UNITED STATES
COURT OF FEDERAL CLAIMS

TIMOTHY AND MARIA DWYER, PARENTS OF COLIN DWYER, A MINOR,	)	
Petitioners,	)	
v.	)	Docket No.: 03-1202V
SECRETARY OF HEALTH AND HUMAN SERVICES,	)	
Respondent.	)	

REVISED AND CORRECTED COPY

Pages: 89 through 337

Place: Washington, D.C.

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SECRETARY OF HEALTH AND HUMAN SERVICES,	)	
Respondent.	)	

Courtroom 6, Room 507
National Courts Building
717 Madison Place NW
Washington, D.C.

Tuesday,
July 22, 2008

The parties met, pursuant to notice of the Court, at 8:00 a.m.

BEFORE: HONORABLE DENISE VOWELL
Special Master

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C O N T E N T S

E X H I B I T S

PETITIONERS' EXHIBITS:	IDENTIFIED	RECEIVED	DESCRIPTION
20	251	--	Article, An Open Label Trial of Esataloprine In Pervasive Developmental Disorders
21	281	--	Mapping Autism Risk Loci Using Genetic Linkage and Chromosomal Rearrangements

E X H I B I T S

RESPONDENT'S EXHIBITS:	IDENTIFIED	RECEIVED	DESCRIPTION
13	158	--	Immunosciences Lab data re testing for autism
14	165	--	7-17-07 letter from Vojdani re Immunosciences Lab
15	167	--	6-16-06 letter from Vojdani re Immunosciences Lab
16	169	--	1-6-06 notice of sanction action against Immunosciences Lab
17	170	--	CLIA Annual Laboratory Registry, 2005
18	171	--	6-7-05 letter from California Department of Health Services re conditions not met
19	175	--	Immunosciences Lab settlement agreement

P R O C E E D I N G S

(8:00 a.m.)

THE COURT: We're back on the record in the matter of Dwyer v. Secretary of HHS.

Mr. Powers, you have the floor this morning.

MR. POWERS: Yes. Good morning, Special Master. The Petitioner at this point would like to call to the stand Dr. Elizabeth Mumper, M.D.

Special Master, being on the record and as Dr. Mumper approaches the stand, just one perhaps a little bit more than a housekeeping matter, but I conferred with counsel for Respondent, and understanding the time and the scheduling pressure that we are under today the parties have agreed explicitly that from Dr. Mumper's testimony in the King and the Mead cases there was approximately 50 to 55 minutes of testimony that was non case-specific, background testimony.

I have identified the pages in the transcript that would capture that testimony. The agreement is, and again I think it has been implied that the transcripts would be filed in the Dwyer case, but I wanted to designate those pages specifically so that we can cover some of that material in five minutes here rather than doing it again for 55 minutes.

THE COURT: Exactly. And since I was present during that testimony in the Mead and King cases, I've heard it before.

MR. POWERS: Yes. And if there is a beauty to doing the omnibus proceeding and having all three of you sit in each of the test cases, this is an example of how that should work.

And so just for the record then if I could, Special Master?

THE COURT: Please.

MR. POWERS: The pages we would be looking at would be from Day 4 of those proceedings. It would be Day 4, which was May 15, and the transcript pages are 1187 through 1228.

We would just again notify the Court and the parties that we'll be designating those pages and filing them as an exhibit here and incorporate that testimony by reference in Dr. Mumper's comments today.

THE COURT: And it is possible that those page numbers themselves may change as you all go through the process of making corrections to those transcripts, but based on the transcripts that are currently filed we'll use those page numbers.

MR. POWERS: Thank you, Special Master.

Good morning, Dr. Mumper.

DR. MUMPER: Good morning.

THE COURT: Let's go ahead and swear her in this case, even though she was sworn in the omnibus proceeding.

Dr. Mumper, you've got your right hand up.

Whereupon,

ELIZABETH MUMPER

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

DIRECT EXAMINATION

BY MR. POWERS:

Q Now I'll say good morning, Dr. Mumper.

A Good morning.

Q And for the sake of the record and the court reporter, could you spell and say your full name for the record?

A Elizabeth Mumper, E-L-I-Z-A-B-E-T-H, M-U-M-P-E-R.

Q Are you a doctor?

A Yes. I'm a pediatrician.

Q How long have you been a pediatrician?

A Since 1983.

Q Now, you heard the discussion just a moment ago about the testimony that you gave in the King and Mead matters. Do you recall that testimony in May of

this year?

A Yes.

Q Do you recall at the beginning of that testimony spending a fair amount of time talking about your background and skills and training?

A Yes, I do.

Q To summarize, I just wanted to have you describe in this case your professional experience and take us all the way from medical school to today, your professional experience.

A Medical school at Medical College of Virginia in Richmond, internship at the University of Massachusetts, residency at the University of Virginia. I was asked to be chief resident, which involved an extra year, in pediatrics.

I spent five years in private practice, 11 years teaching in a residency program, and since 2000 I have had a private practice that is partially general pediatrics and partially dealing with children who have developmental and behavioral problems.

I'm also the medical director of the Autism Research Institute and the clinician in charge of the physicians' training, the clinicians' seminars for Defeat Autism Now.

Q What's the name of your private practice?

A The Rimland Center.

Q So your three professional positions right now would be director of the Rimland Center, correct?

A Yes.

Q And director of the Autism Research Institute?

A Yes.

Q And then the third would be the medical director for Defeat Autism Now?

A Right. And that very much goes with the Autism Research Institute responsibilities.

Q So the responsibilities in those two positions do overlap?

A Yes. That's correct.

Q Did you prepare an expert report in this case, Colin Dwyer's case that you're here to testify about?

A Yes, I did.

Q When you prepared that expert report, what materials did you have available to you that you relied on to prepare that report?

A At that time I had a number of medical records, not the actual complete medical records, and at that time I also had information from various labs and physicians.

I had a rough draft of the mother's affidavit and other materials as forwarded by your law firm.

Q Did you also have available to you the materials that you relied on to prepare a report and offer testimony in the King and Mead cases?

A In that I had done them I did not physically have them present with me at the time I prepared the report.

Q Would it be fair to say that you relied on those materials in preparing your report?

A Yes.

Q Since preparing your report and appearing here today, is there any additional information that you have available that you used that you're going to rely on in your testimony?

A Several things. One is that I received more medical records after the filing of my report. Specifically of interest were some even-number pages from Dr. Bock that I did not have the first time due to a scanning error.

I also had the opportunity last night to listen to the parents' testimony on audio and to meet Mrs. Dwyer.

Q When you say you listened to the parents'

testimony on audio, you're speaking about Maria and Timothy Dwyer's testimony?

A That's correct.

Q So you were not here in person to hear their testimony, but you heard the audio download?

A That's correct.

Q Also since you testified in May, are there any additions to your curriculum vitae that would be relevant to your skills and qualifications to testify?

A Other than being invited to attend an autism think tank in mid June and several lectures in Italy, no.

Q And when you say several lectures in Italy, are those lectures that you've already done?

A Yes.

Q What was the subject of those lectures in Italy?

A Essentially I was talking about the use of assessment of medical problems in children with autism to guide therapeutic decision making.

Q Were you invited to give those to medical professionals?

A Yes. I was invited by a medical professional. The audience was medical professionals and also parents.

Q Finally then, since your testimony in King and Mead you mentioned that you were invited to a think tank. What think tank is that, and what's the subject matter that's going to be discussed at the think tank?

A Well, actually we already were at the think tank. They invited 30 leading clinicians in the autism field and researchers in autism from around the world to participate at a think tank at Ratinaling in California.

The subject of it was to get an update on the advances in the research and try to coordinate that with the clinicians' work in order to move the autism treatment and research agendas forward because we face such an overwhelming number of these children.

Q So what you're describing and certainly what you testified to in the King and Mead cases, would it be fair to say that a significant portion of your practice is devoted specifically to neurodevelopmental disorders, including autism?

A That's true.

Q Approximately how many children do you see in a given year in your pediatric practice? Just approximately.

A Oh, gosh. About 1,750, 1,700.

Q And do you have a rough sense of what percentage of those children that you see in your practice are children with autism spectrum disorders?

A The percentage in numbers is lower than the amount of time I spend with them because they're more time consuming. About half of my time is spent taking care of children with autism spectrum disorders.

Q So it would be fair to say that's a significant focus of your professional practice and your professional training and professional background?

A That's true.

Q Now let's shift gears a little bit and start speaking specifically about Colin Dwyer's case.

A Okay.

Q You did mention that you saw his medical records in the course of preparing your report. Is that correct?

A That's correct.

Q Could you describe in general the impression you had of Colin Dwyer's overall physical health from birth through the age of one year based on your review of his medical records?

A It seemed that he was a healthy baby who was the product of an uncomplicated pregnancy. He did not

have a difficult birth. He was healthy initially and had an uncomplicated newborn stay.

From reviewing his well-baby checkups, it seemed that he was actually quite healthy with only a few things that are typical in children in the first year of life like a little bit of a pink eye or eye infection, but nothing that would lead me to characterize him as a chronically ill child.

He seemed to be meeting his developmental milestones as recorded by his pediatricians on his well-baby checkups.

Q When you say recorded by his physicians on the well-baby checkups, what specific developmental milestones do you recall seeing in the medical record that lead you to conclude that he was meeting his developmental milestones?

A Well, at each of his well-baby visits the pediatricians ask questions about development in several domains -- gross motor skills, fine motor skills and language typically.

As we went through the first year of his life, the pediatrician documented that he was doing things such as rolling over, sitting up, crawling, cruising and walking, and actually in terms of gross motor development he was actually a little bit

precocious in that he was walking by nine months and walking very well by a year.

In terms of his language development, they talked about him initially smiling and being responsive and making eye contact as early as seven weeks of age. They talked about him having babbling and then developing three to five words by his one-year checkup.

Three to five words is really very good for a child at one year. We typically expect them to have momma or dada, usually dada, plus one or two other words and so that seemed to me to imply very normal language development at that time.

Q In fact, let's take a quick look at one of the medical records. This would be Exhibit 1, page 70.

A I have it.

Q We'll pause, Dr. Mumper, because we want to get it up on the screen here so that the Special Master and counsel can be able to see this too.

Take a look at your monitor there, Doctor, and let's make sure that the paper you have in front of you is the same thing you see on the screen. Is that the same thing?

A Yes.

Q Do you recognize that document?

A Yes. It appears to be the one-year checkup for Colin Dwyer.

Q There is a highlighted section about one-fourth of the way down on that page. Can you describe for the Special Master what that highlight is and why it's significant to you?

A The highlight is looking at the classic developmental milestones. The first is language, and the pediatrician recorded that he was saying three to five words, which is normal for age.

The second word, which is pincer, refers to pincer, being able to pick up objects or bring the thumb and index finger together. That's also a classic one-year-old milestone that you would like to see the child exhibit at that time.

And then they wrote exploring, which means that this is a child who had gross motor skills enough that he was able to actually explore his environment.

Q So based on this chart note from his one-year well-baby visit, do you see anything at all on that chart indicating that there were any delays or problems with his health or development?

A No, I don't. In fact, the pediatrician actually documented that he or she heard babbling and

an occasional word, so this wasn't just based on the parents' history.

Q And speaking of the parents' history, you did hear, you mentioned earlier, via the audio download the testimony of Mr. and Mrs. Dwyer that was given yesterday, correct?

A Yes, I did.

Q Did you hear anything in their testimony that was inconsistent with the description of Colin's health, well-being and development up to the first year of life from the parents?

A No, I did not.

Q Let's start moving into the second year of life. We're going to put another exhibit up for you, Doctor. This would be Exhibit 1, page 67.

A I have it.

Q And we'll get it up on the screen. I think we will quickly have a routine for handling the exhibits.

I want you to take a look at your monitor and confirm that what you see there is the same thing on the paper. Is that correct?

A Yes.

Q Do you recognize that document?

A The 15-month well-baby checkup for Colin

Dwyer.

Q What on that well-baby checkup doctor's note do you feel is significant? If you could explain that to the Special Master?

A The pediatrician documents developmental milestones which includes the phrase: Talking some, running, climbing, problems sleeping like brother.

Q You mentioned talking some. Is that note significant to you as a pediatrician?

A My interpretation is that the pediatrician was documentating that the child was talking and that that implied that he was having normal talking at that time.

I would expect if there were concerns that he or she would have said something like not talking enough or language delayed or words less than expected. So the fact that he was talking some I view as a recording of a normal milestone.

Q Again, anything in this record that would indicate that Colin Dwyer was developmentally delayed?

A No. I do not see evidence for that.

Q Also again referring to the parents' testimony, you recall there was testimony about his development up through 15 months.

Is there anything in the parents' testimony

that you listened to that's inconsistent with this history of meeting milestones and generally being in good health?

A No, there was not.

Q In your review of the medical records and review of the other material specific to Colin's case, did you see anything in those records before the age of 20 months indicating that he was failing to meet any developmental milestones?

A No, I did not.

Q Did you hear anything in the parents' testimony that indicated to you as a medical doctor that Colin was missing developmental milestones before the age of 20 months?

A No, I did not.

Q At some point did you see anything in the record indicating that Colin might not be meeting a developmental milestone?

A The well-baby visit of 20 months on July 10, 2000.

Q Let's go ahead and take a look at that. I assume there is a medical chart note on that, Doctor?

A Yes.

Q That would be Exhibit 1, page 63.

A That's correct.

Q That's on the computer monitor there on the witness stand. Take a look at the monitor, and let's just make sure we're all looking at the same paper.

A Yes.

Q Do you recognize the document that's on the screen?

A Yes.

Q What is that document?

A The 20 month well-baby checkup for Colin Dwyer.

Q Now, you mentioned this was the first indication that you saw in the record of a possible developmental delay. Can you point that out in the note to the Special Master and explain the significance?

A There are a couple of notations. One is about a fourth of the way down the page where the record denotes that he says a few words, and in parentheses it says three to five.

That would be the expected language for a child somewhere in the range of one year to 14 months of age, so the quantification of the words there suggests a delay.

Then under the Impression there is a notation about the speech, and in the Plan they

suggest a follow-up at two years, but make the notation to check the speech at two years.

Q What is the significance of those notes to you? What does that tell you as a medical doctor who treats children? What does that tell you about Colin's developmental course at the age of 20 months?

A It suggests that the pediatrician is starting to be concerned about the child's language, but that they were not panicked at his lack of language such that they instituted any kind of emergency evaluations.

Q Again, similar questions to what we covered before. You did listen to the parents' testimony yesterday. Anything in their testimony that is inconsistent or at odds with the information you see in the exhibit that you just described?

A No, there was not.

Q So in summary then, Doctor, from birth up to 20 months do you have an opinion about Colin's developmental progress?

A It seems that he was meeting developmental milestones at least as documented by the 15-month checkup.

At the 20-month checkup, even though gross motor skills and fine motor skills such as eating were

normal, there was an initial concern about speech. So it is difficult for me to pinpoint the exact time that the speech may have become a problem, but by 20 months of age it's raising a flag for the pediatrician.

Q And there is certainly nothing before 15 months? In fact, up through 15 months it would be fair to say the record reflects more likely than not that he was meeting his language milestones?

A That is correct.

Q I want to back up for just a moment, back up chronologically. You talked about Colin's birth.

A Yes.

Q Did you also have a chance to review his birth records? If you recall, these were records that only became available fairly late in the proceeding and certainly after your report. Did you get a chance to look at the birth records in particular?

A Yes, I did.

Q You touched on your review of his birth and delivery earlier. I want to go back to that topic.

Did you see anything in his birth record indicating that he was unhealthy or in distress during labor, delivery and birth?

A No, I did not, but I did see one important finding when I got the birth records that I did not

include in my report. It appears from the records that he actually received a Hepatitis B vaccine at birth, which was not reflected on my initial report.

I would not have expected that because in the report that I had there was documentation that he had gotten a Hepatitis B vaccine at 13 days of age and then another one at seven weeks and then another one at about six and a half months.

The initial series for Hepatitis B is typically three vaccines. Some hospitals give Hepatitis B on day one. Our hospital many years ago decided not to do that and so our local pediatricians typically give Hepatitis B either at one or two months and start the three shot series then.

The reason that I'm concerned about this is that it actually means that he got three injections of Hepatitis B vaccine prior to two months of age, which is a time that I perceive as special vulnerability, particularly with regards to handling potential toxic insults.

And so the thimerosal that he received on day one of birth plus what he received at 13 days and seven weeks makes me relatively more concerned than I initially reflected in my first report.

Q Do you have the report handy with you, Dr.

Mumper?

A Yes.

Q Because I just want to be as clear as we can in discussing the facts in your report. If you would turn to page 3?

A Right.

Q You'll see that the shots that Colin received are listed, and the ethyl mercury content per shot is listed. Do you see that sort of in the middle of the page?

A Yes.

Q I want to make sure we capture what you're saying. You're saying that the Hepatitis B in your report shows three different immunizations, and you're saying there would actually be one additional immunization that was given on November 10?

A That's correct.

Q And that would then change the total mercury exposure from 237.5 micrograms to 250? Is that correct?

A That's correct.

Q Okay. Did you see anything in the medical record indicating that Colin's mother, during the time that she was pregnant with Colin, was exposed to anything that might be associated with the appearance

of autism in a child that a woman would be carrying to term?

A No, I did not see it in the record, and I actually asked about that also last night and did not perceive any risk factors based on both the written record and her report.

Q What risk factors were you considering and looking at and then ruling out? Can you describe those for the Special Master?

A We're concerned about potential illnesses of the mother during pregnancy, especially viral illnesses such as influenza or rubella.

We're concerned about potential exposure through medications, specifically valproic acid, thalidomide, terbutaline, and in asking her about those medications last night and reviewing the records there was no history that she received any of those.

She also did not give a history of having an unhealthy pregnancy. In fact, her husband referred to her several times as a healthy girl during the pregnancy.

Q And from your review of the medical records, listening to the testimony and speaking with Mrs. Dwyer in person, is there anything to indicate that she was smoking cigarettes during her pregnancy?

A No. She specifically told me she did not.

Q That she was consuming alcohol during her pregnancy?

A No.

Q Now we're going to go back in time and catch up with where we were chronologically. We spoke about the chart notes at 20 months. I want to then start moving forward in time from that 20 months.

What was the next thing that you saw in the medical record that you recall that indicated that Colin Dwyer may have developmental delays of some sort?

A At a visit marked 3-22-01 -- it was marked as a two-year checkup; he was actually a little more than two years at that time -- I initially thought this was just a routine checkup, but I realized last night that the parents actually had concerns at that time that they brought to the attention of the pediatrician.

Q Let's go ahead and get that medical record on the computer monitor. I'm going to ask you a couple of questions about that. This is going to be Exhibit 1, page 60.

A I actually think it's page 61 maybe.

Q Let's try page 61. My apologies. What you

see on the monitor now, is that what you have on the page in front of you that you were describing?

A That's correct.

Q Okay. If you could explain for the Special Master what this document is and what you find significant?

A This is the record of his two-year checkup, and in the record it's reflected under Neurologic Exam speech/language delay, and there appears to be an exclamation point after that.

The pediatrician goes on to form an impression that the child had a speech/language delay, and under the Plan said to EI, which is an abbreviation for early intervention, for speech.

They go on to say: I stress the importance of the evaluation or I stress the importance. I'm adding of the evaluation.

Q Now, you're describing that language specifically. I'm assuming it's significant language to you as a professional pediatrician. Is that correct?

A Yes. That's correct.

Q Why is that language significant, particularly compared to the note at 20 months?

