green's care?

16 A Right.

17 Q Okay. Was William also on secretin?

18 A He had taken secretin.

19 Q Secretin.

20 A No, it's okay. He took secretin I want to
21 say twice. I don't have the record in front of me.
22 Again, this was because we suspected, and ultimately
23 Dr. Buie determined correctly, that William had
24 pancreatic insufficiency. So one of the things that
25 we thought would really help Will was secretin, and it

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1 did. He became more alert and he became more
2 talkative after he had the secretin.

3 Q And how many times did he have that?

4 A He had it at Harvard, which is when the
5 Pavoke challenge (phonetic) happened. I think he had
6 it at least once, maybe twice, with Dr. Green, but I
7 don't remember specifically.

8 Q William was also chelated under Dr. Green,
9 is that right?

10 A He was chelated.

11 Q And there were a number of different methods
12 for the chelation. Can you describe some of those?

13 A Well, as to the best of my knowledge,
14 William has had three chelators. He's had the DMSA,
15 which he took orally, he's had DMPS, which he got
16 intravenously, and he's had calcium EDTA.

17 He had problems with the DMSA because all
18 chelators -- and, again, I'm not a chemist and I'm not
19 a doctor, I'm just a guy that reads the internet,
20 okay, and I can tell you because I know about this and
21 from Dr. Green, that chelators are sulfur-based and
22 yeast likes sulfur.

23 So when William was taking the oral
24 chelators it was causing yeast overgrowth for him. So
25 we'd get rid of the yeast and we'd chelate, and then

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1 after we'd chelate he'd have yeast and we'd get rid of
2 the yeast. Then finally, when William was capable of
3 undergoing intravenous chelation, that's when we
4 started with DMPS.

5 Q And what was the route for the EDTA, the
6 calcium?

7 A Suppositories.

8 Q Okay. And was there a cream chelator, too?

9 A You know, I think at one point because we
10 were trying to avoid -- Will's a kid that has had more
11 than his fair share of invasive procedures. I think
12 we'd all agree with that. So we were trying to find
13 something that was not invasive, and so we tried
14 transdermal DMSA, also to avoid the yeast.

15 I don't recall that worked very well. We
16 did that for a very brief period of time I think.
17 Also, we may have had transdermal glutathione as well.
18 We tried transdermals, mostly transdermal glutathione.

19 Q Now, when William was under Dr. Green's care
20 and you're trying various supplements and the
21 different types of chelation, the IVIg, were you
22 keeping careful track of what William was on and what
23 he was off of? Did you keep a record of that?

24 A Yeah. The short answer is yes. We had a
25 chart which we tried to keep track of which we'd mark

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1 kind of the schedule according to what John Green
2 would give us, and we'd go in and mark it off and make
3 sure that he wasn't double dosing or, you know,
4 getting too much of something.

5 Q And did Dr. Green also keep careful track of
6 what William was on and off?

7 A As far as I know, yeah.

8 Q And there were times when he would go on a
9 few different things and off a few other things at the
10 same time?

11 A The short answer is it was more of an
12 adjustment process. We didn't scrap stuff and just
13 say let's just dump this.

14 What we would typically do is we would be on
15 some type of a protocol for two to three months,
16 William would make progress, we'd either have
17 intravenous, usually, embolus. We'd get up in the
18 middle of the night, we'd collect the urine, we'd put
19 it in the thing and send it off. And we'd get back
20 kind of where he was. So we'd see what his metals
21 looked like, we'd find out kind of what his
22 supplementation looked like, and then we'd have an
23 appointment with Dr. Green and we would adjust where
24 we needed to be.

25 So there wasn't any kind of this scrapping

1 wholesale what we were doing. What it really was more
2 like is when William's first tests came out he was
3 basically starving to death, so we had to keep him
4 going through massive amounts of supplements. As he
5 recovered, we got rid of those.

6 So, you know, it was like, well, he doesn't
7 need as much supplementation for zinc anymore, he
8 doesn't need this anymore and why don't we try this
9 and see if this will help.