A To be stressing the importance of an early

intervention evaluation and to put an exclamation point after the words speech and language delay imply to me that this person was very concerned about the lack of speech.

And I suspect that they looked back in the record and saw normal language at a year and then listened to the parents' history where the child was actually losing language milestones and losing words in addition to not continuing to progress. That to us is a very big red flag to look for problems.

Q Among the problems that one would look for when you see this kind of red flag, is regressive autism one of those problems?

A Yes, it is.

Q In your review, and we can pull this down unless, Doctor, is there anything else in that note that you wanted to describe for the Court?

A No.

Q Okay. In your review of the records and talking to Mrs. Dwyer, listening to the parents' testimony, do you have an opinion about whether Colin Dwyer regressed into autism at some point in his life?

A I do believe that he regressed into autism.

Q Can you tell the Court what that opinion is based on just in general now that we've gone through

the chart details?

A Finding numerous documentation, both in the medical record and by parent report, of normal developmental milestones and then finding clear documentation in the medical record and parental concern about a loss specifically of language milestones and also documentation of behavioral changes.

Q Do you recall what behavioral changes stuck out for you that were significant that inform your opinion that Colin regressed?

A It was quite dramatic to listen to the parents describing this child, who would go around the streets of New York in a stroller and be very interested in his environment and have play with his brother and appropriate social interactions and then change into a child who did not want to sit in the stroller, who started having tantrums, who withdrew from social interactions that he had particularly enjoyed previously;

Who went from doing very creative play with blocks where he would enjoy building structures and then knocking them down, which is very age appropriate behavior for a toddler, to very rigid play where he took the blocks and lined them up and became very

upset if the organizational structure was disrupted.

He went from a child who responded positively to seeing his mother come home from work and expressing distress when she would leave to someone who wanted to withdraw and sit in the corner.

He went from a child who at his 13-month age at Christmas was interacting as you would expect a 13-month old baby to do and expressing interest in his environment to a baby who the next Christmas was withdrawing from interaction and ended up being a spectator to Christmas essentially.

So that very vivid clinical description as well-articulated by both parents in conjunction with a pediatric record that reflected normal development and then red flags makes me very concerned about a regressive picture.

Q And this regressive picture that you described in Colin, is that something that you've seen before in your professional practice?

A It is.

Q Is that pattern something that you term regressive autism?

A It is.

Q Is that a pattern of progress and regression something that you see reflected in the medical

literature as regressive autism?

A Yes. There have been several documentations in the medical literature trying to determine what percentage of children have regressive autism, how to clearly document that, but it is documented to occur.

Q In reviewing Colin's medical history and listening to his parents' testimony and speaking with Mrs. Dwyer, did you notice anything in the record suggesting that he had behavioral problems that began at about six months of age?

A I did not.

Q Do you also recall looking at his growth chart from birth up through the age of two?

A Yes, I do.

Q Do you recall that at around six months his weight was about the 25th percentile?

A Actually, I have at about six months that -- let me make sure I'm looking at the right one. That his weight was at the 50th percentile at six months.

Q And then as you move forward does his weight percentile change from 50 percent, sort of the very middle of the bell curve?

A Yes, it does. At both 12, 15, 20 and 29 months it is around the 25th percentile, but clearly following that curve and not showing any further

drop-off.

Q And at the 25th percentile, is that within one standard deviation of the 50 percent mean?

A Yes, and it basically means that he weighs more than 25 percent of babies his age.

Q Would that be considered a normal weight for a child of his height and age?

A Yes, it would be.

Q And it would be because it's within that first standard deviation of the mean, correct?

A Right. When you look at where he initially started out on his growth chart, which was just a little bit above the 50th percentile, looking at the growth pattern overall it is within what I would consider normal.

Now, having said that, whenever we see a child starting to cross percentiles we try to evaluate possible reasons for that. Things could include not eating enough or being chronically ill or having chronic diarrhea and malabsorbing, so one would consider the potential medical problems and be alert to the possibility of either gastrointestinal problems or central nervous system problems or a wide variety of metabolic problems.

Q But certainly nothing in the medical record

indicating that during the period where he went from 50 percent to 25 percent, there's nothing indicating that he had behavioral problems in that period of time, is there?

A No. I did not see that. Now, at 12 months it was mentioned that he was having some sleeping problems similar to that which his brother had, and sleeping problems are very common in all children, including children with autism. And so that itself I would not consider a behavioral problem, but that was noted in the record.

Q Is there anything from the testimony of the parents that you heard indicating that Colin Dwyer had behavioral problems beginning at about the age of six months?

A No. I did not see any documentation of that.

Q So you've now described Colin up through the age of two years old.

A That's correct.

Q Now, at some point Colin did receive a medical diagnosis indicating that he might have an autistic spectrum disorder. Do you recall that?

A That's correct.

Q Okay. We're going to go ahead and put

Exhibit 1, page 55 to 56, on the computer screen there for you. Do you recognize that document on the screen?

A Yes. That is a neurologic consultation by Dr. Irving Fish, who was a pediatric neurologist.

Q How old was Colin at the time that he was examined?

A Well, the record shows two and a half. He was born in November, so he was two years and seven months.

Q Just over two and a half years old. The record speaks for itself here, but what's the significance of this document as you would explain it to the Special Master?

A The pediatric neurologist diagnosed him with a pervasive developmental disorder with significant autistic features.

Q Why is that significant to you?

A Because someone who is trained with expertise in pediatric neurology diagnosed him with an autism spectrum disorder at this time.

Q Is that the first point in the medical record that you saw any indication that he had an autistic spectrum disorder as a diagnostic suggestion?

A Yes, it is.

Q Okay. We can pull that document down and move forward a little bit in time.

Now, do you recall in your review of the medical records and from the parents' testimony that Colin was ultimately taken to a Dr. Bock?

A Yes, I do.

Q Are you familiar with Dr. Bock?

A Yes. Dr. Bock is an integrative physician who practices in New York, and I have co-lectured with him on a number of occasions, been at think tanks with him, had the opportunity to have personal conversations with him about medical problems in children with autism.

Q Now, in preparing your expert report there's a section that begins on page 4 of your report where you describe and ultimately rely on a series of laboratory tests and analyses, correct?

A That's correct.

Q These laboratory tests that we're going to talk about here in some detail, these were all ordered and supervised by Dr. Bock? Is that right?

A I think they all were. It's possible that there are one or two labs that were ordered by Dr. Russell. The labs came from both of those sources.

Q Okay. Let's go ahead and walk through some

of these labs. What I'm going to do, just so that you know, Doctor, is we will put specific pages up here that you referenced in your report, and I would like you to explain to the Special Master what those pages describe and why they're significant to you. That's going to be our general approach.

A Okay.

Q We'll try to move through these in a fairly brisk manner because they are captured in the report here. Let's start off with Petitioners' Exhibit No. 4, page 100.

A I have it.

Q And on the computer screen is that the document on the monitor that you have in front of you?

A Yes, it is.

Q What is that document, and why is it significant to you?

A This is a laboratory evaluation that is based on a blood test that is looking for antibodies against neurofilament. The reason that this is significant to me is that Colin at this time showed an elevation in neurofilament antibodies.

The other name for neurofilament antibodies has to do with glial fibrillary acidic protein, and with regard to our understanding of potential

causation based on thimerosal toxicity, this lab result lends credence to our concern because it's demonstrating these glial fibrillary acidic protein antibodies.

The GFAP is a way in which structure and integrity is provided to astrocytes, and with antibodies against those I am concerned about loss of structural integrity of the astrocytes and ongoing neuroinflammatory processes.

The significance of the test is to make us consider in the differential diagnosis of his problems that there may have been a toxic insult that is affecting the glial fibrillary acidic protein that suggests neuroinflammation or reactive gliosis.

That ties in to Dr. Kinsbourne's testimony about the neuroinflammation in children with autism and the pathology in the astrocytes and problems with the glial cells, et cetera.

Q Now, the reference range on this is given as I think it's zero to 50, and the level that's recorded here is 53.

A Right.

Q As somebody who looks at these results, how elevated is that, in your opinion?

A It's very mildly elevated because it's only

three units above the upper limit of normal.

I have seen children with this value as high as 63 I think, so I don't want to overstate that this is a huge elevation. I just want to use it as part of our composite picture that it is abnormal and that you would not expect a normal child without neuroinflammation to be showing these antibodies suggestive of neuroinflammation and reactive gliosis.

Q Let's go ahead then and move to page 102 of Petitioners' Exhibit 4.

A I have it.

Q Can you describe for the Special Master what that is and again why it's significant to your opinion in this case?

A This again is a blood test, and here the lab was looking for antibodies against myelin basic protein. This is significant to me because myelin is like an insulation in our nerve cells that's very important for speeding transmission of neurologic impulses. To be making antibodies against that potentially would lead to degeneration of the myelin sheath.

The most classic example of this is a disease called multiple sclerosis. Again, I would not expect a normal child without an autoimmune or

neuroinflammatory process to be making antibodies to his myelin basic protein in the first few years of life.

Q Now, again it's a level of 57, and the normal reference range, the upper limit of normal, would be 50. Can you describe as somebody who has reviewed these types of lab reports before how elevated that is?

A This is more elevated than the other test we just looked at. I do think this is a significant elevation. It's about 15 percent elevation I believe, if I did my math right, so I would take that very seriously.

Q We're going to move ahead then and look at Petitioners' Exhibit 4, page 96. Do you see that on the monitor there?

A Yes, I do.

Q Okay. Again, can you describe what this is and what it tells you and why it's significant?

A This is a blood test looking at oxidative stress. Oxidative stress is a big part of our theory of causation in that when children or adults are under oxidative stress it impacts their ability to handle heavy metal toxicity.

This is showing that his levels of reduced

glutathione, which is the good form of glutathione that is expected to help a person with detoxification, immune function, mitochondrial function, acting as an intracellular antioxidant and helping the gut epithelium was low, in the red zone if this happened to be in color.

The test also shows a quite elevated lipid peroxide measure. Lipid peroxides take it a step further and actually look at lipid damage by oxidants and excessive free radical activity. Free radicals are a cause of cellular damage, a cause of aging, and excess free radicals or loose electrons are a marker for oxidative stress.

So not only do we have low levels of the glutathione that has all those important functions that I mentioned, but we also have high levels of the lipid peroxides showing this actual damage by oxidants.

Q This is a lab result of July 16, 2002. Is that correct?

A That's correct.

Q Okay. We also have a lab result from December 17, 2002, from later in that same year. This would be page 78 of Exhibit No. 4.

A That's correct. This is essentially the

same test with very similar results, so I don't think I need to explain it all again.

Q We're now going to move into pages 96 and 97 of Petitioners' Exhibit 4, and we'll start with page 96. Is that what you have there on paper and on the screen?

THE COURT: Page 96?

MR. POWERS: Yes, Special Master.

THE COURT: That's the one we started with on oxidative stress.

THE WITNESS: Yes. I think we've already done this one.

MR. POWERS: Yes.

THE WITNESS: Sorry.

MR. POWERS: So it would be page 97, the plasma cysteine panel.

THE WITNESS: Okay.

MR. POWERS: Okay. I'm sorry. It would be page 97. Thank you, Special Master.

THE WITNESS: Okay.

BY MR. POWERS:

Q Okay. And this is testing that again was done in July of 2002 at the same time that the testing you just described was done? Is that correct?

A That's correct.

Q And what does page 97 tell you, and why is it significant?

A This is a blood test looking at cysteine. Cysteine is the amino acid that is the prerequisite for glutathione formation and has a crucial role in neuroprotection. It is showing that it is outside the normal range in the low range.

By inference, if you have a low cysteine you would expect a low glutathione as we documented, and so this is another confirming laboratory value to document abnormalities in this methylation pathway and therefore a reduced ability for the child to handle heavy metal toxicity.

Q And let's then go to page 80.

A That is a blood test looking at plasma sulphate. Sulphate is part of the detoxification mechanisms by which we all are able to excrete toxins, and this is demonstrating that it is below the normal range.

This is to be expected, given what we know about the child's glutathione status and cysteine status, but is more specific documentation of that detoxification function of that whole metabolic pathway showing that it would be functioning suboptimally.

Q We're going to move on then to Petitioners' Exhibit 4, page 93. Do you see that on the screen?

A Yes, I do.

Q Okay. Could you describe for the Special Master what this document is, what it tells you and why you believe it's significant?

A This is a urine looking at toxic metals. It was obtained on September 20, 2002.

It is crucial to note that according to both the mother and the treating physician, who I spoke to personally about this, this was not actually a post-provocative urine with DMSA. It was actually the baseline urine.

A common practice is to initially do a urine for toxic metals in the natural resting state of the child and then to give a chelating agent to try to look at body burden of various toxic metals.

In this specimen, which is actually a baseline specimen, mercury is essentially nondetectible where the reference range would be less than three micrograms per gram of creatinine. That is significant, particularly once taken in context with the post-provocative specimen.

Q Let's take a look at the post-provocative specimen, and this I believe would be page 90 of

Exhibit 4.

A That's correct.

Q Okay. So what you see on the monitor, is that the post-provocative test?

A Yes.

Q Again, if you could describe for the Special Master what you see on this test and why it's significant for you?

A What I see here is that after receiving an agent called DMSA, which is a chelating agent that will potentially help the body get rid of lead and mercury, that the mercury rises to 17 micrograms per gram of creatinine, where normal would be less than three, and the bar essentially goes off the chart into the very elevated range.

This to me suggests that through the use of the DMSA, which acts by complexing in the metallothionein complex and displacing the heavy metals, this child excreted a dramatic amount of mercury. This implies that he had a significant body burden of mercury.

Q Now, you say it implies he had a significant body burden. Is it possible that it simply reflects ongoing exposure to sources of mercury?

A One would not be able to answer that

question purely on the basis of this lab test, but there are other tests in his record which lead me to believe that he did not have any ongoing sources of mercury exposure.

Q Okay. Let's go ahead and take a look at some of those. We'd be looking at Petitioners' Exhibit 4, page 131.

A This is a test on red blood cell elements.

Q And let me interrupt. So the tests we just looked at were urine tests?

A Yes. That's correct.

Q Okay. And so this is a test that's based on drawing blood from Colin?

A That's correct.

Q Okay. Please go ahead.

A The red blood cell elements test is frequently used to make sure that children have adequate amounts of essential nutrients in their blood.

So the first part of the test that's marked Nutrient Elements is giving an idea about the amounts of essential elements like calcium and zinc and magnesium and selenium in this child compared to a normal range, and it's given in the range of percentiles.

So, for example, this child, who was on a gluten-, casein-free diet, was having a calcium level that was around the 75th to 80th percentile, so that gives you evidence that his calcium was being adequately replenished by his supplements.

We particularly want to look at nutrient elements in children where either chelation is anticipated or ongoing because the biggest morbidities associated with chelation are when the chelating agent grabs an essential element instead of a toxic element. So Dr. Bock was being conscientious I believe, trying to make sure that he had adequate zinc and adequate selenium and other essential nutrients.

The bottom half of the test reflects potentially toxic elements, and the reason that this is very important to me is that the very first step when you're trying to deal with anyone who may have evidence of toxic exposures is to ensure that they're not having ongoing toxic exposures. Otherwise you just end up chasing your tail as you try to get rid of something as they continue to be exposed. So in looking at this, we see that with regards to mercury he did not have evidence of ongoing toxic exposures.

I had the opportunity to ask the mother if they had moved to a different environment between the

time he was a baby and the time this test was taken such that perhaps there was an environmental component at the time of his infancy that might explain this big mercury burden, but she explained that no, they were in the same house, that there wasn't any change in industry in their neighborhood that would have been providing an environmental source of mercury.

And so I thought this was very interesting in that it showed evidence against ongoing exposures that had happened within the previous 120 days.

Q Let me interrupt. Why is that 120 days significant?

A Because your red blood cells turn over quickly, and on average you've replaced them within 120 days.

So if you're going to look for evidence of lead or mercury in the blood, you essentially are looking for acute exposures and so by not having any mercury present here in the red blood cells I do not see any evidence that he had ongoing exposures, and that I believe argues that we have to at least consider that his body burden could be coming from his inability to have handled the thimerosal in his vaccines.

Q Now, it's not that it's proof positive that

that's the case, but would it be your testimony that it's consistent with that possibility?

A That's correct.

Q Okay. Let's look at Petitioners' Exhibit 4, page 75. Do you see that?

A Yes. That's another red blood cell element test that was done in December of '02. Two potential things of importance here to me.

One is that once again the mercury level in the red blood cells is not high, suggesting no ongoing exposures to mercury. The other thing that is important to me is that the selenium level is low.

Selenium is one of the mechanisms that the body uses for glutathione function and to help get rid of mercury and so it will be used up when the body is trying to do that process.

We frequently supplement children with selenium. It may be in their multiple vitamin, or sometimes we give them extra selenium. It's one of the nutritional ways of helping the body itself to mobilize heavy metals the way that nature intended.

Q Now, I want to pause here for a moment on this idea of mercury exposures.

Do you recall testimony from the Mead and the King cases that one source of mercury exposure in

infants is through the mother's breast milk?

A Yes. That's correct.

Q And that would be the methyl mercury that would be passed that the mom is exposed to in the breast milk and goes into the child. Do you recall that sort of testimony?

A That's correct. Yes.

Q So it's true then that one potential source of mercury exposure for Colin Dwyer would have been breast milk, correct?

A That's correct, but in this case the mother explained to me that she did not breast feed; that the baby was formula fed from the beginning.

Q So in terms of exposures to mercury, can you see anything in the family's testimony, conversations, medical record indicating any exposures to mercury other than the thimerosal-containing vaccines administered to Colin?

A No, I did not detect evidence of other exposures.

Q I'm going to move ahead to Petitioners' Exhibit 4, page 67. What is that document, and can you explain what you see there and why it's significant to the Special Master?

A This is a urine test done at Metametrix

Laboratories that is looking at a wide variety of markers in the urine that are essentially breakdown products of metabolism that are excreted in the urine.

We use these tests in order to get biochemical footprints that might give us some insight into what is going on on a cellular level with these children.

The things that I found interesting and informative in this record included the lactate and then some of the citric acid cycle markers. The reason that the high lactate was significant to me, lactate is an anaerobic breakdown product of glucose, and it tends to be higher under anaerobic, nonoxygen environments.

One of the things that can lead to an increase in lactic acid is lack of mitochondrial ATP, implying potential for mitochondrial function. Now, I need to be very clear for the Special Masters that there is a very long differential diagnosis of what can cause a lactic acidosis. It could be something like recent vigorous exercise or septic shock or significant vomiting and diarrhea that includes dehydration.

So I am not in any way suggesting that a high urine lactate is the biomarker to prove

mitochondrial dysfunction and abnormalities in anaerobic metabolism specifically. I'm saying that with that high marker once again it reflects the potential for an ongoing metabolic acidosis and is consistent with our concerns about the low glutathione in this child and therefore poor mitochondrial function and all the other things I elaborated.

The other thing that is significant to me as a clinician is the citric acid cycle markers, many of which were elevated. A pattern that is seen in people who have heavy metal toxicity is that each of the heavy metals can have potential impacts on that citric acid or Krebs cycle. That cycle is how we produce cellular energy in the form of ATP.

So when you have so many markers that are high, one of the things in your differential diagnosis is heavy metal toxicity. Specifically an elevated succinate is one of the markers for an increased requirement of CoQ10, which is important in mitochondrial oxidative phosphorylation.

Another important thing is that the fumarate and malate that are elevated can be markers of CoQ10 deficiency, and also the malate can be elevated with B_{12} deficiencies, so again as a piece of evidence, even though these isolated markers cannot be over

interpreted, looking at the pattern I see a pattern that suggests interference with the citric acid cycle, which is a very fundamental biochemical mechanism by which we produce cellular energy and therefore allow our cells to do our jobs.

Q Let's go ahead and look at page 126, and we're going to quickly have page 81 after that, but we'll start with page 126 of Exhibit 4.

A This is a lab test that is a pretty traditional lab test. It's basically a chemistry screen.

I mentioned in my report the fact that a number of Colin's CO_2 measurements in his blood were minimally decreased. When I got extra records, I actually found that there were some that were decreased as low as 19.

I need to be very clear to the Special Master that a low CO_2 also has a huge differential diagnosis, and it can be low in acute illness and dehydration and any number of things that would cause metabolic acidosis.

It also can be low if the child is screaming for a long time before the blood draw, and so I only offer this as consistent evidence that this child may have had a mild ongoing metabolic acidosis.

In the records that I received after I wrote my report I actually did see a normal value, which I had not seen at the time that I wrote the report. This I would characterize as a soft marker consistent with our concerns, but not diagnostic in any way.

Q And when you say this, you're referring to really the entire sequence of results that would show reduced carbon dioxide levels in the blood?

A Right. There are several. It just documents that this was present over time on a number of different occasions.

Q Okay. And then we're going to look at page 116 of Exhibit 4 also.

A Okay.

Q And do you see that on the screen?

A I do.

Q All right. What is that, and why is it significant?

A This test is a urine for amino acids, and this is one of the tests that we can use to assess the child's ability to build body tissue since amino acids are the fundamental building blocks by which we build our bodies.

There is a pattern here that he has extremely low levels of a number of different amino

acids. The ones I was particularly struck by were initially ones that are in that methylation pathway, including methionine, which is the essential amino acid that's at the beginning of the methylation sequence; taurine, which is an amino acid that's very important in the function of bile and also in detoxification, as well as some neuroprotection; and abnormalities in cystathionine and cystine.

The taurine in particular you'll notice has a value of 14 where the reference range is 110 to 700, so this is an extraordinarily low amount of taurine, again telling me that he was at a relative disadvantage in terms of his methylation biochemistry and in terms of his detoxification biochemistry.

Other things that are important to me is that his arginine, which is the first marker, was low. That's important for leukocytes and immune function. In general, glutamine was also low, and that's very important as a nourishing cell for the gut epithelium.

The overall pattern of very low amino acids is concerning about his nutritional status and therefore his ability to do normal metabolic processes as I would like.

Q Now, we've covered in going through these exhibits the issues that you discuss in your report.

You do describe getting some additional medical records between the time that you wrote and filed your report and your appearance here today. Is that correct?

A That's correct.

Q And among those were some additional records basically from Exhibit 4? Excuse me. Additional pages from Exhibit 4 that were the even-numbered pages that were not included in some of the scanning, correct?

A That's correct.

Q Are there any pages within that set of documents that were of particular significance to you?

A Yes. One that I found very interesting was the Metametrix urine organics profile. I think it's Exhibit 4, page 68.

Q Page 68? Okay. Let's go ahead and put that up.

Again, this is not discussed in your report, but you have something to offer to the Special Master in describing this part of the evidence? Is that correct?

A That's correct.

Q Okay. If you could go ahead and let the Court know exactly what this document is, what it

tells you and why it's significant to your opinion?

A The first thing that I looked at were the neurotransmitter metabolism markers. Analytes Nos. 23 and 24 are oxidation products of epinephrine, norepinephrine and dopamine, and if they accumulate they can actually act as cumulative neurotoxins if they're not appropriately removed.

When these levels are high the increased rate implies that there's either increased synthesis or increased synthesis and degradation of those products. These are products of catecholamine biochemistry.

The catecholamines are those things that put us into fight or flight response, and so I view these as again contributing evidence that this is a child who may have been in ongoing sympathetic overdrive. The sympathetic nervous system keeps you agitated. The parasympathetic nervous system is the one that would keep you calm, relaxing, digesting your food, et cetera.

It seems to me that these metabolites are consistent with the parents' story that Colin was severely hyperactive, referred to as having severe attention problems, hyperactivity and climbing the walls was one of the phrases that was repeatedly used

by the teachers. So again, in isolation nothing that I would point to as a marker, but in concert with the rest of the evidence supporting evidence for our concerns.

He also had an elevation in Analyte No. 25. This reflects serotonin biochemistry. This marker can be elevated if a child is on SSRIs, or it can imply that there's an increased release of serotonin from either the central nervous system, the platelets or the intestine. Again, in an isolation not a big deal. In concert, more potentially supporting evidence.

The thing I was most interested in were Markers Nos. 26 and 27, kynurenate and quinolinate. The reason that I found this particularly interesting is that when I was lecturing in Australia once I had a long conversation with Dr. Richard Lord about quinolinate as a marker linking the immune system and the brain and the fact that it interacts with NMDA receptors and glutaminergic neurons, which those are items of interest in this particular trial because of our theory of causation.

Overstimulated neurons can degenerate, and this is called glutamate neurotoxicity. So one of the things that's been looked at is the ratio where a high quinolinate and a low kynurenate would lead you to

suspect neurotoxicity, and it's a matter of some research interest because any agent that would change the balance of the synthesis of these two, of the substances of kynurenate and quinolitic acid away from this excitotoxicity potential and towards the neuroprotective potential is of therapeutic interest.

So you'll see that this is comparing the child on a percentile basis, and his quinolinate is above the 80th percentile and his kynurenate, which is the neuroprotective factor, is low, below the 20th percentile, so that I thought was interesting in terms of the impact on neurotoxicity and the way that this ratio seemed to be saying that at least at this point in time the child was tipped in the balance toward neurotoxicity and away from the balance of neuroprotective.

Q And when you say neurotoxicity, are you using that as a corollary to neuroinflammation?

A Yes. Neurotoxicity or neuroinflammation. I need to be very clear that that marker does not in any way say what the neuroinflammation might be from.

Q And when you say the brain's immune system, are you talking about the brain's innate immune system?

A You know, I don't know that I have enough

biochemistry to answer that question with certainty, so I'd like to say that I don't know for sure.

Q Okay. But certainly with neuroinflammation?

A Yes.

Q Okay.

A And then the other two markers that I'd like to mention in passing is No. 32, which is glucarate. That's a biomarker for a process called glucuronidation, and that's compatible with induction of enzymes to try to handle either toxic episodes or pesticide exposure or fungicides.

It's actually not, to the best of my knowledge, what's used for mercury, but it is a marker that he may be also trying to detoxify other things by other measures.

And then the pyroglutamate, No. 33, is significant to me because that's a marker for either glycine deficiency or glutathione deficiency. Glycine is one of the components of glutathione. Glutathione, I think we've made clear our belief at how important that particular substance is.

So what I'm trying to show here is a constellation of how we've shown glutathione as low directly. We've shown that cystine, the precursor, is low. We've shown that another urinary marker also

reflects the glutathione deficiency, so from multiple labs at multiple times I believe it shows a consistent pattern.

Q Now, having described this last exhibit, page 68, and described all of the lab results you talked about in your report, are there any other laboratory results that you feel are significant to draw the Court's attention to, or does this exhaust the laboratory result portion of your opinion?

A Well, I wouldn't say it exhausts it, but it was what I prepared as the most illustrative for the Special Master.

Q Now we're going to stop talking about the lab results, and as we come to a conclusion here I want to talk a little bit specifically about Colin Dwyer's course of care and his overall health.

Now, you've described how he developed normally. Do you recall that?

A Yes.

Q And you described how he regressed, and you offered your opinion that it was regressive autism, correct?

A That's correct.

Q Based on your review of the medical records and listening to the parents' testimony, what's your

impression of Colin's course of care just generally from the point of his diagnosis at two and a half years of age moving forward?

A First, it became very apparent that the parents moved heaven and earth to get all the help they could get for their child. They went into debt, they changed career paths, and they paid a huge amount in terms of time, travel and money to try to get him the services that he needed.

They sought out the best resources available to them geographically and the best schools, paying very high prices for good behavioral interventions for him. They traveled to meet a doctor that would try to address his medical needs.

I would say that looking at his pattern in general over time, he has clearly exhibited some periods where he seemed to be progressing well, some periods where he had challenges, some periods where he plateaued and then other periods where he made progress again.

So at this time he is in a much better place than he was at the age of two when the father couldn't take him out in a stroller or they couldn't interrelate with him. Now he is able to do some things socially and be involved in some family

outings, but I understood the father to say that they still couldn't take this child to a restaurant, which would be something that I feel bad about because he's old enough that they should have been able to do that for many years now.

So I'm very impressed by their dedication, and I am very concerned that he is likely to have some residual problems in his future, as the father so well articulated yesterday.

Q Now, based on everything that you've reviewed in preparing for your testimony, in preparing your report, ultimately did you reach a medical opinion about what might have caused or contributed to Colin Dwyer's autism?

A After looking at the records carefully, I came to the opinion that thimerosal-containing vaccines must be on the list of differential diagnoses of what could have caused this problem; that I reasonably looked for and did not find other alternative sources of mercury; that I looked for and did not find other alternative diagnoses for his pattern of regressive autism.

Because his laboratory data and clinical course showed evidence of so many of the medical problems I would expect to be in a child with autism

who had difficulty excreting toxins, it is my best professional judgment, based on what I know as of this date, that thimerosal-containing vaccines substantially contributed to his medical problems and his regressive autism.

Q Anywhere in the medical record did you see any evidence that Colin Dwyer is mentally retarded?

A I did not.

Q Is there anything in your review of the parents' testimony, in conversations with treating doctors, in conversations with the parents, supporting the conclusion that Colin Dwyer is mentally retarded?

A I did not see any evidence of that, and in talking with the mother she shared a story that I believe was also in her testimony about very experienced clinicians, including a Ph.D. psychologist at the McCarton Center, explicitly telling her that Colin was a good problem solver, that he had good cognitive abilities and that he was progressing well intellectually.

Q And do you recall that his receptive language skills have improved over time, while his expressive language has lagged behind?

A That's true.

Q And the improvements in his receptive

language, would that be consistent with the conclusion that he is not mentally retarded?

A Yes. I do not find evidence that he's mentally retarded, although I have not personally examined the child.

Q Correct. In your differential diagnosis would you look for potential genetic causes of autism? Are there identifiable genetic causes of autism in your experience?

A There are definitely identifiable genetic causes of autism. There are several ways you can look for that.

One of the most important things is to do a careful physical exam for what we would call dysmorphic features. These are things such as abnormalities of the spacing between the eyes or abnormally low-set ears or particulary shaped faces or abnormalities in the hand lines or the fingers or the nails.

Whereas I will say I did not -- see several notations in the records that there was no dysmorphology, I did not see a very detailed genetic exam that specifically listed everything that they looked for. But on the basis of several experienced clinicians, a pediatric neurologist and an excellent

developmental pediatrician examining the child, they did not seem to find evidence of genetic abnormality.

I did look carefully through the records to see if chromosomes had ever been done, and I did not find evidence of that. The mother told me last night that no one had actually recommended that to them, so I must assume that they thought that the yield on doing chromosome testing was very small in this child.

Q And when you say they had the opinion that it would be a low yield series of tests to do, are you referring to the medical professionals that treated Colin?

A Yes. I am sure that both Dr. Bock and I would assume Dr. Fish and from what I know of Cece McCarton, they would have all had that foremost in their mind to do if they felt it was indicated I believe.

Q And the fact that they did not indicate it at all, does that tell you that there was not a genetic component that ought to be pursued?

A It tells me that they did not suspect it. I can't say that we've ruled it out because we haven't done all the possible genetic tests that could be done in this child. There's ever-increasing amounts of micro arrays that could be done to detect subtle

abnormalities.

Q And finally, the medical opinion that you just expressed about the role of thimerosal-containing vaccines in Colin Dwyer's regressive autism. Is that an opinion you hold to a reasonable degree of medical certainty?

A It is.

MR. POWERS: Thank you.

I have no further questions right now, Special Master.

MR. JOHNSON: Could we have five minutes?

THE COURT: Why don't we take a little longer and take a midmorning break, a restroom break, before we begin. Let's reconvene at 25 to.

(Whereupon, a short recess was taken.)

THE COURT: We're back on the record. Mr. Johnson, you may cross.

CROSS-EXAMINATION

BY MR. JOHNSON:

Q Good morning, Dr. Mumper. Good to see you again.

A Good morning.

Q As you know, my name is Vo Johnson. I'm representing the government in this case.

I want to start off by asking you a little

bit about what you relied on. I know you were asked some questions on your direct about what formed the basis for your opinion.

I believe you mentioned, and I just want to confirm, that you have not performed an evaluation of Colin Dwyer. Is that correct?

A That is correct.

Q And I take it you did not speak with his parents prior to preparing your report in this case? Is that correct?

A That is correct.

Q You mention in your report that you did discuss the case with Dr. Bock. Is that right?

A That is correct.

Q Why did you want to discuss the case with Dr. Bock?

A In the initial medical records that I received I saw that September 20 and September 22 urine test, and it appeared to me that it would not be standard practice or logical to do two post-provoked urines so close together.

I suspected that the first test had been mislabeled through an administrative error, so I wanted to ask him about that and clarify that.

Q So it was the September 2002 urine tests in

particular that you were concerned about that you wanted to speak with Dr. Bock about?

A That's correct.

Q Was there anything else that the two of you discussed when you met with him?

A You know, we really didn't. We were at this think tank and so I only asked about that lab and then we ran out of time.

Q I want to talk now about the test results that you discussed in your report and here today.

A Yes.

Q You discussed two different lab results from a laboratory, Immunosciences Laboratory. Is that correct?

A That's correct.

Q And these are the antibodies to neurofilament and the antibodies to myelin basic protein?

A That is correct.

Q I'm going to refer to antibodies to myelin basic protein as anti-MBP if you don't mind.

A That is perfect.

Q Doctor, do you order or have you ordered tests from Immunosciences in your own practice?

A Yes, I have.

MR. JOHNSON: I want to show you a document that I found on their website. I want to I guess mark this as a trial exhibit.

Special Master, in the May hearing we were up to Respondent's Trial Exhibit 12, so we're going to start with Exhibit 13 if that's all right with you, or would you prefer to do it --

THE COURT: I'm just trying to think of what's going to be less confusing. Let's go with 13. We'll probably renumber your exhibit then to be the next --

MR. JOHNSON: The next sequence that follows?

THE COURT: Yes. That makes more sense, considering we're going to file these records in each other's cases.

(The document referred to was marked for identification as Respondent's Trial Exhibit No. 13.)

BY MR. JOHNSON:

Q Dr. Mumper, have you seen this before?

A I have seen the premier autism panel. I'm not sure that I've seen it actually on this website page.

Q Is it your understanding that this is a panel of tests that Immunosciences performs or offers in connection with autism?

A That is correct.

Q In your opinion, is this the standard panel of tests for autism?

A I would say no, this is not a standard panel for autism.

Q Do you order any of these tests for your own patients?

A Sometimes. Frankly, one of the reasons that I don't order them very often is that they are expensive, and there are other tests that I typically would do first that might be able to be run through a local lab.

But I have ordered a number of tests from them, and I've also used their lab for some research work that we've done.

Q You mentioned that the tests can be expensive. Do you have an idea how much these tests -- for example, the premier autism panel. If you ordered all of those tests, how much would that cost?

A I think it's about $600, but expanded panels can be as much as like $1,200.

Q You agree that autism cannot be diagnosed

through laboratory testing, correct?

A I do agree.

Q The first Immunosciences Lab test that you discuss in your report is the one that showed mildly elevated IgM neurofilament antibodies, correct?

A That's correct.

Q And this was a blood test, correct?

A That's correct.

Q You state that this test result lends support to the conclusion that Colin experienced a neuroinflammatory process as described by Dr. Kinsbourne, right?

A I did.

Q Dr. Kinsbourne said nothing about IgM neurofilament antibodies being a marker for neuroinflammation. Is that correct?

A I think that is correct.

Q And you're aware that a group of researchers has looked for serum and CSF markers of inflammation in autism. Is that right?

A That's correct.

Q And this was a study where the lead author was Dr. Zimmerman? Is that correct?

A That's correct.

Q IgM neurofilament antibodies was not a

marker that Dr. Zimmerman looked for in the serum or CSF of his autistic subjects to detect neuroinflammation. Is that correct?

A I actually do not know. I remember there was a huge list of things they looked at in the CSF, and the thing that was really markedly elevated was interferon gamma, but I do not honestly recall if this was on that list.

Q Are you aware that circulating antibodies to neurofilament proteins have been demonstrated in many disorders, such as Alzheimer's and ALS?

A Yes.

Q Isn't it just as likely that those findings are secondary to the ongoing pathological process, as opposed to being the cause of the process?

A The significance of that value to me was not necessarily looking at cause versus effect, but as a marker for neuroinflammation and reactive gliosis.

Q Isn't it true that certain medications could also cause elevated levels of antibodies to neurofilament protein?

A That may be true.

Q The second Immunosciences test result that you discuss is the anti-MBP finding, which you state is a marker for autoimmunity. Is that right?

A That's correct.

Q The mechanism of neuroinflammation proposed by Dr. Kinsbourne is not an autoimmune response. Is that right?

A It is a neuroinflammatory response. With immune dysregulation we will often see evidence of autoimmunity, and we have evidence from the medical records as early as four months of age that Colin may have had immune dysregulation in that he exhibited what was recorded as nummular eczema versus tinea capitis.

So I don't know for sure whether that was eczema or a fungal infection, but in either event it is suggesting that at four months of age he had some signs of immune dysregulation.

Q Dr. Kinsbourne is not proposing demyelination as part of his mechanism in this case. Is that correct?

A I think that's probably true. He's talking about neuroinflammation and reactive gliosis.

Q And Dr. Pardo, upon whom Dr. Kinsbourne relies, does not endorse an autoimmune basis for autism. Is that right?

A That may be true. Again, to document autoimmunity in a particular patient is not to say

that autoimmunity is the cause of autism.

Q And Dr. Deth's oxidative stress model is not based on autoimmunity. Is that right?

A That's correct.

Q Neither the myelin basic protein nor IgM neurofilament antibody test is diagnostic of any disease. Is that right?

A That's correct.

Q They are very nonspecific findings.

A That's correct.

Q And isn't it true that these antibodies have been reported as elevated in normal individuals with no disease?

A That is true in some cases. Exactly.

Q And because these markers were measured in the serum rather than the CSF, they provide no direct evidence of what is going on in Colin's central nervous system. Is that right?

A I guess I would quibble with how you get direct evidence. In this case, to get direct evidence of neuroinflammation I guess we would really need to have done a brain biopsy on him in 2002.

I can tell you from personal experience that even wanting to look at CSF in children with autism for the presence of inflammatory markers is widely

perceived as an invasive procedure, so those of us who might want to be able to document it more directly are constrained from doing so by standards of care criticisms.

So we have to rely on other markers, and it's not a direct marker, but I would argue that a clinician would not have the ability to do a direct assessment in a living child.

Q For whatever reason, that evidence is just not present in this case, correct? The CSF testing is not present in this case?

A That's true.

Q Do you know what protocol Immunosciences used to perform these two lab tests?

A You know, I don't. I have visited Immunosciences Labs on two occasions and talked to the director and viewed their facilities, but I am not a lab scientist so I can tell you that when I visited and had it explained to me it made sense to me at the time, but I could not reproduce the protocol.

Q Do you know how Immunosciences established its reference ranges?

A I do not know the details of that, no.

Q Do you know whether these reference ranges take the age factor into account? In other words, are

they normed for children?