10 Q What exactly do you mean by William was
11 starving to death? Explain that a little more,
12 please?

13 A Well, the studies that came back, and you
14 have them, and, again, I'm not a doctor, but he was
15 bloated and he had intestinal insufficiency, is what
16 it appeared, malnutrition. He was not absorbing
17 stuff.

18 Q The records at one point note that William
19 had certain oral habits. What would those have been?
20 Do you recall? Dr. Green was talking I think perhaps
21 in regard to the pica that William had. Can you
22 explain that a little more?

23 A He may have had pica at one point, you know?

24 MR. POWERS: Excuse me. I actually will
25 request not so much as an objection, but if the

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1 witness is going to be asked to comment on specific
2 comments in a doctor's record that he be able to at
3 least see the record if he's going to be questioned on
4 what the record says.

5 SPECIAL MASTER CAMPBELL-SMITH: I think
6 that's Exhibit -- and --

7 MS. ESPOSITO: The pica would be William
8 Mead Exhibit 12 at 14.

9 SPECIAL MASTER CAMPBELL-SMITH: Do you have
10 a copy for the witness?

11 MS. ESPOSITO: We could get that in just a
12 minute.

13 SPECIAL MASTER CAMPBELL-SMITH: Before we do
14 that, I would like to hear what he recalls from memory
15 regarding pica.

16 THE WITNESS: I remember that in early
17 January, around that period --

18 SPECIAL MASTER CAMPBELL-SMITH: January of
19 what year, Mr. Mead?

20 THE WITNESS: Yeah, I'm sorry, January of
21 2001. So let me just think about this and make sure
22 that I'm -- it was in the mid to late fall of 2002
23 into when we first started seeing John Green that
24 William, I remember talking about pica. I can't
25 remember what he was eating. I think he may have been

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1 eating some marbles and stuff like that.

2 He wasn't eating -- well, the short answer
3 is I think he had some pica. If you have a reference,
4 I'd be delighted to look at it.

5 BY MS. ESPOSITO:

6 Q We're going to pull that up for you. Again,
7 this is William Mead Exhibit 12 at page 14. We're
8 highlighting the bottom of the left-hand side. The
9 notation says pica in past.

10 A Well, okay. Let me just take a minute to
11 look at this. This looks like it's dated May 8, 2002,
12 which would have been almost -- it says past pica.
13 Where is this record from?

14 Q The Pfeiffer Center.

15 A Okay. Pfeiffer Center was a trip that we
16 took to Naperville just to have William looked at for
17 -- what showed up was a copper/zinc imbalance. Along
18 with the heavy metal problem that William had, his
19 test showed that he had copper/zinc imbalance. The
20 Pfeiffer Center we learned was doing stuff having to
21 do, and Dr. Bill Walsh had done a lot of stuff, with
22 the copper/zinc imbalance.

23 They were working on what was called we were
24 told a metallothionein supplement. So we made a
25 decision that we were going to travel out there, and

1 this would have been in May of 2002 when we made the
2 trip. I guess by history somebody reported that he
3 had pica in the past.

4 After William got sick he did have pica. He
5 wasn't eating cadmium batteries. He was, you know,
6 eating blocks, and gravel and stuff. That was not a
7 big habit for him. That was not something William did
8 every day. He did it in the past a few times.

9 Q Okay. Thank you. Is it correct that Dr.
10 Green would first prescribe a treatment for William
11 and then bill you for the treatment, like for a
12 certain supplement? Did you get them from him?

13 A Part of the Evergreen Clinic, it's business,
14 is it has a supplement. We got supplements from a
15 number of different places: Kirkman Laboratories, we
16 got them from Evergreen, we got them from Nature's Way
17 and a number of different places. Some of the
18 supplements we found were actually less expensive at
19 the Evergreen Center, so we would order them from him,
20 and he'd send them to us and he'd bill it for us.