A I do not think they are normed for children, but for things like neurofilament antibodies and myelin basic protein antibodies the values for children would be expected to be less than people as they aged and had more damage as a result of aging or disease.

Q But you don't believe that these reference ranges are normed for children?

A I do not think they are. That's correct.

Q Do you know whether Immunosciences Lab has ever been accredited by the College of American Pathologists?

A I do not know if they have. I do know that their work, their lab reports, come with disclaimers about use for research and careful clinical applicability and those types of things.

Q Do you know whether Immunosciences is currently performing any clinical testing?

A I believe they are not.

(The document referred to was marked for identification as Respondent's Trial Exhibit No. 14.)

BY MR. JOHNSON:

Q I'm going to show you what we've marked as Respondent's Trial Exhibit No. 14. This is a letter that I found on the Immunosciences website.

A Okay.

Q Doctor, have you seen this letter before?

A Yes, I did. Yes, I have.

Q And does this letter reflect that Immunosciences has in fact stopped performing clinical testing as of July 21, 2007?

A Yes, as I just testified to.

Q Do you know why Immunosciences stopped performing clinical testing?

A My understanding from talking to Dr. Vojdani and some Health Department officials is that his lab was investigated for their testing as it related to mold, looking for evidence of chronic mold exposure as a potential cause of chronic illness.

My understanding from Dr. Vojdani is that the investigation was perhaps precipitated by a Court case in which mold testing had been used, and the plaintiff who had claimed damage from mold had won a huge settlement.

The Health Department was concerned about the possibility of lawsuits being settled on the basis of that mold test and wanted to investigate the lab

with regard to that.

Q So it's your understanding that the problems at Immunosciences Lab were limited to its mold testing?

A Well, that is my understanding, but I have not investigated all of the depth of the investigation nor read any of the official documents, so I really do not have full knowledge of that.

(The document referred to was marked for identification as Respondent's Trial Exhibit No. 15.)

BY MR. JOHNSON:

Q I'm now going to show you Respondent's Trial Exhibit No. 15, which is another letter that I found on the Immunosciences website.

A Okay.

Q Doctor, have you seen this letter before?

A I believe I have, yes.

Q Did you receive this letter since it's addressed to Our Valued Clients and Associates? Was this sent to you?

A Yes.

Q This letter is signed by Dr. Vojdani.

A That's correct.

Q And I believe you testified at the hearing in May that you have an article in press regarding one of your research projects on which Dr. Vojdani is the lead author. Is that right?

A That is correct.

Q Do you know what C-L-I-A or CLIA stands for?

A CLIA. Certified Laboratory -- I can't remember because we always refer to it by the acronym. I'm sorry.

Q Okay. Just for the record, it's Clinical Laboratory Improvements Amendments of 1988, and we'll just refer to it as CLIA for ease of reference.

A Okay.

Q Do you know what CMS is?

A According to the letter, it might be Centers for Medicare and Medicaid Services.

Q That's correct. CMS regulates all clinical laboratory testing on humans in the United States through CLIA in order to ensure quality laboratory testing. Is that right?

A Uh-huh.

Q Dr. Vojdani's letter states in the third paragraph that CMS had found deficiencies during a 2004 CLIA survey of Immunosciences that led it to conclude that the lab's test results since 2002 may

not be accurate and reliable. Were you aware of those findings by CMS?

A Yes, since I got this letter.

(The document referred to was marked for identification as Respondent's Trial Exhibit No. 16.)

BY MR. JOHNSON:

Q I'm now going to show you Respondent's Trial Exhibit 16, and this is a letter from CMS. Doctor, have you seen this letter before?

A Yes, I have.

Q Did you receive this letter?

A Yes, I did.

Q This letter does in fact say at the beginning of the second paragraph on the first page that:

We are writing both to inform you of the current sanction action and to alert you that test results you received since June 10, 2002, from Immunosciences Lab, Inc. may not be accurate or reliable. Is that what that says?

A That's correct. I would like to add for the Special Master that when I received this letter I did call Mary Jew as suggested in the last line. I can't

remember the details now, but I talked to three different people on the staff.

I tried to get information about what particular concerns they had because I was trying to figure out for the labs that I had done on my patients if this were a global concern or if it was related to the mold or if there were tests that I was using that I may still be able to rely upon.

I was very frustrated in not being able to find out from those people, who I think their hands were tied in terms of talking about an ongoing investigation, what the problems were.

(The document referred to was marked for identification as Respondent's Trial Exhibit No. 17.)

BY MR. JOHNSON:

Q We may be able to provide some of that information now. I'm going to show you now what's been marked as Respondent's Trial Exhibit 17.

This is the CLIA Annual Laboratory Registry from 2005. Have you seen this document before?

A No, I have not.

Q Look on page 5 of this document. Does this indicate that Immunosciences' CLIA certification was

being revoked due to condition level noncompliance?

A Cancellation of approval to receive medicare payment due to noncompliance. Yes.

(The document referred to was marked for identification as Respondent's Trial Exhibit No. 18.)

BY MR. JOHNSON:

Q I'm going to show you Respondent's Trial Exhibit 18. These are actually excerpts from a much larger report.

This is a report from the survey that CMS did of the Immunosciences Lab. Based on your review of this document, does that appear correct to you?

A Based on my 30 second review, that does appear to be correct.

Q If you'll turn to the fifth page of the trial exhibit? This document lists a number of findings in connection with Immunosciences' general immunology testing. Is that correct?

A It appears that that is correct.

Q Were you aware that CMS noted problems at Immunosciences Lab in connection with its failure to follow written policies and procedures for an ongoing mechanism to monitor, assess and correct problems in

the preanalytic systems?

A No. I did not have access to that information.

Q And were you aware that CMS found that the laboratory failed to determine calibration procedures and control procedures based on established performance application?

A No. I wasn't aware of the specifics.

Q And were you aware that CMS found that Immunosciences Laboratory failed to verify the continued accuracy of the test system throughout the laboratory for portable (sic) range of test results?

A I'm sorry. What was that phrase? A portable range?

Q Reportable range.

A Oh, reportable range.

Q This is subparagraph (g).

A That appears to be what the document says. I was not aware of the specifics.

Q Okay. And under subparagraph (i), the CMS found that Immunosciences Laboratory failed to establish the statistical parameters of unassayed control materials used for its various in-house ELISA test systems?

A I was not aware of that.

Q And these findings all relate to Immunosciences' general immune testing. Is that correct?

A It would appear that that's the case.

Q Okay. And if you'll now look on the next to the last page of the trial exhibit?

Were you aware that CMS found with respect to the anti-MBP and neurofilament test in particular that Immunosciences failed to have written policies and procedures for patient preparation, specimen collection, specimen storage and preservation, conditions for specimen transportation and specimen acceptability and rejection?

A And what was the date of that that it was not in place? It seemed to be on the website when you cited it earlier and when we sent specimens in 2003 we were able to obtain written instructions about the specimen submitted. They came actually in the test kit.

Q I believe this was a survey from 2004, so CMS had apparently found at this time that at least whatever written procedures that they had were not adequate. Is that correct?

A Well, that may be what they found. What I was trying to explain to you is as a clinician the

test kits come in a box. They're the tubes and then a series of explanations about how the specimens need to be prepared.

So I can only testify as to what I know and to what you show me that's in the lab document, but I'm trying to explain that we had procedures to follow when we submitted our blood samples in 2003.

Q And all I'm asking you is that at the time that CMS performed this survey it found those aspects of Immunosciences Laboratory's practice to be inadequate. Is that correct?

A Yes. That would be apparent from the document.

Q Okay. And if you now want to look at the last page of the trial exhibit?

Isn't it also true that CMS found at the time it performed this survey that with respect to the anti-MBP test and the neurofilament test that Immunosciences failed to provide documentation to show the laboratory director's review and approval for those procedures?

A It does suggest that there was no documentation to show his review and approval. I do know from talking to him that he did review those procedures, so how much of this was a matter of

paperwork versus actual analysis I can't say.

Q In Dr. Vojdani's letter of January 16, 2006, he indicates that Immunosciences planned to sue over the survey results. Were you aware of that?

A I wasn't sure if he was going to sue. He said vigorously fight or something that effect, which I wondered if he meant to go through administrative channels. So I didn't know the specifics of what he meant by that.

THE COURT: And that was referring to Respondent's Trial Exhibit No. 15?

MR. JOHNSON: Yes, Special Master. Thank you.

(The document referred to was marked for identification as Respondent's Trial Exhibit No. 19.)

BY MR. JOHNSON:

Q We have a copy of the settlement agreement from that lawsuit. It's been marked as Respondent's Trial Exhibit 19. Focusing on paragraphs 1, 2 and 3, if you want to review those?

(Pause.)

A Okay.

Q It appears that one of the conditions of the

settlement was that Immunosciences would obtain accreditation through the College of American Pathologists or else it would voluntarily withdraw from the CLIA program and cease testing on human specimens. Is that correct?

A That does seem to be the case.

Q Based on the fact that Immunosciences is no longer performing clinical testing, isn't it reasonable to infer that they did not receive accreditation through the College of American Pathologists?

A Or that they chose not to pursue it I would think would be the two possibilities.

Q Doctor, based on this information, do you have any concerns about the reliability of the Immunosciences test results?

A Yes, I do. I was not aware that the MBP or the neurofilament testing was under contention, and if that were the only thing that I was relying upon to make my judgment I would be concerned that I had over read the labs.

So I would give relatively less credence or perhaps even be forced to discount the reliability of those two particular lab tests given the information in the settlement agreement, which I wasn't privy to

knowing the details of.

Q The next test results that you discuss in your report are results from Great Smokies Lab, which purport to show abnormal glutathione, lipid peroxide, cystine and plasma sulfate levels. Is that correct?

A That's true.

Q This testing was done in July and December 2002. Is that correct?

A That's correct.

Q So that would have been when Colin was in July approximately three and a half years old and then in December a little over four years old. Is that right?

A That's correct.

Q So to the extent that these results indicate anything about whether Colin was in oxidative stress at the time, they don't tell us whether Colin was in oxidative stress at the time he received his immunizations. Is that correct?

A That's exactly correct.

Q These tests were blood tests? Is that correct?

A That's correct.

Q Do you know whether these tests were normed for children?

A I do not know the answer to that question.
Q And as you note in your report, a number of other factors can explain oxidative stress, such as poor nutrition. Is that right?
A That's correct.
Q Would you agree that a mercury efflux disorder is still a hypothesis at this point?
A Yes.
Q Low cystine and plasma sulfate levels can't be diagnostic of that disorder. Is that right?
A That's correct.
Q And those levels could also be explained by a number of other factors. Is that right?
A That's correct.
Q The next testing data that you discuss in your report is the mercury testing.
You talked about some of the results during your direct testimony, but I'd like to go through all of the mercury testing that's in the record if you don't mind.
A Okay.
Q The first test that we were able to locate is at Petitioners' Exhibit 4, page 131. I believe this is one that you did discuss. This was a test. The specimen was collected on April 19, 2002. Is that

right?

A Uh-huh.

Q And this was a red blood cell elements test? Is that right?

A Yes.

Q And as I believe you testified -- or I can't remember if you testified actually -- was there any chelating agent administered in connection with this test?

A Not in connection with this test. I would have to correlate it with Dr. Bock's notes and the parent history to know if he was actually getting a chelating agent during this time.

Q But as you sit here today, you have not tried to make that correlation?

A I looked at the records with the labs, but I can't recall if he was on chelating agent or not.

Should we find that out, or do you already know the answer?

Q I don't know the answer actually. If you'd like to look, that would be fine.

(Pause.)

A It would appear that 4-19-02 was the time of the very first visit to Dr. Bock, so there is not evidence that he would have been on a chelating agent

at that time.

Q And the result from this test for mercury was that it came back in the nondetectible limit. Is that correct?

A Right.

Q The next test that we found was the September 20, 2002, test, and this was a urine toxic metals test. Is that correct?

A That's correct.

Q And I believe you testified that although this report indicates that there was a chelating agent administered, you don't believe that there was for this particular sample. Is that right?

THE COURT: And you're referring to Exhibit 4, page 93?

MR. JOHNSON: Exactly. I apologize.

THE WITNESS: Yes. That's correct.

BY MR. JOHNSON:

Q And the result for this test was mercury was in the nondetectible limit?

A That's correct.

Q The next test was the September 22, 2002, test which is at Petitioners' Exhibit 4, page 90 and I believe you testified that this was the post-provocative test. Is that right?

A That's correct.

Q This test result showed that mercury was at 17 micrograms per gram of creatinine. Is that correct?

A That's correct.

Q The next test result that we found was from November 3, 2002. That's when the sample was collected. This was another urine toxic metals test. Is that correct? This is Petitioners' Exhibit 4, page 85.

A That's correct.

Q This was a post-provocative test, correct?

A That is the way that it's labeled.

Q It appears that the chelating agent, DMSA, was administered in connection with this test. Is that right?

A I'd like to check the contemporaneous medical record again if I could, please.

(Pause.)

THE COURT: Pages 11 and 12 may be helpful to you.

THE WITNESS: I just found them. Thank you. Okay. Now I'm there.

So, yes. I can't find anything in the actual medical records that would say specifically

about this lab test, but he did say decreasing DMSA to 100 I think is what he's saying. That may have been on October 30. Yes.

BY MR. JOHNSON:

Q Okay. So that would indicate that Colin was on DMSA at the time that this sample was collected? Is that correct?

A Right.

Q And this test result showed no detectible mercury. Is that correct?

A That's correct. It shows elevation in the lead, which DMSA also helps mobilize.

One of the studies that we've done at ARI is looking at the relative rates of lead and mercury excretion. One of the patterns that we've seen is that frequently lead will be elevated first and then mercury will come out second, but there was not any mercury coming out at this time on provocation. That's correct.

Q All right. Let's look at the next test, which is at Petitioners' Exhibit 4, page 75. This is from a sample collected on December 11, 2002.

I believe this is a result that you did discuss during your direct testimony. Is that correct?

A That is correct.
Q And this is the red blood cell element test?
A That's correct.
Q Again, as you testified, this test also resulted in nondetectible mercury. Is that correct?
A Yes. That's the red blood cell test reflecting no acute exposures.
Q Al right. Let's look at the next test that we were able to find, which is at Petitioners' Exhibit 4, page 73,
This is from a sample collected on December 29, 2002, and this is a urine toxic metals test. Is that correct?
A That's correct.
Q The test report indicates that DMSA was administered in connection with this test. Is that correct?
A That's correct.
Q And this test also showed nondetectible levels of mercury. Is that correct?
A That's correct.
Q And the last test that we were able to find is at Petitioners' Exhibit 4, page 63. This is from a sample collected on March 2, 2003. Is that correct?
A That's correct.

Q And this is another urine toxic metals test? Is that correct?

A That's correct.

Q And the report indicates that DMSA was administered in connection with this test. Is that right?

A That's correct.

Q And again the results from this test for mercury was nondetected. Is that correct?

A That's correct.

Q So in the medical records there's only one test that showed mercury outside of the reference range. Is that correct?

A That's true.

Q And that was the provoked test from September 22, 2002. Is that right?

A That's correct.

Q Doesn't Doctor's Data say in bold right on the test report that reference ranges are representative of a healthy population under nonchallenge or nonprovoked conditions?

A That's true.

Q So we just don't know what the normal range would be for a provoked test. Is that right?

A It is difficult to know what that would be

on a provoked test on either sick populations or healthy populations.

Q The fact that on future post-provocation tests Colin excreted no mercury, doesn't that indicate that he wasn't having problems excreting mercury on his own?

A Does the fact that on post-provocation testing he was not excreting mercury imply that he was actually able to excrete it on his own and therefore did not need the provocation? Is that what you're suggesting?

Q That's what I'm asking.

A I don't know if that's a conclusion you can draw.

I'm taught that mercury excretion is variable and dependent on a number of factors and so that would be one of the things that I would consider, but I would also consider the fact that he had mercury stores that were not accessible to the chelating agent.

Q So that would just be a possibility that you would consider?

A Yes.

Q On page 6 of your report you go on to discuss a number of other tests that you yourself

state are not specific to any particular clinical presentation or symptom and are widely recognized to have causes other than metal toxicity or neuroinflammation and are not at all specific to autism spectrum disorders. Is that correct?

A That is correct.

Q Would you agree that the single post-provocation test from September 2002 is the only evidence in the record specific to mercury?

A That would be true.

Q If that test result were not reliable -- take it away; you can't rely on it -- would you still be able to offer an opinion in this case that thimerosal-containing vaccines contributed to Colin's autism?

A Without that piece of evidence I would be left with a number of lab tests that would be consistent with, but not specifically suggestive of that, so I guess that would be true.

Q And the post-provocation test from September 2002 is not specific to a particular species of mercury. Is that right?

A That is true.

Q So it tells us nothing about Colin's exposure to ethyl mercury as opposed to methyl

mercury. Is that right?

A That's correct.

Q And none of the other tests that you're relying on are diagnostic of mercury toxicity. Is that right?

A That's correct.

Q In fact, none of the other tests that you're relying on are diagnostic of exposure to mercury in any amount. Is that right?

A That would be true.

Q And all of the other test results that you're relying on could be explained by factors other than exposure to thimerosal in vaccines. Is that right?

A That's true. However, one would need to correlate with the child's history or the child's medical record what other things would be causing the ongoing oxidative stress, depleted glutathione, mild metabolic acidosis, abnormalities in amino acids, et cetera.

Q And you've indicated in your report even that something like poor nutrition can explain oxidative stress and things of that nature?

A It can be a contributor to oxidative stress. That's correct.

Q And there's not a single test in the record that is diagnostic of neuroinflammation. Is that right?

A That would be correct. I'm not aware of any test that's diagnostic of neuroinflammation short of biopsy and pathology, but I may have missed some.

Q And in fact I think you testified at the hearing in May that you're not aware of any good clinical markers for neuroinflammation. Is that still your understanding today?

A I would say that if you had CSF markers of inflammation that would be a nice, indirect test, but I'm not aware of any gold standard test for neuroinflammation.

Q In May you testified that you selected the Mead case because he had a history of antibiotics, multiple ear infections, respiratory infections, allergies and asthma. Do you remember that testimony?

A Yes, I do.

Q Those facts are not present in this case, are they?

A That's true.

Q Colin was generally a healthy baby. Is that right?

A That's true.

Q In fact, I think according to the record he only had two ear infections during the first 20 months of his life. Is that your understanding?

A I think that's correct.

Q You testified that you selected the King case because of his mother's antibiotic use during pregnancy and evidence of potential synergistic reactions to other exposures. Do you remember that testimony?

A That's correct.

Q And those factors are not present in Colin Dwyer's case. Is that right?

A That's correct.

Q In both the Mead and King cases you testified that your opinions were based in part on the apparent efficacy of various treatment methods employed by Dr. Green. Do you remember that testimony?

A Yes, I do.

Q Isn't it true that Colin's parents reported to Dr. Russell that the Defeat Autism Now treatment protocols they tried were ineffective?

A They may have reported that. They did tell me last night that they felt like some aspects were associated with his progress, but one of the

difficulties here is that he was receiving multimodal interventions and so it is difficult to isolate the efficacy or lack thereof of any isolated intervention when behavioral strategies and biomedical strategies were occurring at the same time.

Q So based on that answer, it's just we don't know whether the treatments were effective?

A That's correct.

Q But at least at the time that the parents were going to see Dr. Russell they reported that they did not believe that they were effective. Is that correct?

A I think that is reported in the record.

MR. JOHNSON: Thank you. That's all that I have.

THE WITNESS: Thank you.

THE COURT: Dr. Mumper, you testified that the low CO_2 levels have wide differential.

THE WITNESS: That's correct.

THE COURT: And that screaming before blood draw or a child who screamed a lot would be within that differential?

THE WITNESS: Yes.

THE COURT: And the records reflect that Colin is a screamer?

THE WITNESS: Yes, so that's why I was trying to be very careful and say that that was a very soft marker with all kinds of qualifications.

THE COURT: And you testified that the low amino acid levels in Colin were another sort of soft marker?

THE WITNESS: The fact that they were low, I would classify that as more than a soft marker because the fact that his methylation amino acids were so low I think is pretty direct evidence, especially the very low taurine of 14.

The issue is that because poor nutrition can contribute to the low amino acids, you're looking at an end result that may be from poor nutrition that would nonetheless impact the ability of the methylation biochemistry and the detoxification biochemistry.

THE COURT: So it would be both a marker and a cause?

THE WITNESS: That was a great way to put it. Thank you.

THE COURT: The records reflect Colin as a problem eater.

THE WITNESS: That's true.

THE COURT: And a problem protein eater.

THE WITNESS: That's exactly true.

THE COURT: And that would affect amino acids?

THE WITNESS: Yes. And that may well be why they were so dramatically low across the board because he was not taking in the precursors of protein, but when he had the low levels that would go on and have further impact on things like oxidative stress, ability to detoxify.

THE COURT: I have no further questions. Mr. Powers?

MR. POWERS: Yes, I do have questions for redirect.

REDIRECT EXAMINATION

BY MR. POWERS:

Q Hello again, Dr. Mumper. I want to follow up on some questions that the Respondent's attorney asked you.

Do you recall a question that he asked you if there were any medications that one might be taking that are associated with elevated levels of IgM neurofilament antibodies? Do you remember that question?

A Yes, I do.

Q Do you see anything in the record indicating

that Colin Dwyer was taking medication that would have led to elevated IgM neurofilament antibody levels?

A No, I did not.

Q You were also asked a question about IgM levels being associated with Alzheimer's and other diseases. Do you remember that question?

A That's correct. I do.

Q Do you see anything in the medical records suggesting that Colin Dwyer suffered from any disease that would be associated with elevated levels of IgM antibodies?

A Other than possibly neuroinflammation, no. Not Alzheimer's, not other neurodegenerative diseases that typically affect older people.

Q So there's nothing in the medical record implicating any other drugs or any other diseases other than what you've described that would be associated with his elevated levels. Is that correct?

A That's correct.

Q You were also asked whether these antibody tests, the neurofilament antibody tests, were diagnostic of autism. Do you remember that question?

A Not specifically, but I may well have been asked it.

Q The autism diagnosis in this case.

A Right.

Q Who reached that autism diagnosis when you go back to the medical records?

A Well, that diagnosis was reached by the pediatric neurologist, and the important factor is that autism is fundamentally a disease that's diagnosed on the basis of history and symptoms.

There are a list of criteria, and therefore the constellation of criteria is what is able to make the diagnosis. That's why we're so careful to say that there is no currently available biomedical marker for autism.

Q And you're certainly not claiming in your opinion that your assessment of Colin as suffering from an autism spectrum disorder, that specific conclusion does not rely on any lab result, does it?

A Oh, that's absolutely correct.

Q You also were asked questions about reference ranges and the normalization of those ranges for children versus adults. Do you recall that line of questioning?

A Yes, I do.

Q It was your testimony that the levels of the normal ranges, you would actually expect a child to be lower, typically to be lower within the reference

range than an adult would be, correct?

A I would expect that, but if the actual survey data has not been collected I'm not aware that I could prove that.

Q But if the reference ranges are set to fit within an adult, an adult would typically be expected to have more of these neurofilament antibodies in his or her system as a consequence of aging, correct?

A That's correct. That's my understanding.

Q So in that sense would it be fair to say that the reference range as it applies to children, again you would expect children to be lower?

A That's correct.

Q And it's a very conservative range?

A That's correct.

Q So in a sense these mildly elevated levels in a child based on the adult range are a higher elevation than it would be for an adult, correct?

MS. RICCIARDELLA: Objection, Special Master. Counsel is testifying. Should we swear in Mr. Powers?

BY MR. POWERS:

Q Would it be your testimony that --

A What I would say is that expecting children not to have evidence of neuroinflammation or

neurodegenerative diseases, a level of 53 or 57 in a toddler would be more concerning or more unexpected to me than those same levels in a middle-aged or elderly person. Is that fair? Yes.

Q You were asked a question about the mercury efflux disorder as being a hypothesis. Do you remember that question?

A Yes.

Q And what was your answer to that question?

A I don't remember.

Q Would you trust me if I said that your answer was that yes, it was a hypothesis?

A Yes. It is one of the things that we are postulating in regard to causation. It's part of the model. Is that fair?

Q Yes. Well, it's your answer so if that's your answer then are you aware of testimony or facts that are in evidence through the Mead and the King cases supporting the idea that mercury efflux is an actual condition that may exist in people?

A That was I think the substance of Dr. Aposhian's testimony in the Mead and King cases.

Q And would you be relying on Dr. Aposhian's testimony to posit the idea that children might have a mercury efflux disorder?

A Yes, I do. Through my discussions with Dr. Aposhian, he has a vast amount of experience with heavy metals, particularly mercury, that predates mine and so, yes, I do rely on his analysis of the experiments and the literature.

Q You were also asked questions about what a normal result would be of a post-provocation urine test for metals. Do you recall that question?

A Yes, I do.

Q And do you recall saying that one doesn't know exactly what a normal result would be? Do you remember that discussion?

A Right.

Q Do you consider a lab result with a finding that is five times or greater beyond the reference range to be normal under any definition of normal?

A I do not consider that to be normal. I consider it very concerning.

And I do think that one of the difficulties we're going to have in setting norms is to find people that are not living in an industrial society and are not exposed to heavy metals because I think the normals pre Industrial Revolution would have been very different than the so-called normals now.

Q And whatever the reference range is, would

you describe Colin Dwyer's post-provocation urine test where it was five times beyond the reference level, would you describe that as normal or abnormal?

A Abnormal.

Q You were also asked whether there was anything else in the record specific to mercury exposure other than the provoked chelation tests.

A Yes.

Q Would you consider Colin Dwyer's immunization record to be evidence of mercury exposure?

A Oh, that is evidence of mercury exposure. That's correct.

Q You were also asked about whether there is evidence of acute mercury toxicity in Colin Dwyer. Do you recall that?

A No, I don't. I do not find evidence of acute mercury intoxication in him, however.

Q Okay. The lack of evidence of acute mercury intoxication. Does that change your opinion at all about whether thimerosal-containing vaccines contributed to his regression?

A No, it does not. With thimerosal-containing vaccines and the mechanism as we best understand it, the concern is for relatively low amounts to cross

into the immature brain and incrementally increase over time with a great difficulty in getting it out of the brain, a very long half life.

So we have never intended to imply acute mercury toxicity. Our concerns are much more with chronic exposure that accumulates at a critical developmental window and so in this particular case one of the things that concerns me now is that he received a very large load very early -- the Hepatitis B at birth, 13 days and seven weeks -- and then the other routine immunizations.

So at a time when his brain was undergoing a tremendous amount of important work, he received thimerosal-containing vaccines.

Q And again what is your opinion as to what the mechanism of injury is in this particular case? That is, is it an acute exposure to mercury or is it something else?

If it's something else, exactly what is your medical opinion here as to what contributed to Colin's autism?

A My opinion is that he received a series of thimerosal-containing vaccines and that he was subject over time to accumulation in his brain; that it was a chronic exposure, not acute, and that his symptoms

manifested later as a result of this chronic deposition in his brain, kidney, fat and potentially lymphatic glands.

Q Finally, you were asked a series of questions after each of these lab results as to whether that lab result is indicative of either autism or mercury body burden. Do you remember those questions?

A Yes, I do.

Q Is it your testimony today that you're relying on any one individual test to inform and base your opinion on?

A No. Quite the opposite. It's the constellation of laboratory values in conjunction with the most important piece, which is the history of the child.

There is no easily available biomarker for autism that I'm aware of. I've talked to a lot of researchers about that very issue.

Q Would it be fair to say that it's the collection of this wide range of tests that informs your opinion rather than any one test result in and of itself? Is that fair?

A That's absolutely correct, but the pattern that is striking to me in this case is the number of

different labs that collectively support a picture of a child with known mercury exposure, known excretion of mercury with provocation and then multiple other lab tests that would be evidence of the metabolic processes going on in his body that were either causally or subsequent to those kinds of problems with toxicity.

MR. POWERS: I have no further questions. Thank you, Special Master.

THE COURT: Mr. Johnson?

RE-CROSS-EXAMINATION

BY MR. JOHNSON:

Q Dr. Mumper, with respect to the September 22, 2002, post-provocation mercury test you just testified that it's your belief that that result is abnormal, correct?

A That's correct.

Q There is no data that would support that statement. Is that correct? There is no data to show what normal reference ranges would be for post-provocation testing. Is that correct?

A To my knowledge, that is true.

MR. JOHNSON: Thank you.

THE COURT: Let me get this straight, Dr. Mumper. I want to make sure I understand that.

If I took 100 three-year-olds off the street out in front of the White House today and we chelated them, you're telling me that there is no data that would give us a reference range for where they would fall on mercury post-chelation?

THE WITNESS: I'm not aware that that has been done. It desperately needs to be done. One of the things that we are doing at our research institute is to try to compare porphyrin testings in normal children versus controls because that data has not been established.

It's classically hard to get people to volunteer their children at very young ages for research experiments in which they're being used just to set a control -- I've tried to do it in my practice -- especially if it involves anything either invasive or troublesome like taking home a kit and collecting a first morning urine and bringing it back.

It's difficult to get people to participate in that, but I agree that it definitely needs to be done.

THE COURT: Okay. And there is no data then that would show in anyone the increase between pre-chelation and post-chelation levels of lead or mercury?

THE WITNESS: There is data that shows that it increases, but the quantification of the amounts that correlate with a specific body burden have not been determined, to my knowledge.

THE COURT: When we chelate and we measure the amount of mercury excreted afterwards -- mercury, lead, whatever heavy metal --

THE WITNESS: Right.

THE COURT: -- I understood that to be a measurement of body burden of mercury.

THE WITNESS: It is reflective of an increased body burden.

I'm saying that what I don't have is the data to tell you that a four-year-old child would go from .01 micrograms per gram of creatinine to 17 micrograms per gram of creatinine if he had a total body burden of X grams of mercury. I don't know how to get that information.

THE COURT: What I'm having trouble understanding is why you can say that 17 is extraordinarily high. What do you base that on?

I'm not arguing with you, Doctor. I just want to understand --

THE WITNESS: Yes.

THE COURT: -- what the basis for the

opinion is if there is no reference.

THE WITNESS: The basis for my opinion I would have to say is discussions with leaders in the toxicology field and extrapolations from experiences in older populations, but there is a dearth of that information in the pediatric population.

THE COURT: Okay. Questions from either side based on mine?

MR. POWERS: Not from the Petitioner, Special Master.

THE COURT: All right.

MR. JOHNSON: Nothing further for Respondent.

THE COURT: Dr. Mumper, you may step down.

(Witness excused.)

THE COURT: Mr. Powers, Mr. Ferrell, where are we going from here?

MR. POWERS: Well, Dr. Mumper was the last of the three witnesses Petitioner planned to call in this case, so we're done with our case in chief in Colin Dwyer's claim for compensation.

THE COURT: Okay. Government, are you prepared to proceed with your first witness?

MS. RICCIARDELLA: Your Honor, I know it's a little early for a lunch break, but if we could have a

break before we put on our witness?

THE COURT: How much time do you need? It is early for a lunch break, and I anticipated that we would be pushing on through lunch in order to ensure that we get Dr. Leventhal out of here.

MS. RICCIARDELLA: If we're going to push on through lunch, if we could have a half an hour now? My direct is not going to be that long.

THE COURT: Okay. You're the one who has to get him out of here on time.

MS. RICCIARDELLA: I understand.

THE COURT: So if you need a half an hour, we'll reconvene then at let's make it five after.

(Whereupon, a short recess was taken.)

THE COURT: All right. We're back on the record.

Dr. Leventhal is on the stand. Would you raise your right hand?

Whereupon,

BENNETT LEVENTHAL

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

THE COURT: Thank you.

Ms. Ricciardella, you may proceed.

DIRECT EXAMINATION

BY MS. RICCIARDELLA:

Q Good morning, Dr. Leventhal. Would you please state your name and current position for the record?

A My name is Bennett Leventhal. I'm a Professor of Psychiatry at the University of Illinois College of Medicine in Chicago.

Q And could you please spell your name for the record?

A My first name is spelled B-E-N-N-E-T-T, and my last name is spelled L-E-V-E-N-T-H-A-L.

Q And would you please briefly review your educational background since high school?

A I completed my undergraduate -- well, sort of completed my undergraduate -- training at Louisiana State University. Then I went to medical school at Louisiana State University in New Orleans.

I was a house officer the first year at Charity Hospital in New Orleans and completed my residency in general psychiatry and child and adolescent psychiatry at Duke University in Durham, North Carolina.

Q And do you hold any board certifications?

A I'm board certified in general psychiatry, and I'm also board certified in child and adolescent

psychiatry.

Q And do you hold any licenses?

A I'm licensed to practice medicine in North Carolina, Virginia, Louisiana, Indiana and Illinois.

Q And would you briefly describe your academic employment history?

A When I finished my residency I was on the clinical faculty at Duke while I was in the Navy and was also on the faculty at Eastern Virginia Medical School.

Then I moved to the University of Chicago, starting there part-time in 1978, full-time in 1980, and I remained there until 2005 when I moved to the University of Illinois.

Q And are you a member of any professional societies or organizations?

A I'm a member of a lot of them.

Q Highlight a few for us, please.

A Probably too many. American Psychiatric Association, American Academy of Child and Adolescent Psychiatry, American Association for Advancement of Psychiatry, Society for Biological Psychiatry. That's probably enough.

Q And have you ever been honored for your work in autism specifically?

A I have been fortunate enough to be honored a couple times.

Q Could you just describe a few of those honors?

A I've received awards from the organization called MAAP, which is the More Able Autistic Persons, higher functioning autistic individuals. It's the families. I've been honored by that organization.

I recently learned that I'm going to receive an award from the American Academy of Child and Adolescent Psychiatry for lifetime achievement in work with individuals with developmental disabilities.

Q And when did you learn about that honor?

A Last week.

Q You mentioned that you're currently at the University of Illinois at Chicago. Do you hold any teaching positions there in your specialty?

A I'm a Professor of Psychiatry.

Q A full professor?

A I'm a full professor with tenure.

Q How long have you been teaching?

A Well, I started teaching when I was a resident, but I've been teaching for 30 years or more.

Q Who do you teach?

A I teach residents in general psychiatry,

fellows in child and adolescent psychiatry, medical students, residents in pediatrics, nursing students, social work students, students in psychology, and then I have graduate students who work with me on their PhDs.

Q And what do you teach?

A I teach broadly in the area of child and adolescent psychiatry, but probably spend most of my time teaching about developmental disorders and issues in normal and atypical child development not just restricted to autism and developmental disorders, although that's a large portion of my work.

Q Do you teach the diagnosis and assessment of autism and other autism spectrum disorders?

A I do.

Q Do you teach internationally?

A I do.

Q Could you explain how you teach internationally?

A I'm involved in a couple of organizations that are interested in advancing child and adolescent psychiatry practice and research and so I work with a group in Europe called the European Academy of Child and Adolescent Psychiatry.

I also work with a group in the Middle East

called the Eastern Mediterranean Association of Child and Adolescent Psychiatry where I go every year and teach. We've done a lot of work in autism and developmental disorders there.

And then I do some work in Korea, and I've also taught in many other countries -- China, Japan, Australia, New Zealand.

Q And who do you teach when you teach internationally?

A Mostly physicians both in psychiatry, but also pediatrics and pediatric neurology, but also psychologists I suppose and then students in each of those places.

Q Do you also give lectures to professional groups or organizations about autism spectrum disorders?

A I do.

Q To whom?

A Again, mostly to medical groups, although also to psychologists, educators, special educators in particular, but physicians in psychiatry, child and adolescent psychiatry and pediatrics, pediatric neurology primarily.

Q How often would you say that you give lectures?

A It would be unusual for me to go more than a week without giving a public lecture. Maybe two weeks. I suspect I give somewhere in the neighborhood of 100 per year.

Q Do you also lecture internationally?

A I do.

Q Do you devote time to family-based organizations pertaining to autism?

A I do.

Q Could you explain what you do?

A Well, I work, as I've indicated before, with the MAAP organization, with the Autism Society of America, with the local autism societies in Illinois, not just with the state organization, but the regional organizations.

I work with them and occasionally in other states, particularly in Indiana, Iowa, Missouri. I work with folks in those areas. I'm from Louisiana, so occasionally I go back home and help out there, even more so since the hurricane because they've had a shortage of folks.

Q I'd like to talk about your experience as a child psychiatrist over the past 30 years, specifically as it pertains to autism spectrum disorders. Do you currently have a clinical practice?

A I do. I'm not in private practice. I never have been. I'm in the university-based practice, but I see patients through that practice.

Q Could you describe your clinical practice?

A Well, when I'm in town and not lecturing or doing other things I probably see about 20 hours of patients a week. Probably three-quarters of those are developmentally disabled individuals.

Q Are you affiliated with any hospital?

A The University of Illinois Hospital, and I'm also affiliated with a local hospital in Chicago called Chicago Lakeshore Hospital where we have our teaching inpatient service.

Q As part of your clinical practice, do you diagnose children with autism spectrum disorders?

A Yes, ma'am, I do.

Q Approximately how many times have you diagnosed a child with an ASD?

A I'm sure it's thousands.

Q Over the course of your career?

A Yes, ma'am.

Q Approximately how many do you diagnose per month?

A Well, it sort of depends on the month and what my travel schedule is, what we're doing at the

time.

Right now we're engaged in a very large study and so I might see as many as two to three new cases a week when I'm in town. Sometimes it's as little as one to two a week or one a week. So somewhere between 50 and 200 new cases a year.

Q As part of your clinical practice, do you treat children with autism?

A Yes, ma'am.

Q Approximately how many are you currently following?

A Well, since they never go away they're with me forever, which is great. I follow all my kids into adulthood.

So if you do it long enough, 30 years, I have probably 400, 500, 600 kids. They're not all kids anymore, but they're kids to me.

Q What's the age range of your patient population?

A One and a half to 50, 60.

Q Do you meet with parents as part of your clinical practice?

A Absolutely. You can't practice without parents.

Q Why is that?

A Well, there are lots of reasons. First of all, they're the ones who are in charge. They make the decisions.

Secondly, we need them. I mean, they're the ones that have the information. They know the child far better than we ever will, and they are the ones that end up having to bear the burden so supporting them, making sure they understand what I understand and I understand what they're thinking and feeling for themselves, their child and their other kids is part of the practice.

One of the problems in autism practice is that the stress on families is just gigantic. There are very high divorce rates, very high stress levels in the families, and so if we're going to treat the child we have to manage the stress levels in the family and keep the families together. It's an inherent part of the practice. You can't do it without it.

Q Do you meet with other family members besides the parents?

A Always. When I get done with an evaluation, one of the things I do is I'll meet with the parents, go through our findings, but then I routinely offer to meet with everybody in the family so it's not uncommon

that grandmothers, aunts, uncles, grandfathers, cousins will come in. We sometimes have to use a large conference room.