21 Q There was a point in 2005 when William's
22 mother stopped taking William to see Dr. Green, is
23 that right?

24 A She and I were in the middle of a divorce
25 and she made the decision, at least at that point,

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1 that she would stop taking him, and he continued
2 anyway and he continued being seen by Dr. Green. By
3 the way, we're on exactly the same page as far as
4 that. I think that's a period of about three weeks
5 that that issue arose. So I started taking him, she
6 stopped.

7 Q William's immunization record has been filed
8 in his case as William Mead Exhibit 1 at 3. Pull that
9 up for you.

10 A Uh-huh.

11 Q Does this record accurately reflect all of
12 the immunizations that William received?

13 A You know, I'm going to have to defer on that
14 one. What I can tell you is that as far as I know
15 reviewing William's medical chart when it was provided
16 to me and looking at this, there's not a shot that's
17 reflected on here that he didn't get.

18 Q And before you had said there was another
19 shot in April or May of 2000?

20 A Yeah. Again, I'm going to refer you back to
21 the medical chart. What I remember is that he went in
22 for another reason and that somebody made the
23 decision, even though he had been inoculated within
24 the prior I want to say six to eight weeks, they gave
25 him a fifth DTaP in May of 2000.

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1 Q Are you sure it was the DTaP?

2 A I'm not absolutely certain. I just know
3 they gave him a fifth inoculation, and I think it's
4 the DTaP. I don't have the charts, again, in front of
5 me.

6 Q Do you know if there's any other record of
7 that other shot in the records that have been filed in
8 his case?

9 A I don't.

10 MS. ESPOSITO: Okay. Thank you very much.
11 No further questions.

12 SPECIAL MASTER CAMPBELL-SMITH: Any
13 redirect?

14 MR. POWERS: Yes, just very, very briefly,
15 Special Master.

16 SPECIAL MASTER CAMPBELL-SMITH: Thank you.
17 I have a few questions as well, but please.

18 MR. POWERS: Okay.

19 REDIRECT EXAMINATION

20 BY MR. POWERS:

21 Q Mr. Mead, the care and treatment you
22 received from Dr. Green, would you describe that as
23 outside the mainstream of medicine?

24 A Not being a doctor myself I can't tell you
25 whether it is or is not. What I can tell you is

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1 having known Dr. Green and having known a number of
2 other physicians that are following a similar
3 protocol, that's got a variety of different nicknames
4 to it. I know that he has personally, and they have
5 received a lot of criticism for that and have been
6 portrayed as being outside the mainstream.

7 What I will also tell you is that everything
8 that has happened for William has been done, as far as
9 I can tell, safely. So if he's gotten chelation, it's
10 been applied safely and everything that's been done
11 medically. I can say this as a former medical
12 malpractice attorney, I'm not going to send my kid to
13 a quack, I guess is the shorthand of that.

14 Q So then William never had an adverse
15 reaction that you would attribute to any of the care
16 and treatment provided by Dr. Green?

17 A Absolutely not. What I can tell you without
18 overdramatizing it is we owe where William is today to
19 John Green and to the doctors that were brave enough
20 to do what they needed to do. John Green has
21 personally taken a real beating.

22 He's practicing medicine, and he has over
23 1,000 patients at this point internationally, and he's
24 received in other countries, like Italy, and all over
25 the world, but, you know, a prophet is without honor

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1 in his own land. He has really received a lot of very
2 direct and adverse publicity, and even though he's
3 done that, he's gone forward with it. We owe William
4 to him. It's that simple.

5 Q Is William better off now than he was when
6 he began treating with Dr. Green?

7 A The election question. Yeah. I'm being
8 flip. My kid jumped into bed with me three weeks ago
9 and asked to read a book with me and he's done it four
10 times in the last four weeks. This is a kid that they
11 said would never talk and would be in an institution.
12 We've done all of this stuff, the low-dose naltrexone,
13 the chelation, and all of it has without a doubt in my
14 opinion contributed to where William is now.