I explain to them what the disorder is and what we understand about it, what the treatment is going to be, and then I ask them to play a role both in supporting the family, but also sometimes there are things they can do quite specifically.

We also pay particularly close attention to the children, the siblings. It depends on the developmental level and so on, but they bear a significant burden as well, and to the extent that they can understand we want to explain it to them.

To the extent that they want to help, we want to help them help. To the extent that they want some of their own time away from it we help set that up, so we have to include them in the process as well.

Q When a child is brought to you for an evaluation, are you the one who makes the evaluation?

A Yes.

Q Do you take the history yourself?

A I take it myself. I do my own physical exams, and I do the entire process with my own hands, eyes and ears.

Q Now, you mentioned that you currently have a

study underway. Do you also have a research practice?

A I have a rather large research practice.

Q Could you describe your research practice?

A Well, we're doing a number of studies. Right now I'm a part of one of the five NIH designated autism centers of excellence, so we have --

Q What's an autism center of excellence?

A The National Institutes of Health a couple years ago decided that they needed to create centers that had the capacity to bring a lot of expertise to bear on the study of autism, and they had a competition amongst various academic centers around the country.

There were five or six selected to receive large grants to set up the infrastructure to provide research support in autism, and we were one of those centers.

Q And when you say we, who is we?

A Well, there are a large group of us scientists and clinicians who are involved in the center. Ed Cook is the principal investigator. I'm one of the co-investigators. I run actually the clinical core for that center, so I'm responsible for all the evaluations, for all the patients that are in those studies.

And then we are doing a number of research projects ranging from MRI studies, brain imaging studies, to pharmacogenetic studies, understanding how genes may predict the response to certain medications, understanding some of the very critical elements of the disorder.

One in particular is the difficulties with insistence on saying that it's the inflexibility of the peculiar stereotype behaviors that are an inherent part of autism are often quite disabling, so we're trying to understand not only the biological substrates of those, but perhaps how that contributes to the genotype of the disorder.

And then we're doing also some preclinical studies with animals. We're working on a project to try to breed animals that may exhibit some of the symptoms of autism.

Obviously mice and rats can't do the same thing, but if we can build some models then we may be able to think about both understanding causality, but also specific kinds of treatment for specific symptoms.

Q Were you one of the authors of the Autism Diagnostic Observation Schedule, also known as ADOS?

A Yes, I was.

Q What did your participation entail with that?

A Well, from the beginning where we had to determine the items, the presses that are used in that, then executing them, then assessing whether they were working or not and then restructuring it and doing it again until we found an instrument that was highly reliable and valid. That was through most of the process.

Q Now, according to your CV you've published over 120 articles related to child psychiatry. Does that sound correct?

A Yes. Probably a few more since then.

Q Are those all peer reviewed?

A Yes.

Q Do any pertain to autism spectrum disorders?

A Yes.

Q According to your CV, you've also published 20 books and book chapters. Does that sound correct?

A That's about right.

Q Do any pertain to autism spectrum disorders?

A I'm sure some of them do.

Q Now, according to your CV you currently serve on the Panel of Professional Advisors of the Autism Society of America. What is the Autism Society

of America?

A It's a very large parent organization, an advocacy group that's concerned about the research, but also services, legislation related to individuals who have autism.

Q And what does it mean to serve on the Panel of Professional Advisors?

A I was invited to be part of a group that advises the organization on scientific matters, so they send questions to us, ask us to help them try to set scientific policy. We provide them advice and guidance. The organization sets it. It's not ours to do.

Q Your CV also states that you're currently a member of the Board of Advisors of the Association for Science and Autism Treatment. What is that?

A That's another organization that's trying to look at evidence-based practices. There's a group of advisors to them who review studies and try to help them ascertain whether they meet a sufficient scientific standard to become part of practice.

Q Are you a reviewer for any journals?

A I review for lots of journals.

Q Could you name a few?

A *American Journal of Psychiatry*, *Archives of*

General Psychiatry, *Journal of the Academy of Child and Adolescent Psychiatry, Journal of Child Psychology*, *Psychiatry in Allied Professions*, *New England Journal of Medicine*, *Journal of the American Medical Association*, *Pediatrics*.

Q That's enough.

A Too many.

Q Now, your CV has been filed as Respondent's Exhibit DD in this case. Is Respondent's Exhibit DD an accurate summary of your publications, background and experience?

A Yes, ma'am.

Q Doctor, have you ever testified before in a Court of law?

A I have.

Q Approximately how many times?

A Maybe 15 or 20 times.

Q Could you describe the types of cases?

A Probably the two most common are cases related to child abuse and neglect or marriage and divorce cases. I've also been involved in a few other odds and ends.

Q Have you ever testified in the vaccine program before?

A No, ma'am.

Q Have you ever consulted for a pharmaceutical company?

A I have.

Q Could you explain?

A I've consulted with pharmaceutical companies on study design and most recently with Johnson & Johnson as we tried to help them bring Risperdal into the market. It was the first drug that has an FDA indication for autism.

They were going to drop that because it was about to go off patent and so they tried to move it along, and some of us helped consult with getting that through the FDA process so we now at least have one drug that's officially approved for autism.

Q And why did you agree to testify for the United States Government in this litigation?

A There are two reasons. One is a number of my colleagues asked me to do it and said that it was important, but I'm very concerned about families with kids with autism, and I'm concerned that they might be being led down the wrong track.

We work too damn hard to take care of them to see them waste resources on things that are not helping them and to put their kids in jeopardy. It's just not something I can stand and so I think there's

a chance to try to make a difference so I'll try.

Q I'd like to turn now to the facts of this case, to the medical facts of Colin Dwyer. Did you review the medical records that have been filed in this case?

A I reviewed the materials that were given to me. Yes, ma'am.

Q And did you listen to the testimony of Maria Dwyer and Timothy Dwyer yesterday?

A I certainly did.

Q Were you present in the courtroom?

A Yes, ma'am.

Q And did you review the affidavits of Maria and Timothy Dwyer?

A Yes, ma'am, I did.

Q Did you read the medical report filed by Dr. Elizabeth Mumper in this case?

A Yes, ma'am, I did.

Q And were you present in the courtroom today to listen to Dr. Mumper's testimony?

A Yes, ma'am, I was.

Q In your opinion, Doctor, did Colin's receipt of thimerosal-containing vaccines cause or contribute to his autism?

A No, ma'am.

Q Do you agree with the diagnosis of autism in this case?

A I think it's highly likely, but it's not definitive.

Q In your opinion, has proper testing been done on Colin?

A No, ma'am.

Q Could you explain what you mean by that?

A Well, there are standard diagnostic procedures that are pretty much well accepted around the world actually for the proper diagnosis of autism, and the gold standard is using the Autism Diagnostic Interview, the ADI, and the ADOS, the Autism Diagnostic Observation Schedule, jointly. But then you have to use collateral measures as well.

Some of them have been done with Colin the Vineland social maturity scale, but cognitive testing is also an inherent part of the process because one has to be able to adjust the perspective on symptoms based on cognitive functioning and language ability, and cognitive testing, proper cognitive testing, hasn't been done.

Q Over the course of your clinical practice, have you evaluated and treated children with the same symptoms as described in Colin Dwyer's medical

records?

A I would say Colin Dwyer's medical record is basically the record of most of the cases I've ever seen.

Q Is there anything unique or different about Colin's clinical course than in the autistic patients that you have in your practice?

A No, ma'am.

Q Are you familiar with the term regressive autism?

A Yes, ma'am. I've heard it.

Q When did you first hear that term?

A It started in the late 1990s, early 2000 range.

Q In what context? Do you know?

A Well, when Andrew Wakefield was trying to make his case the notion that there was a unique group of individuals who had a regression as part of their disorder that was separate from the rest of autism became part of the discussion. It never really entered the scientific community.

There's no formal diagnosis called regressive autism, and most of us have not -- despite the fact that we tried very hard to see if this was a distinct phenotype, we haven't been able to support

that. It would be useful if it was a distinct phenotype, but it turns out not to be.

Q Do you use the term regressive autism in your practice?

A No, not at all.

Q From your review of the evidence, would you characterize Colin as having suffered a regression?

A What I would characterize Colin as having is a progressive illness that included loss of some skills at certain points along the way, but it was a progressive process. It wasn't like he was motoring along and dropped off the edge of the cliff and then went forward.

Q How would you describe Colin's condition?

A Well, again I haven't seen Colin, but at least from the records it would sound like this was a child, as is often the case with autism, who had what was apparently a normal pregnancy and delivery with few odd things, completely nonspecific, that then progressed to look like he was a normal baby at birth, and then things start to give you a hint that something is not quite right.

Again, one has the advantage of 20/20 hindsight as well, but when you look back the growth curves start to slip shortly after six months.

Q I'd like to ask you about that because in your report you state that Colin's behavioral aspects associated with ASD may have begun as early as six months of age when he started to lose weight as demonstrated by growth charts.

A Correct.

Q Could you explain what you meant by that in your report?

A Well, if you look at his growth charts he starts off with everything -- his height, weight and head circumference -- are all tightly linked together, and then starting shortly after six months, at least from the pediatrician's record, his weight starts to fall off, but his height and head circumference stays the same. They only start to fall off some months later.

It's not uncommon for us to see kids with autism start to become finicky eaters even as early as four, five, six, seven, eight, nine months and so it's quite possible that the behavior was subtle and no one might have noticed it. It was just he was just a little bit of a picky eater.

It may have already started to affect the way he was eating or how much he was consuming. That may have been the beginning of the falloff and the

progression of his illness.

Q You also state in your report that Colin has yet to receive fully appropriate cognitive assessments. Could you explain what you mean by that?

A Yes. I mean, the problem is that cognitive functioning -- people in the lay public sort of think of cognitive functioning as measured by IQ. IQ is a single number, and it's not a very useful single number.

It would be kind of like telling you the score of a baseball game last night was seven. You know, does that mean one side had seven and the other had nothing or four plus three? I mean, you don't know anything.

IQ is like that, but as it turns out there are elements of cognitive functioning that are really quite crucial for adaptation, and when one designs intervention you have to know those elements of functioning because you want to build on strength, and you need to work around weakness.

So when we do testing what we want to do is look at an individual's verbal cognitive abilities, the things that we depend on language to manage, but then we also want to look at nonverbal cognitive abilities, and those are things like mathematics or

problem solving, puzzles and things like that.

We sometimes can take problems and by teaching if someone has verbal deficits, which is common in autism, and we understand what the verbal cognitive deficits are in an individual we can sometimes twist those very tasks into nonverbal tasks and teach them how to manage certain things.

There's another part of it, and that is you need to know what someone's cognitive level is. If you think someone is a genius but in fact they have an IQ of 50 or 60, making demands of them for an IQ of someone who has 130 or 140 is unfair and reasonable. As is commonly the case, it's also unfair and unreasonable for the families because it creates a set of expectations and demands that may be unreasonable and then they have a sense of failure and not succeeding.

So trying to understand really where the child fits developmentally at a level of cognitive functioning, as well as language functioning, as well as adaptive functioning, as well as behavioral functioning, is a critical part of getting the whole clinical picture.

Q Now, in your report you state that Colin has autism spectrum disorder, likely comorbid, with

moderate mental retardation.

We heard testimony yesterday and today by Dr. Mumper that questions that conclusion. What's the basis for your conclusion that he may have moderate mental retardation?

A So again because appropriate testing hasn't been done I can't say that for sure, but there are three pieces of evidence, maybe four.

Piece of evidence No. 1 is that autism is commonly comorbid with mental retardation. It depends on what studies you look at or what the surveys are. Between 70 and 80 percent of individuals are comorbid with mental retardation. A range, but they're comorbid.

Secondly, there were two tests that were done in the record. One was the Bayley Scale of Infant Development. While it's not a great indicator of cognitive ability, when those were done, and I don't remember exactly when they were done, but the bayley, think the standard score was a 56 or 57, which would be in the moderate -- it correlates roughly with IQ.

Remember, I'm dubious about single numbers, but that would correlate roughly with an IQ in the 50s or maybe low 60s, which would be mild to moderate

mental retardation.

Similarly, there was a Stanford-Binet test done at one point, which is also a cognitive measure, and that was in the same range. Again, Stanford-Binet has a set of problems: A single number, how much is verbal because he has verbal problems and understanding the verbal testing and so on is an issue, but at least it points in that direction.

And then if you look at his Vinelands repeatedly, his adaptive functioning, the scales, they're all in a rather low range, which would put him again consistent with someone who had mild to moderate mental retardation, probably moderate.

But in the end until one does the right test you can't say for sure, but all those indications would strongly suggest that you need to do those tests so you know what you're dealing with.

Q We heard testimony that because Colin responded to PECS therapy that that is evidence that he has proper cognitive functioning. Do you agree with that?

A It's not an indicator of that at all. We use PECS for individuals with mental retardation as well.

Q What is PECS?

A PECS is P-E-C-S. It's Picture Exchange Communication System. Basically you give individuals -- you can do it in a number of ways, but they're basically drawings that an individual can pass to somebody to say I want a cookie, so they go through and they give you a thing for a cookie. You can use it for schedules and tasks.

It's a way of communicating that doesn't require the production of words. You largely use it as iconic images, although -- I mean pictures, sometimes literally photographs and sometimes ideographic drawings.

For some kids you use words as well as the drawings because they can read, but they can't speak the words. Different groups of kids can use this kind of exchange system as a way to communicate along the way.

The reason it's so important is communication is crucial because often times we see kids with autism who have difficulty communicating. It becomes very frustrating, and that leads to serious behavior problems because they can't tell you what they want or how they're feeling, what they need or that they don't want to do arithmetic or they don't want to go outside, so if they can show you that

sometimes it --

Or when you can give them a choice of options that they want to pursue, it sometimes makes it a lot easier for them a lot of times, and it makes it a lot easier for the rest of us because we know what they're thinking and feeling. We can then interact with them.

Q And is PECS used for children with mental retardation as well?

A All the time. Sure.

Q You also mention in your report that Colin has not had appropriate genetic testing. Could you explain why that's important?

A Well, first of all, the current best view of autism is that it's a genetic disorder, but, more importantly, there are a number of genetic conditions that are associated with autism specifically, and for those there are discrete genetic markers and we'd want to know that.

In particular, Fragile X syndrome, which is highly associated with autism. There's a genetic abnormality on Chromosome 15q that is associated with autism, and it's important to know that because people who have a 15q duplication are at high risk for sudden death and we need to monitor them for cardiac

deficits, so we want to check for that.

And then tuberous sclerosis is associated with autism. There are genes for tuberous sclerosis, and those need to be looked for. There are a number of other possible genetic variants that are associated with the disorder, and they're coming faster and faster and faster so in the next year or two we'll probably have more variants that we'll know about and going ahead and doing the testing so we have markers. It helps us know which kids to call in when we have a finding.

For example, we discovered the 15q abnormality, and then we discovered the sudden death thing, so now we can go to our registry of all the kids that we've seen and tested, and all the ones that are 15q we've notified all those families that they're at increased risk for sudden death so that we can deal with that.

The same thing with Fragile X. It's not here yet, but there's a treatment in animals with Fragile X that actually remediates some of the disabilities associated with Fragile X. I would expect in the not too distant future we'll see human trials.

Being able to find those families quickly

and say your child has Fragile X, here's the study, here's a possible treatment, we want to be able to make that available as instantly, as quickly as we possibly can.

Q The fact that none of Colin's physicians have recommended that genetic testing be done, is that evidence that the testing is unnecessary?

A No. I mean, it's completely necessary. For me the goal is saying what would I do for my child? I would definitely have my child genetically tested.

I mean for all the blood draws and sticks he's had, you know, you could get the blood easily. Frankly, you could do it from a swab of his cheek, so it's noninvasive, easy to do and not terribly expensive, given what else has been spent on him.

Q Doctor, Dr. Mumper relies very heavily on the various laboratory reports in this case to support her hypothesis, and she talked today that she doesn't rely on single laboratory results, but a constellation of labs or the labs in concert.

If this were your patient and you were presented with these laboratory results, what would you recommend be done?

A In response to these laboratory results?

Q Yes, sir.

A I don't think any of them are particularly pertinent to this clinical case. I probably wouldn't have ordered most of them.

The ones that are there where there are abnormalities, the so-called abnormalities are at the margins, but even then one of the things we were taught in medical school and we teach our students is that clinical practice isn't driven by a lab test.

Clinical practice is driven by the care of the patient, so you have to take the laboratory finding and correlate it with some kind of finding in a patient. Just because you have an abnormal lab finding or particularly a marginally abnormal lab finding it may have no relevance at all to the practice and what you actually do and what it means in terms of the causal relationship of the patient.

In this case, looking at all these labs, I didn't find any of them particularly relevant to the case at hand.

Q If Colin were your patient, would you order or in any of your autistic patients would you order that such labs be done?

A I want to say something that may be a little bit odd, but we don't actually -- I don't use the term autistic patients. How about children who have

autism, because they're children. They're people first. They happen to have a disease, but they're first of all people.

I think to just say they're autistic is too dismissive and not fair. And so they often have ideas and opinions, a sense of humor, preferences, and I think we often forget that because we think they just sit in corners and twiddle, and that's not what they do. Sorry.

Q No. That's an excellent point. Let me rephrase. The autistic children that you have in your practice. Would you order such laboratory testing be done?

A For children with autism, I wouldn't order most of the laboratory tests that were ordered here. They're just unnecessary. There's some that might be useful, but most of them are not useful.

Q If you thought that a child with autism had neuroinflammation, what would you recommend be done?

A Well, I would do several things. First of all, I would consult with a neurologist. Although I do a lot of work with these kids and basically know what to do, I always think two heads are better than one so I might as well get someone else to think along with me. I would do an LP though.

Q What's an LP?

A Lumbar puncture. I'd get spinal fluid. I mean, why take a peripheral measure or a guess when you can go as close to the source as you can and look in the spinal fluid? You can almost always when there's inflammation find that.

And then I would probably seriously think about doing a brain scan of some kind. In the kinds of inflammation that have been talked about here, you almost have a high probability of finding that on an MRI, or there are other scanning techniques that one could use to see things like gliosis or inflammation in certain areas.

Q Doctor, if you saw a blood test that was four times the normal range would that mean that the results were abnormal?

A No.

Q Why not?

A Well, because why something is abnormal, why something has a particular value, depends on lots of factors.

Let me give you an example. If we had a child who had not eaten -- you saw him first thing in the morning and hadn't eaten all night -- you might get a blood sugar of 60, 70, and then you gave him a

bowl of Frosted Mini Wheats and took his blood again. He'd have a blood sugar of 300, 350.

That would be completely normal because in the context in which that occurs it could be completely normal. You have to understand both, if you will, the metabolism of sugar and how that gets managed, but also the conditions, the context in which it took place.

Q Just because a child is smaller than an adult, does that mean that the reference ranges for their laboratory values would be lower?

A No, not at all. I mean, you can't make that assumption at all. In some cases their reference ranges are higher. In some cases they're lower. It depends on what the measure is.

For example, some liver enzymes kids actually their livers work a lot better than adults because they haven't been damaged by alcohol and cigarettes and all the stuff that adults do to damage their livers and so they actually can metabolize drugs faster in some cases and their liver enzymes may be higher. In some cases they're lower. It depends on what the particular index is and also depends on a particular level of functioning.

For example, a child who's crying, their CO_2

level will be different than a child who's not crying, or a child who's been running around a lot, their lactic acid level will be different so it's context and what the pathophysiology of the particular measure is. You need to know both.

Thirdly, you have to know development, developmental age. Things change over time.

Q In your report you also mentioned that a dysmorphology exam was not detailed enough or was unclear from the records how detailed the dysmorphology exam was.

Why is that an important examination that should be done on a child with autism?

A Because the way we look in our body forms often is reflective of events that may have occurred in utero, some of them genetically determined, some determined by other factors as well.

And so when we have a child who has a developmental disturbance because external manifestations of the central nervous system may be seen in skin development or in development of particularly the face and head, we have to look very, very carefully to make sure that there aren't any abnormalities that are commonly associated with syndromes.

In fact, the pictures were not very acute and very difficult to discern because they went by very quickly yesterday, but just in my quick look at it it may be that his ears are a little bit low and turned posteriorally. That could mean something of profound importance clinically, and it looks like his eyes may have been a little bit wide set.

Well, someone has to sit down and actually do those measurements and look at other parts of his body. He could have certain kind of pigmentation that might be consistent with certain neurologic diseases that reflect themselves in the skin.

One is tuberous sclerosis. It requires something called a Wood's lamp examination to see if that pigmentation change is taking place in the skin. You have to look for all these things to make sure that you understand every possible thing that's associated with the disorder.

Q You're not diagnosing a dysmorphic condition in Colin based on the pictures, are you?

A No, no, no. Not at all. It would just for me reinforce my concern that it wasn't done meticulously.

There are people who are quite specialized at this. I'm okay at it, but if I wasn't sure I would

send him to a dysmorphologist.

Q Doctor, do you find any clinical or scientific evidence in this record that would lead you to believe more probably than not that Colin's autism is caused by thimerosal-containing vaccines?

A I don't think there's any evidence that it's caused by thimerosal-containing vaccines.

Q The hypotheses that Dr. Mumper has put forward in her report and that she testified to today regarding her belief that Colin's autism was caused by thimerosal-containing vaccines, are those hypotheses generally accepted in the autism medical community?

A No, they're not.

MS. RICCIARDELLA: I have no further questions.

THE WITNESS: Thank you.

THE COURT: Mr. Powers, do you need a recess before we begin?

MR. POWERS: You've taken the words out of my mouth, Special Master. I would appreciate a recess.

THE COURT: How much time would you like?

MR. POWERS: Can we take 45 minutes? That would give us a chance to actually grab a quick bite to eat. I don't expect my cross is going to be much

longer than the direct.

THE COURT: Any problem with that? That takes us to 1:00. You need to get Dr. Leventhal out of here by 3:00 as I understand it?

MS. RICCIARDELLA: Correct. That's okay.

THE COURT: Okay. All right. We'll reconvene then at 12:30.

(Whereupon, at 11:46 a.m., the hearing in the above-entitled matter was recessed, to reconvene at 12:30 p.m. this same day, Tuesday, July 22, 2008.)

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A F T E R N O O N S E S S I O N

(12:31 p.m.)

THE COURT: All right. We're back on the record.

Mr. Powers, feel free to cross-examine.

MR. POWERS: Thank you, Special Master.

Whereupon,

BENNETT LEVENTHAL

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

CROSS-EXAMINATION

BY MR. POWERS:

Q Good afternoon, Doctor.

A Good afternoon, Mr. Powers.

Q I was just going to introduce myself, but you've got my name already. I am Tom Powers. I'm one of the attorneys representing the Dwyer family and Colin Dwyer in particular.

At the outset, I wanted to ask a few questions with what you relied on to generate your expert report and what you relied on in your testimony today. Did you read any of the transcripts of hearing testimony for the King and Mead cases that were heard in these general causation proceedings earlier?

A No, sir.

Q Did you read any of the Petitioners' side expert reports that were submitted in the King and the Mead cases?

A No, sir.

Q Did you read any of the Respondent or government side's expert reports that were filed and generated in the King and Mead case?

A No, sir.

Q So the entirety of what you reviewed and relied on to generate your expert report and that you rely on in your testimony would consist of Colin Dwyer's medical records. Is that correct?

A Yes, sir.

Q And Dr. Elizabeth Mumper's report that was filed in this specific case, correct?

A Yes, sir.

Q And listening to Mr. and Mrs. Dwyer testify yesterday? Is that fair?

A Yes, sir, and Dr. Mumper this morning.

Q And then Dr. Mumper this morning. So aside from those, is there anything else that you relied on to prepare your report or to present your testimony today?

A Not that I'm aware of. No, sir.

Q Now, how long have you been practicing as a psychiatrist?

A I finished my residency and fellowship in 1978, so 30 years.

Q In the years preceding 1978, isn't it true that psychiatrists attributed autism in large part to what is called the refrigerator mother or the lack of affection, a lack of bonding? Was that the general cause of autism that was attributed in describing the etiology?

A No. That's not actually accurate.

Q Refrigerator mom was a descriptive term generated by Dr. Bettelheim sort of post Vienna, post World War II, to describe what he believed was the cause of autistic spectrum disorders. Isn't that correct?

A It was one of his concepts, but Dr. Bettelheim wasn't a psychiatrist. He was actually not even a psychologist. He was an educator.

Q And in the years since then that theory of causation has been disproven, correct?

A It was never proven, so other theories have taken form. It was never a proven theory.

Q The theory that you believe is that autism is entirely genetic? Do you believe that autism is

entirely genetic?

A No, sir.

Q Do you see room for environmental contributions to the appearance of autistic symptoms in some children?

A Yes.

Q Can you identify what you believe to be known environmental contributors to the appearance of autistic symptoms in children?

A Well, we know very well that the environmental interventions make a difference in the modification of environmental symptoms, so things like ABA affect the clinical presentation of the disorder.

Education, speech and language, change the clinical presentation of the disorder. Those are all environmental interventions.

Q And not speaking of environmental interventions, but you would agree with me that environmental exposures can actually be the biological cause of autism?

So, for example, prenatal exposure to thalidomide. Do you believe that prenatal exposure to thalidomide can cause autism?

A I think what you're trying to do is make a sweeping generalization. As I think Mark Twain once

said, no generalization is worth a damn, including this one.

I think generalizations just aren't terribly useful here. You have to talk about specifics, so if there's --

Q And that's why I asked --

A If I can finish my answer, I'd be happy to.

Q Well, I asked you a specific question. Do you believe that prenatal thalidomide exposure can contribute to the appearance of autism in some children?

A This is not a matter of belief.

Q Let me put it this way. As a scientist, would you recognize that there is an association between prenatal thalidomide exposure and the appearance of autism?

A What do you mean by association?

Q A causal relationship.

A That's not been demonstrated, so the answer to that is until it's demonstrated I can't really tell you.

Q Do you believe or do you think that the evidence supports an association between terbutaline exposure prenatally and the appearance of autistic symptoms?

A I'm not aware of any causal mechanism that would support that.

Q Are you aware of any scientific data or scientific literature that would support an association between maternal rubella and the appearance of autistic symptoms in the child?

A You just used the word association, so there are data on the association between maternal rubella and autism.

Q Would it be your scientific opinion that those associations are suggestive of a causal link between maternal rubella and the appearance of autistic features in some children?

A It's not been demonstrated, so until it's demonstrated I don't know whether there's a causal link. There's a big difference between association and correlation and causality.

Q And that's why I'm asking you specifically if you believe that there is a causal association between these various prenatal exposures and the appearance of autistic symptoms in children who were the product of those exposed pregnancies.

Do you believe that there is scientific evidence supporting a causal relationship?

A As I said to you, I don't believe. There's

what I know and what I don't know and what I'm not aware of.

I have no knowledge of a causal link between rubella and autism. There's an association, but there's not a causal link to my awareness.

Q You mentioned that you've diagnosed thousands of children as suffering from autism or having autism. Among those thousands of children that you have diagnosed as suffering from autism, what percentage of those children have a known, identifiable genetic cause of their autistic disorder?

A A very small percentage.

Q Can you estimate how large or how relatively small that is?

A One percent, maybe two percent.

Q You mentioned that you had testified in other litigation settings. You described child abuse cases. Do you recall that?

A Yes, sir.

Q And child custody cases?

A Yes, sir.

Q You also mentioned odds and ends of other testimony. Have you ever appeared as a witness in a civil lawsuit involving autism?

A I have.

Q What were the facts of that case, if you could briefly describe them?

A Sorry. Technically it was an autism spectrum disorder. It was a Rett syndrome case.

Q Okay.

A And it was a special education case.

Q And so would this be a dispute between parents and a school district attempting to get services for their children?

A In that particular one I'm thinking of, yes.

Q And what side of the case did you participate as a witness on?

A It was on the child's side.

Q Have you ever appeared as a witness in any litigation involving pharmaceutical companies?

A Not that I'm aware of.

Q Now, you did mention that you do consulting work with some pharmaceutical companies, with drug manufacturers?

A I've done a small bit. Not much.

Q Are you a member of the speakers bureau for any pharmaceutical company or drug manufacturer?

A Not that I'm aware of.

Q Let me ask you this. Do you receive research support from Abbott Labs?

A I don't personally receive research support. The university I work for receives, has contracts with them.

Q Do you receive research support from Eli Lilly or GlaxoSmithKline?

A The university has contracts to provide research.

Q Are you a member of the speakers bureau for Eli Lilly?

A I'm not aware that I'm a member of the speakers bureau for them. I have spoken at events that they've sponsored.

MR. POWERS: Okay. I took a look at an article on which you're the author.

Scott, do we have copies of that?

THE COURT: I'll tell you what, Mr. Powers. Let's just call this one 20, and we'll fill in any holes we have to later. This will be Petitioners' Trial Exhibit 20.

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

BY MR. POWERS:

Q Dr. Leventhal, what we have now described as

Petitioners' Trial Exhibit No. 20, you have a copy of that in front of you.

That's an article called An Open Label Trial of, and I'll ask you to say what that word is.

A Esataloprine.

Q In Pervasive Developmental Disorders. You're listed as one of the authors on that article. Isn't that correct?

A Yes, sir.

Q And then if you look at page 8 of 9 on this article there's a section called Limitations that's in bold italics, and then down underneath that there's a disclosure section. Do you see that disclosure section?

A Yes, sir.

Q Okay. In that disclosure section it says that Dr. Leventhal receives research support from Abbott Labs. That's a drug manufacturer, isn't it?

A Yes, sir.

Q It also says that you receive research support from Eli Lilly, GlaxoSmithKline, Shire, Pfizer and Forest Laboratories, correct?

A Correct.

Q These are all drug company pharmaceutical manufacturers?

A Yes, sir.

Q It also says that you're on the speakers bureau of Eli Lilly, GlaxoSmithKline, Pfizer, Bristol-Myers Squibb and that you have consulting relationships with Abbott, Eli Lilly, Janssen, McNeil, Pfizer and GlaxoSmithKline, correct?

A Right.

Q And I'm assuming this is information that you provided to the journal that published this article that you were a co-investigator of?

A That's correct.

Q So the information that's contained in this article, to the best of your knowledge, in 2004, which is when this was published, this information is correct as contained in the disclosure?

A It's correct, but I think what happens is when you fill out the form for the journal there are a limited number of things that you check. So if you speak for a drug company, it doesn't say speakers bureau. You just check that I've given lectures funded by the drug company.

I would point out that this, while it was published in 2004, it was really from 2003, and I'm not participating in many of these any longer.

Q Which ones of these are you still

participating in, and which are you not participating in?

A At this point the research support -- again, the research support is to the university. It doesn't come to me personally so I have no financial gain from that. It's just the way the contracts are written, and I'm not even the principal investigator on some of these studies. It's just we try to be as open as we can about possible perceptions of conflict.

And I don't speak for anybody else anymore with the exception of -- actually, I'm not speaking for anybody anymore. In the last year I did give some talks for Lilly and for Bristol-Myers Squibb. I don't have any consulting relationships at this time.

Q And the consulting relationships and the speakers relationships, those are things you would have been compensated for at least back when you were participating in 2004, correct?

A Not always. Sometimes they would be to the university.

Q There was some discussion about regression, and my recollection of your testimony is that you don't believe that there is a regressive phenotype of autism. Is that a fair summary of your testimony on that issue?

A Well, first of all, it's not a matter of faith. The data that we've looked at, and we've tried mightily to see if there was a phenotype of a regressive form of autism. The data just don't support it.

We've looked at it from a number of different perspectives, and just the data don't support a particular subtype of that sort.

Q I'm going to hand you a scientific journal article that was introduced into evidence in these cases. It's the Petitioners' Master Reference No. 72.

I know you're taking a look at that right now. Have you ever read this article before?

A I don't recall. I may have. I don't recall it though.

Q Are you familiar with any of the principal investigators, Drs. Pardo, Vargas or Zimmerman?

A I don't know them, no.

Q Do you know of them?

A Not really.

Q Okay. I'm going to direct your attention to if you look at the bottom right-hand, the pagination, Doctor, it will say page whatever of 12. Turn to page 9 of 12, please.

Now, if you look up at the top left-hand

corner there's what looks like a flow chart. Do you see that?

A Yes, sir.

Q And in the bottom right-hand corner of that flow chart you see a category called Autistic Phenotype. There's regression listed, there's epilepsy listed and there's mental retardation, correct?

A Yes, sir.

Q Would you disagree with the authors of this paper that there is an autistic phenotype that would include regression?

A That's not actually what you asked me before. Secondly, you said is there an autistic phenotype that includes regression, and for these authors I don't know because I haven't read the paper.

I can't really tell you what they mean by the term regression. If it means the loss of some acquired skills, then I would agree that some types of autism, some people with autism -- most people with autism -- lose skills as part of the progression of their disorder, but that does not mean that there's a unique regression or autistic regression phenotype.

It's just one of the parts of the progression of the disorder, just like seizures is

part of the progression of the disorder or mental retardation is part of the progression of the disorder. It doesn't mean it forms a unique phenotype, which is two different points.

Q Now, do you believe that it's possible, or actually not possible. Do you believe that some children actually develop normally, make completely normal progress even when looked at retrospectively, and then regress?

A Well, there is a disorder called childhood disintegrative disorder in which children are said to develop until the age of three and then lose skills.

Within the rest of the autism spectrum, our general impression is that when we look back carefully we almost always find a failure to progress appropriately, just like it was the case here, and so things just didn't fall off the edge of the cliff at the age of 20 months. There was progression.

There was probably some loss or change in eating changes or perhaps some other things, and his language didn't develop appropriately because he only had -- it depends on what you read 3 words or the mom's testimony yesterday -- 10 words at 20 months. He didn't progress.

So what we often see, and in fact there's

some studies that have suggested around 20 to 24 months parents start to really realize something is terribly wrong, and when they look back retrospectively they often find some of the early signs of the disorder beginning somewhere around 15, 16, 17, 18 months. That doesn't mean they weren't there before. It just means that they weren't necessarily seen before.

When we take careful histories or look at photographs, videotapes, we often find that there's some inkling that the problems began well before 20 or 24 months.

Q Now, you say often, but would you concede that there are some minority of cases of autism where even looking retrospectively and even vigorously looking retrospectively for lack of normal development before the regression that there are in fact cases, some percentage of children who regress, even retrospectively, you would agree had a normal course of development? Isn't that right?

A I think over the course of the past 10 or 15 years as we've really done a lot more work I would first say that that picture is exceedingly rare and that our general view is it probably is a more defect in collecting history or collecting data or an

inability to measure certain kind of things in preverbal children.

Now, that's starting to change as we have new kinds of measures and we're starting to be able to diagnose earlier and find particular symptoms that may appear earlier that are continuous with the symptoms we see in children at two, three and four.

So the answer to your question succinctly is is it possible? Yes. Have I seen cases where it wasn't evident earlier? Yes, but it's pretty rare, and as we get more sophisticated at identifying behavior and looking at development it's becoming even more rare.

Q Do you know Dr. Rust at the University of Virginia who practices in Charlottesville?

MS. RICCIARDELLA: Objection, Special Master. I mean, this is supposed to be specific causation as to Colin Dwyer. We're 20 minutes into cross-examination, and Mr. Powers hasn't asked one question as it pertains to Colin Dwyer.

MR. POWERS: This is about his direct testimony on regression.

THE COURT: I'll permit the questions. Go ahead, Mr. Powers.

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BY MR. POWERS:

Q Do you know Dr. Rust?

A I do not.

Q You do not. Do you have any knowledge that Dr. Rust was called as an expert witness on the government's side of these cases, in William Mead and Jordan King's cases, that were heard back in May?

A As I told you, I know nothing about those cases.

Q So if Dr. Rust, as a clinician and a pediatrician and an expert in autism, identified that about 20 percent of his patients even retrospectively showed normal development, no early problems and then regressed, would you dispute what Dr. Rust finds? Was that 20 percent something that you would take issue with?

A I can't dispute what Dr. Rust told you because that's what he told you. However, what I think I just said, but I'll repeat it for you, is that over the course of the last 30 years of my practice in the beginning we used to think it was about a third of the kids had regression, but it was a defect in measurement and in history taking.

As we've gotten more proficient at it then that number went from 30 percent to 50 percent to 70

percent to 90 percent and so what's happened as we've learned more about the disorder, more about its progress and we've become more sophisticated at measuring behavior, cognition and language in very young children that our picture has changed.

And so that's why I arrive at the conclusion that I rarely see it because we're very good at taking early histories and very good at measuring early behavior. And, secondly, that in the rare instances where we don't have discrete evidence we assume it's actually our failure to find it rather than the fact that there was no failure to progress.

Could I be wrong? Yes, but I don't think so because the trend has been moving to make that 30 percent, 50 percent, 70 percent, and I think probably Dr. Rust is a pediatrician. He doesn't do what we do. I don't know.

I mean, I don't know exactly what he does, but my guess is that if in his experience as he got more sophisticated doing early childhood evaluations that number will squeeze smaller and smaller as well. It's just the progress of science. It's not anything else.

Q In Colin Dwyer's case, the one point in the medical record that I recall you cited to as lack of

not normal progress was a change in his weight, the weight growth chart. Do you recall that testimony?

A Yes, sir.

Q Now, in reviewing the expert report, I didn't see any other reference specifically to the medical records indicating anything that would support the contention that Colin was not developing normally up until about his second year of life.

Can you point us and point the Special Master to something specific in the medical record showing that Colin Dwyer was failing to make normal progress?

A Yes, I can. Let me take three points. Point 1 is the growth chart, which is multiple measures beginning early on.

You see that his weight falls off over the course of time, and then his height and head circumference drops to catch up with that lagging behind, as is often the case when children aren't eating well. By the way, that's reported repeatedly. The percentile ratings for his height are at every visit.

Secondly, the report which Dr. Mumper alluded to today where the pediatrician used the term some language or some words. I don't remember exactly

what the word was. I would interpret that quite differently than Dr. Mumper.

If a child was developing normally and if you read the rest of Dr. Baker I believe was the pediatrician at the time, they would put within normal limits or okay where it describes it, but some language to me is a doubt about the language production.

And then thirdly, in mom's testimony yesterday she listed the words that he knew at 20 months, which is four months before 24 months or two years, and she listed about seven to nine words -- I wrote them down; I don't remember exactly -- which is way behind what one would expect.

Let me finish.

Q You might have misunderstood my question. I said before his second year of life. What you're just describing is at 20 months and 24 months.

A Do you mean before the beginning of his second year of life?

Q Yes.

A Before the beginning of the second year? Before 12 months?

Q Right.

A I'm sorry. I misunderstood you. I

apologize.

Before 12 months, the only thing that I saw at that point in the record was the falling off of the growth curve.

Q And that is not diagnostic of autism spectrum disorder, is it?

A No, not at all.

Q That could be related to a whole number of issues that have nothing whatsoever to do with autism, correct?

A It could.