15 MR. POWERS: All right. Thanks.

16 SPECIAL MASTER CAMPBELL-SMITH: Ms.
17 Esposito?

18 MS. ESPOSITO: No recross.

19 SPECIAL MASTER CAMPBELL-SMITH: Thank you,
20 Mr. Mead. Just a few questions that I have that are
21 sort of follow-on -- points -- some of the questions
22 that Ms. Esposito asked. I'd like to begin with did
23 you generally accompany or who generally took William
24 to his doctors' appointments and would have been the
25 person to provide the medical history?

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1 THE WITNESS: That's a very good question.
2 I would say as far as Dr. Wittkopp is concerned,
3 probably 80 percent of the time, his pediatrician, up
4 until two years of age, his mom did the lion's share
5 of that. I was there for a few. After William got
6 sick and we embarked on this together, I think I was
7 there 85 to 90 percent of the time.

8 We were there together with Dr. Green, we
9 were there together with Dr. Walsh, Dr. Buie. We made
10 those trips together because it was something that we
11 felt pretty passionately about. So with that
12 timeline, the early part of the medical history it's
13 certainly coming mostly from his mom and then later on
14 it's coming from either one or both of us.

15 SPECIAL MASTER CAMPBELL-SMITH: Early part
16 of his history. Would you characterize that as less
17 than two years?

18 THE WITNESS: I would say from the time
19 obviously he was born, his early admissions and up
20 through his well baby visits were something that his
21 mom was doing, although interspersed with that, when
22 we're going into Columbia Memorial, we're going into
23 St. Vincent's for the Albuterol, I'm there for that
24 and for a number of those admissions as well.

25 So for the first two years when he is going

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1 to his standard well baby visits, his mom is doing
2 that, and when he's having his coughing, his sickness,
3 his ear stuff, I'm doing it, his mom may be going with
4 me, we may be going back and forth.

5 SPECIAL MASTER CAMPBELL-SMITH: You mention,
6 and this was one of my questions, his ear sicknesses
7 and his medical records reflect that not only did he
8 have a difficult time with this and was treated with a
9 number of antibiotics, he experienced an eardrum that
10 burst early on and ultimately was diagnosed with
11 asthma.

12 THE WITNESS: The short answer is yes. I
13 don't recall that there was a specific diagnosis of
14 asthma. It was, frankly, one of my frustrations on a
15 completely aside from what we're, that we were dancing
16 around. It was called bronchiolitis, it was called
17 persistent upper respiratory disease, and nobody was
18 going to call it asthma.

19 I'm not sure why, but it had a lot of
20 different names. We were treating it with Albuterol
21 and he seemed to be chronically ill as far as his
22 breathing was concerned.

23 SPECIAL MASTER CAMPBELL-SMITH: And I
24 understand as well that William manifested an adverse
25 reaction to steroid treatment.

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1 THE WITNESS: Yes, I think he did.

2 SPECIAL MASTER CAMPBELL-SMITH: Okay. Some
3 other questions that I had. Ms. Esposito asked you
4 when you first noticed a problem in William, not
5 characterizing what type of problem, when you began to
6 suspect that something other than his upper
7 respiratory problem might have been a problem, when
8 would you date that?

9 You indicated that your first suspicion that
10 something other than his upper respiratory illnesses
11 was a problem with his hearing.

12 THE WITNESS: Right.

13 SPECIAL MASTER CAMPBELL-SMITH: What was it
14 that caused you to believe he was having hearing?
15 That was the change in the speech?

16 THE WITNESS: No, and thank you for asking
17 the question. Not to qualify it, but you have to
18 understand that going back on hindsight, 20/20, I have
19 to kind of pull this out. What I would say is he
20 stopped responding to us during that summer.

21 SPECIAL MASTER CAMPBELL-SMITH: Which
22 summer?

23 THE WITNESS: The summer of 2000. In other
24 words, up until his second birthday, and up until May
25 of 2000, if you had said, hey, Willie, let's go

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1 outside, he would have turned around and looked at
2 you, which is classic, you know, that's how a kid
3 reacts to you.

4 After his second birthday, after those shots
5 and when he got sick, he wouldn't respond to his name,
6 he wouldn't respond to people, you know, hey, William,
7 or noises. He just kind of became focused. That's
8 when we began to suspect that it was his hearing
9 because he wasn't responding to his name in that time
10 period.

11 SPECIAL MASTER CAMPBELL-SMITH: You also
12 indicated, and the medical records do reflect, your
13 complaint that William began to show gross motor
14 problems. Do you recall about when you first began to
15 notice the gross motor problems?

16 THE WITNESS: During that summer. During
17 the summer of 2000.

18 SPECIAL MASTER CAMPBELL-SMITH: During the
19 summer of 2000?

20 THE WITNESS: Yeah. He was walking by that
21 point but he was stumbling. You know, hey, buddy,
22 you're falling over a lot, you know? He'd stumble, he
23 hit his head a couple of times. It wasn't something
24 where I had turned to his mom and said I think he's
25 experiencing gross motor problems. It was the wheels

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1 were coming off is the best way I can explain it.

2 That he seemed to be stumbling, he wasn't
3 answering and we were kind of like, what's going on
4 here? The biggest problem for us was the hearing.
5 That would have been that summer.

6 SPECIAL MASTER CAMPBELL-SMITH: You've
7 indicated a couple of times during your testimony here
8 that you would defer to the medical records generally
9 for what has been written in the medical records at
10 the time --

11 THE WITNESS: I would.

12 SPECIAL MASTER CAMPBELL-SMITH: -- that
13 these events happened. I can draw your attention more
14 specifically. Let me get just for the reference. Do
15 you recall any instances when William was stumbling
16 and falling that really required medical attention?

17 THE WITNESS: Two that I recall. Again,
18 I'll defer to the medical records. There was one
19 episode where I recall that he had fallen down and
20 bumped his head, and, you know, in the kind of that's
21 what happens to kids and kids get one of those.
22 Eleanor has one under here.

23 I think that was someplace between the year
24 and the year and a half is my memory that he fell and
25 he bumped his head. Then there was another where he

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1 had fallen down the stairs, and he cut his head and
2 received stitches. I'm trying to remember when that
3 was. It was 18 months, maybe a little bit after that.
4 Again, I don't have the medical records.

5 SPECIAL MASTER CAMPBELL-SMITH: These
6 instances to which you're referring, those are not
7 part of the times that you would describe as in
8 hindsight seeing William with altered gross motor
9 skills?

10 THE WITNESS: No. Again, to me the before
11 and after is William was a kid that was wrestling with
12 his bronchial stuff, and his ear stuff, and was kind
13 of motoring around and in hindsight had lost some of
14 his language and skills before May of 2000. The kid
15 that emerged over the course after May of 2000, as I
16 said, the wheels came off.

17 He was stumbling around, he may have fallen
18 down a couple of times beforehand, but, I mean, he was
19 sitting down, rocking. I venture to say he almost
20 wasn't even walking. He was spending a lot of time
21 jumping and kind of rocking. That's what I would say,
22 is after May and into early June and July is my memory
23 is that's when the gross motor stopped.

24 It got worse as we went into the fall so
25 that by the time we arrived in November of 2000 he was

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1 having substantive problems walking on a beam and
2 stuff like that.

3 SPECIAL MASTER CAMPBELL-SMITH: You
4 indicated there came a period of time where William,
5 the fruit bowl was one of his favorite things to eat,
6 and notwithstanding the fact that he was eating quite
7 a bit he was having diarrhea. Do you recall what his
8 favorite foods other than the fruit bowl were that he
9 was eating about this time, and would you remind me
10 what time that was?

11 THE WITNESS: The episodes with the fruit
12 bowl is really the end of the summer of 2000 into
13 October, so August, September and October. Again,
14 William, so he was getting the bloating and we would
15 get these fruit bowls. The other thing he liked was
16 he liked cereal a lot.

17 This is before we went on the casein and
18 gluten-free diet. So eating Cheerios, I think he had
19 a lot of Goldfish. He would eat, you know,
20 hamburgers, hot dogs and things of that nature when he
21 was not eating, but the fruit bowl was especially
22 remarkable because it was something aside from meal
23 time he was eating all of the time and going through
24 that a lot. The hives was something that happened in
25 that time period, too.

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1 SPECIAL MASTER CAMPBELL-SMITH: During the
2 later part of the summer?

3 THE WITNESS: Yeah.

4 SPECIAL MASTER CAMPBELL-SMITH: Mr. Mead,
5 you indicated based on my questioning that William had
6 stopped responding to his name and you began to notice
7 differences in the way he would say a word and there
8 would be some sort of babbling or nonsensical
9 utterance in connection with the word.

10 You said there was no clear period of time,
11 that you recall it was sort of a gradual fading away
12 of language.

13 THE WITNESS: Right.

14 SPECIAL MASTER CAMPBELL-SMITH: Did you
15 report this to the pediatrician or your or your family
16 member report a loss of language or noticed difference
17 in language to a pediatrician, and when might you have
18 done that?

19 THE WITNESS: Well, and I think I know that
20 there's a note in there that I do remember seeing that
21 in May, and it was not me, I think it was his mother,
22 reported that he was not talking. It was in May of
23 2000. The note says no language or he's not talking
24 or no language.

25 I think that he, again, had some small

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1 language but what was replacing it was the ta-ka-ta-
2 ka-ta-ka-ta-ka-ta-ka thing. So the point is I think
3 that we, we being me and probably Tori, reported that
4 in May of 2000.

5 SPECIAL MASTER CAMPBELL-SMITH: Thank you.
6 Mr. Mead, you indicated that there was a suggestion
7 based on laboratory results that had come back that
8 informed you and that made you suspect that William
9 had these high mercury levels.

10 THE WITNESS: Uh-huh.

11 SPECIAL MASTER CAMPBELL-SMITH: Was there
12 any time that perhaps you or your wife inquired
13 independently and asked a question about -- well, let
14 me back up. When was that Oasis conference? Was that
15 in December?

16 THE WITNESS: No, that was in October.

17 SPECIAL MASTER CAMPBELL-SMITH: October of?

18 THE WITNESS: 2000.

19 SPECIAL MASTER CAMPBELL-SMITH: 2000, okay.

20 And when did you receive the lab reports?

21 THE WITNESS: Late January of 2001.

22 SPECIAL MASTER CAMPBELL-SMITH: Was there
23 any time prior to receiving those reports that you
24 recall having expressed some concern about heavy metal
25 toxicity or autism-related conditions to your

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1 physician?

2 THE WITNESS: I'm thinking.

3 SPECIAL MASTER CAMPBELL-SMITH: All right.
4 I understand.

5 THE WITNESS: I don't recall and the records
6 may belie this, but I don't recall. I remember the
7 coming back, that there had been a lot of discussion
8 about mercury.

9 SPECIAL MASTER CAMPBELL-SMITH: I'm sorry,
10 coming back from Oasis?

11 THE WITNESS: Oasis. That there had been a
12 lot of discussion about that. What I also recall was
13 that William's pediatrician's office candidly
14 abandoned us basically. When we went to Dr.
15 Wittkopp's office to try and get some real answers
16 about what the shots had been, what was happening to
17 William, why this was happening, we weren't getting
18 answers.

19 So what we did in that period of time is we
20 were in hyper mode. We got Dr. Pang onboard because
21 he was a pediatrician who was willing to look at what
22 was going on with William biomedically. I don't
23 recall whether we actually raised the mercury issue
24 with Dr. Pang before we had the panel done or not.

25 In other words, we may have said, well,

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1 people have said that there's this mercury problem,
2 but the first time that William, we actually looked at
3 him for that, was the panel in January of 2001.

4 So I don't think we raised it with Dr.
5 Wittkopp because I think Dr. Wittkopp was frankly
6 really not interested in seeing us as a patient
7 anymore. So we looked at Dr. Pang and went in there.
8 I don't know whether we raised it with Dr. Pang until
9 we got to see Dr. Green. I don't know.

10 SPECIAL MASTER CAMPBELL-SMITH: One
11 additional question just in terms of getting this
12 timeline straight. You indicated that your parents
13 had drawn to your attention and you had begun to
14 notice some things earlier in the summer, in July, and
15 the medical records reflect, and you alluded to during
16 your testimony, that there was some question at the
17 mommy and me program about William fitting in to the
18 program.

19 THE WITNESS: Uh-huh.

20 SPECIAL MASTER CAMPBELL-SMITH: When was
21 that issue drawn to your attention, and what
22 precipitated the beginning to focus on the Oasis
23 material? I understand that it came in the mail and
24 your former wife thought that might be a --

25 THE WITNESS: Okay. If I understand your

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1 question correctly, how did we go from wondering about
2 his hearing problem and looking at this in the summer
3 to the Oasis conference and getting plugged into this
4 thing.

5 SPECIAL MASTER CAMPBELL-SMITH: Yes.

6 THE WITNESS: The short answer is that it
7 was a series of circumstance. When we went to the
8 audiology clinic we were told that the diet would
9 help, and we were given the names of a couple of
10 people, one of whom was a woman named Meghan Paquin,
11 who is the mother of an autistic child.

12 She took us under her wing for a period of
13 about five weeks because she knew all about the diet.
14 She said this is how you do the diet, this is what you
15 eat. We said we're having a lot of trouble with Dr.
16 Wittkopp, and we can't get answers from Wittkopp, and
17 we're desperate, and we're scared because this is now
18 into after the audiology clinic. This is in
19 September.

20 She said, well, we go to Dr. Pang, and let
21 me see if I can get you in to see Dr. Pang. So Meghan
22 also I think was the one who told us about you need to
23 go to the Oasis conference. My response was, you
24 know, I don't want to do that. Tori said, well, I
25 think we should do it, and she ended up going. So

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1 Meghan was the one who got us in to see Dr. Pang, Dr.
2 Pang made the referral to Dr. Green.

3 Meghan was the one who, you know, basically
4 turned us on to the Oasis conference and put us onto
5 the gluten-free, casein-free diet, and then promptly
6 moved to Alaska, so it was like thanks a lot. That's
7 how that happened.

8 SPECIAL MASTER CAMPBELL-SMITH: Thank you.

9 THE WITNESS: Thank you.

10 SPECIAL MASTER CAMPBELL-SMITH: Thank you
11 very much. Have my questions precipitated any further
12 questions from counsel?

13 MR. POWERS: No, they did not, Special
14 Master.

15 MS. ESPOSITO: No. Thank you.

16 SPECIAL MASTER CAMPBELL-SMITH: I thank you
17 very much, Mr. Mead.

18 THE WITNESS: Thank you.

19 SPECIAL MASTER CAMPBELL-SMITH: You're
20 excused.

21 (Witness excused.)

22 SPECIAL MASTER CAMPBELL-SMITH: Well, it
23 appears we're close to a full day today. My perhaps
24 slightly fast indication is we're just a little bit
25 shy of 5:00, and it is my understanding that we were

1 going to recess for the day and to return in the
2 morning to hear from Petitioners' next witness. That
3 would be a member of the King family, Ms. King?

4 MR. POWERS: We would still prefer to do
5 that rather than risk yet again having people go out
6 through the basement garage. We'd prefer to do that.
7 Ms. King has made herself available tomorrow, Dr.
8 Mumper is available tomorrow. If for some reason
9 things did spill over, as you all know, we still have
10 Friday available, too, with our witnesses.

11 SPECIAL MASTER CAMPBELL-SMITH: With that
12 said, I think we are adjourned for the afternoon.
13 Thank you.

14 ALL: Thank you.

15 (Whereupon, at 4:47 p.m., the hearing in the
16 above-entitled matter was adjourned, to reconvene at
17 9:00 a.m. on Thursday, May 15, 2008.)

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REPORTER'S CERTIFICATE

DOCKET NO.: 03-584V and 03-215V
CASE TITLE: Claims for Vaccine Injuries
HEARING DATE: May 14, 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 14, 2008

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