Q And nothing about his, as you describe, falloff in growth in that first year, that didn't have anything to do with his language development or his communication skills, did it?

A We don't know.

Q It doesn't have anything to do and there's nothing in the record you can point to showing that it had anything to do with his social reciprocity skills and his interaction with his sibling and his family, does it?

A I wouldn't be so presumptuous as to say that.

Q The falloff in the weight that you describe doesn't have anything to do in describing changes in

his play or his behaviors, his use of toys. It doesn't have any bearing on any of that, does it?

A At what point? You're confusing me --

Q We're still within that first year. We're still within that first year.

A I wouldn't be so presumptuous to say that.

Q Again, I want to be very clear here. Within that first year of life, the only thing that you see as not typical in his overall development was the change in the growth rate between six months and 12 months. Is that correct?

A That was the only thing that was in the record. Yes, sir.

Q And there was nothing in the parents' testimony beyond the medical record that would indicate anything in the first year that was a problem with language skills or communication skills. Isn't that right?

A That's correct.

Q There was no testimony that there were any problems with play or behavior in that first year of life, correct?

A There was no testimony. That's correct. That doesn't mean it wasn't there.

Q You're certainly not saying the parents

didn't testify, that they were silent on issues of play and behavior. You heard their testimony, and you heard them -- I would assume you heard them -- testify about Colin playing with his older sibling, correct?

A Yes, sir.

Q And you recall their testimony about how he behaved at Christmas when he was 13 months old, correct?

A Yes, sir.

Q And everything about that testimony indicates that was a boy who was typically developing and healthy with no deficits. There's nothing in that testimony suggesting otherwise, is there?

A I didn't disagree with that. I didn't say that at all, but I didn't say that that necessarily means everything was moving along smoothly and on track.

Q I think we've canvassed everything that you've relied on in your testimony, and I'm asking you to direct the attention of the Special Master to something in the record, facts in this case, Colin's case, that indicates he didn't develop normally, and you have not done that.

A I can do it. Are you ready? Here's the problem. First of all, these are terrific parents who

have done an amazing job with this kid in spite of all the challenges.

But the problem is they're not experts. They know their child. They know their child well, but sometimes asking -- the reason that doctors take histories and ask questions of patients is to help them recall and understand things.

One of the things that I told you is a failure in this case is to do a proper diagnostic evaluation. The Autism Diagnostic Interview is a structured examination that very carefully and meticulously asks specific questions about developmental events to help parents recall exactly what happened because sometimes it's so subtle and so nuanced if you don't ask exactly the right question you don't get there.

And so the real problem is not what they said. What they said was the complete truth, their recollection of it, and I completely believe them. The problem is they may not have been asked the right questions. The right information may not have been collected.

As a result, we may not know at this point exactly what was happening. Exactly what was happening. Unfortunately, precision is important here

and there's not a lot of precision in what happened early.

Q And again, the question was simple. There is not anything that you see in his medical record and in his testimony that is evidence of a lack of normal development. Isn't that correct?

A No, that is not correct. The correct answer is there was a failure to find the information that could prove that point.

I can't be held accountable for that, and certainly the mother and father aren't responsible for that, but it wasn't done. If it wasn't done, I can't agree with you.

Q You talked about things being subtle, some of the early signs as being subtle.

In looking at the record here, there's nothing that you would identify as a subtle sign except for the absence of an affirmative record of normal development? There's not anything in there affirmatively that is a sign of lack of progress?

A At last, Mr. Powers, we have an agreement, and that is there is a lack of an affirmative record of normal development. The problem is it's not a comprehensive enough record. I agree with you.

But it's the lack of it. It's not that

there's something directly pointing to it in the first year of life.

Q You talked about this comorbid of mental retardation, and you listed some reasons that absent a diagnosis of mental retardation in the record -- actually, I should make it clear.

You would agree that there is no diagnosis in the medical record that you saw that Colin Dwyer is mentally retarded, correct?

A That's correct.

Q You also recall the mother's testimony about having a conversation with an autism specialist who affirmatively represented that Colin did not appear to be mentally retarded. You heard that testimony?

A That's not exactly what she said. What she said was the autism specialist said he couldn't be mentally retarded because he could use PECS.

Now, she certainly should have taken that on face value because she was relying on that person's expertise, but that logic is completely wrong.

Q But there's nothing in the record indicating that this person who was looking at the issue of mental retardation reached the conclusion that Colin was mentally retarded, correct?

A Well, the only problem with that is if

someone arrived at that conclusion and told that to a mom and they didn't know what they were talking about and so they wouldn't have recognized it probably.

Q So you're saying that this person didn't know what they were talking about and misrepresented the facts of Colin's case to Mrs. Dwyer?

A If they said the ability to use PECS was diagnostic of normal intelligence or typical intelligence or ruled out mental retardation, they made a terrible mistake.

Q And there were other things that that professional told Mrs. Dwyer supporting the idea that Colin was not mentally retarded, including that he seemed to understand things well. Do you remember that testimony?

A I remember that testimony.

Q You also remember probably that there was testimony that Colin was a good problem solver. Do you recall that testimony?

A I remember that testimony.

Q So it wasn't just PECS. It was these other issues. Isn't that right?

A That was the mom's testimony for sure, and that was the argument.

However, when you read the record it's quite

clear that his behavior and functioning, including problem solving, including learning, is extremely uneven and highly variable depending on who measured it or who wrote the note at a particular time.

So one month you might say he's speaking and the next month he's not speaking. One month he's doing one particular skill, and the next month he's not doing that skill. The variability in his performance has to be taken into account and it wasn't at least the way it was reported.

I wasn't there. I didn't hear it, but that variability is really typical of autism; that in some instances, in some circumstances, they do reasonably well. In other circumstances they do not so well.

Q Now, you mentioned some of the tests that he did undergo -- the Bayley, the Stanford-Binet and the adaptive testing.

You mentioned some of the limitations of those tests, correct, and you acknowledge that there are limitations on those tests as a measure of whether a child is mentally retarded or not, correct?

A That's correct.

Q And isn't it true that among the limitations of those tests are the degree to which the child will comply with the testing protocol, correct?

A That's a limitation more on the part of the examiner than on the part of the child. People who are really experienced at testing children with autism have ways of helping them cooperate and participate. It's very rare we have children that we can't test.

Q But sometimes children are just not compliant and are not able to be properly tested, correct?

A No. The problem is that as examiners we can't figure out ways to get them to participate. In good hands that's exceedingly rare.

I can't think of a child in the last two or three years we haven't been able to test. Maybe even longer.

Q But that could be one of the limitations of the test, the ability of the child to pay attention, to comply and to sit through the testing?

A No. It's a limitation of the tester, not the child. You can't hold children accountable for our inability to do our jobs.

Q And there's also the limitation that to the extent these tests rely on verbal responses from the children, if a child with autism is nonverbal, and I actually agree that that's an important point from working with the families that I work with. Their

children have a disease.

Would you agree that autism is a medical disease?

A Autism is a syndrome. It's a medical syndrome, yes.

You asked another question, though, about verbal functioning.

Q Right.

A I don't want to let that go because it's important. That's exactly my point about why you have to do appropriate cognitive testing to examine both verbal and nonverbal skills.

There are many children with autism who can do many things nonverbally, sometimes even in the typical range, but test quite profoundly impaired when you do only verbal tests.

It distorts the clinical picture and distorts our understanding of the child's developmental level, so it's really essential to do that and it wasn't done.

Q Right. But I just want to be clear. When you're talking about the limitations on those tests, that is one of them. To the extent they rely on the child's expressive language, that's going to limit their performance on some of these tests?

A It would certainly on the Stanford-Binet and some of the scales on the Bayley, although you could account for that, but it's certainly part of the problem.

Q Now, you mentioned that it might be medically indicated for an autistic child to do a spinal tap, to do a lumbar puncture and draw CSF. Do you recall that testimony on direct?

A Yes. The question that was asked of me, if I believe or if I thought or understand or had information that a child with autism had some inflammatory process going on in the central nervous system what would I do, and I said I would do three things.

I said I would consult with a neurologist because it may or may not be within my area of sophisticated expertise. Secondly, I would consider a lumbar puncture to get direct measures. Thirdly, I would consider some forms of neuroimaging to try to see if there's evidence of inflammation.

Q Now, a lumbar puncture is a pretty invasive procedure, correct?

A You know, we do it pretty routinely. It's probably no more invasive than doing an IV infusion of chelating agent or glutathione.

I mean, we do it quickly and in good hands. You can get in and out very fast and be done with no more stress than you'd do by sticking needles in kids' arms.

Q And of the children with autism that you see, do you have any idea how many times you have performed lumbar punctures or spinal taps on autistic patients?

A Well, since I've never seen a child who has an allegation of or suggestion of a neuroinflammation, there was no need to do it.

I have seen children who had developmental problems in which they either had a previous intracranial infection or have some evidence of some other disorder and we've done LPs, a spinal tap, but generally on children with autism it's very rare.

Q If a child with autism underwent a lumbar puncture I'm assuming that would be done because of this neuroinflammation issue that you described in your direct.

If there was reason to suspect there was neuroinflammation, you would do a lumbar puncture and it would suggest that there is a medical treatment that would be available. Why would you do a lumbar puncture if there wouldn't be a medically indicated

course of care based on the results?

There would be, if you did a lumbar puncture, some type of medical care intervention that would arise from the results potentially. Isn't that right?

A I'm sorry. You confused me. I don't understand what your question is.

Q I'm saying that if you suspected that a child who has autism might have a neuroinflammatory condition such that you would order up a lumbar puncture, ordering up a lumbar puncture for a child potentially who has neuroinflammation, that would suggest there's a course of medical care available to that child based on the lab results, correct?

A So you're creating a hypothetical for me because it's not the case at hand. You're just saying hypothetically if I thought a child had inflammation would I do a lumbar puncture solely for the reason of instituting a treatment?

The answer to that is it would certainly be my hope to find something that would be available, would be amenable to a treatment, but sometimes you find that it's diagnostic and that there may not be a particular treatment available at this time for that particular diagnosis, but it's still incumbent upon

you to go and look to make sure that you understand what's going on.

Q You also talked about some specific genetic disorders that are associated with autistic features in some children. You mentioned Fragile X syndrome. Do you recall that?

A Yes, sir.

Q Fragile X is a chromosomal abnormality. Is that right?

A Right.

Q And one of the features associated with a child who has Fragile X would be some of the features of autistic disorders, correct?

A There are a significant proportion of children with Fragile X who also have autism, and some have just some of the symptoms of autism and some have none of the symptoms of autism.

Q And typically a child who has Fragile X, as the particulars of that child got older would have sort of a coarsening of their features and for boys an enlargement of their testicles. Isn't that correct?

A Some do, but some don't have any of the dysmorphic features. That's part of the problem.

It can even occur in girls, which is a little bit surprising. They couldn't have enlarged

testes, obviously.

Q Right.

A So the answer is that the physical phenotype -- the enlarged ears, et cetera -- are not always present in every patient, which is why you would do genetic testing to make sure that you didn't miss it.

Q And they're certainly not present in Colin Dwyer's case at least based on your review of the medical records, correct?

A Based on my review of the medical records I didn't see any notice, but then no one raised the question of doing genetic testing or whether he should have Fragile X to be ruled out.

Q And tuberous sclerosis? One of the symptoms of tuberous sclerosis, in addition to some of the features of autism, is seizure disorder, correct?

A Sometimes, yes.

Q Sometimes. You don't see any evidence of a seizure disorder in Colin Dwyer's medical history or in the testimony of the parents, do you?

A No, but that doesn't mean he doesn't have tuberous sclerosis.

Q Right. And in 15q, sometimes seizures are associated with the 15q duplication error, correct?

A Sometimes, but often times not.

Q Often times not, but you don't see them in Colin Dwyer's case?

A We don't see seizures, but he has autism and autism could be caused by 15q duplication so you have to rule that out.

Q Now, what you would describe as genetic contributors to the appearance of autism in children.

Do you recognize that there are some instances where a genotype may exist that would be asymptomatic absent an environmental exposure or environmental trigger that would result in the appearance of the symptoms of autism?

A Certainly that's possible.

Q In your work in looking at possible genetic markers so to speak for autism, have you come across the idea that glutamate in the brain is an issue of some interest for autism?

A Well, glutamate is of great interest because it's the most pervasive neurotransmitter in the brain. It's everywhere, and it's a regulatory neurotransmitter that we don't really understand a great deal about both in pathology and in health.

So, yes, it's a matter of considerable interest, and there's some evidence to suggest that perhaps some specific glutamate receptors may play a

role in the genesis of autism and that there are genetic substrates for that possible abnormality. That's one of the things that we're exploring now.

Q And certainly an excess of glutamate in a brain, particularly in the brain of a developing child, an excess of glutamate can lead to an over excitation of the brain and affect brain function, correct?

MS. RICCIARDELLA: Objection. Special Master, again this is way beyond the scope of direct or anything that is in Dr. Leventhal's report. This is getting into general causation, something that this Court the evidence has said is closed.

THE COURT: I'm going to go ahead and hear it. If I decide to disregard it, I'll disregard it.

Go ahead, Mr. Powers.

MR. POWERS: Yes.

BY MR. POWERS:

Q Dr. Leventhal, in 2007 there was a paper published called Mapping Autism Risk Loci Using Genetic Linkage and Chromosomal Rearrangements. Do you recall that paper?

A I don't recall the title. Who's the author?

Q You're one of the authors. There's an extraordinarily long list of authors.

We're putting this up. I think the list was so long that rather than putting it at the front they put it in the back, so if we could roll through there and take a look at that? Literally it goes onto two pages.

A That's correct.

Q Oh, I know that I saw your name in there.

A I'm in there.

MR. POWERS: Yes. That's what I thought. Okay. I want to draw your attention to page 325 of the article, and I actually have a copy that I can give you and to the Special Master and to Respondent.

THE COURT: And we'll call this Petitioners' Trial Exhibit 21.

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

THE WITNESS: I'm sorry. What page number?

MR. POWERS: Page 325.

THE WITNESS: Yes, sir.

BY MR. POWERS:

Q If you look at page 325, in the last full paragraph on the bottom left side you'll see a discussion of glutamate, and about halfway down there

there's a sentence that begins: Moreover, aberrant glutamate function is often cited as an important element of risk for ASD. Do you see what I'm referring to?

A Yes.

Q Would you agree with the statement in this paper that aberrant glutamate function may be an element of risk for the development of autistic spectrum disorders?

A Well, what this says is it's often cited, but we don't know what the actual role of glutamate is.

Actually, as it's turning out it may not be glutamate itself, but may be one of the glutamate receptors, which plays a role in other elements of neurodevelopment and neurotransmission. There's a receptor called mGluR-4 which plays a more critical role here.

And so glutamate may be leading us in that direction, but it may not be glutamate itself that's the causal moiety, but it may be the disruption in the way the particular receptor develops and then the other neuroregulatory mechanisms that follow from that.

Q And among the neuroregulatory mechanisms

involved in regulating glutamate in the brain would be astrocytes, correct?

A Well, I suppose simply put, but glutamate also plays a role in regulating astrocytes so it goes up and back.

Q Right. There's somewhat of a reciprocal relationship between the astrocytes absorbing excess glutamate, correct?

A Yes. I mean, yes, that's close enough.

Q Okay. But the bottom line is that you do agree with the statement here that aberrant glutamate function, without particular detail, is an important element of risk in autism spectrum disorders? You would agree with that statement?

A The statement doesn't say what you just said. It says that it has been cited, and the references are provided for that. And so it doesn't necessarily mean that glutamate itself is playing a role. Just there are data that have been suggestive that it might be glutamate.

This was published in 2007, written in 2006. This is 2008. The world has changed, and there are actually new data suggesting it may not be glutamate itself, but we don't know.

Q At the very least, glutamate is of

continuing interest. I'm not saying it causes anything. You would agree it's of continuing interest --

A Sure.

Q -- in the brain of people with autism?

A Sure.

MR. POWERS: Okay. I have no further questions.

THE COURT: Redirect?

MS. RICCIARDELLA: Yes, ma'am.

REDIRECT EXAMINATION

BY MS. RICCIARDELLA:

Q Dr. Leventhal, at the start of Mr. Powers' cross he asked you what you reviewed and relied on for your opinions in this case, and he said that he canvassed the amount of information that you relied on.

In addition to the medical records and reading the expert report of Dr. Mumper and listening to the parents, did you also rely on your 30 plus years of experience as a child psychiatrist in rendering your opinions in this case?

A Yes, ma'am.

Q You were also asked if in your opinion there are any environmental contributions to autism, and you

said perhaps. In your opinion, are thimerosal-containing vaccines one such environmental contribution to autism?

A As hard as we've looked, we see no evidence to support that notion.

Q You also were asked about lumbar punctures. You're not saying that all children with autism should receive a lumbar puncture, are you, Doctor?

A No. Quite the contrary. Very rarely should they get a lumbar puncture, but if there's indication of central nervous system disease that can be diagnosed by a lumbar puncture they should get one.

MS. RICCIARDELLA: I have no further questions. Thank you.

MR. POWERS: No further questions based on that.

THE COURT: Dr. Leventhal, I just have one question for you, and it has to do with the exchange between you and Mr. Powers. I felt like I was watching a tennis match at some point there.

THE WITNESS: No love, though.

(Laughter.)

THE COURT: Well, that's part of a law firm here that has a little Love in it, but I want to make sure I understand what it was you were saying.

The way I do this is I restate what I think you were saying, and you tell me whether I'm right or wrong, okay?

THE WITNESS: Yes, ma'am.

THE COURT: What I heard you to say is that you don't have confidence in the adequacy of the record that exists here to establish the premise that this child developed normally and then regressed.

THE WITNESS: That's correct. You know, it's interesting. I mean, it's not surprising. I mean, it's not that anybody was bad or did the wrong thing.

I think in 2000-2001 it was a time when the prevalence of autism was starting to rise, and it was quite clear that pediatricians in particular who were the people at the front line who would get the first calls, see the kids first, weren't adequately trained to see the nuances to make the diagnoses early.

In fact, there have been massive efforts on the part of the American Academy of Pediatrics -- I've actually worked with them on that primarily at the Illinois level -- to go into pediatricians' offices and begin to train them so they can start to pick up these key developmental indicators.

The answer is this record is inadequate

because a pediatrician might not have thought in 2000 or 2001 to even ask the kinds of questions that they would ask today or we ask today.

THE COURT: Okay. Questions from the other side based on mine? Let me just ask Ms. Ricciardella first. She gets to go first. Anything?

MS. RICCIARDELLA: No, ma'am.

THE COURT: Okay. Then it's all yours, Mr. Powers.

RE-CROSS-EXAMINATION

BY MR. POWERS:

Q You just used the term that the prevalence of autism was rising around 2000 and 2001. You believe the prevalence of autism was rising back then?

A I think it's been rising for longer than that.

Q Do you believe that the incidence of autism within the population is rising?

A So far I haven't seen evidence that suggests that it's rising. However, the definitive study hasn't been done, and I'm doing that now and I'll be able to answer that question for you in about four or five years.

Q We will wait with baited breath, and if you could maybe convince the Special Masters to wait we'll

have some very interesting information, but it is an open enough question that you're involved in a study to look precisely at whether incidence is rising along with the prevalence?

A Well, I wouldn't actually say that. I'd say that the best we can tell right now there aren't strong indicators of an increase in incidence, but we can't answer that definitively so I think we're reasonably comfortable saying if there's an increase in incidence it's not gigantic; that much of the change in prevalence is accounted for by many other reasons.

But it's incumbent upon us as scientists to go and say okay, let's nail this one down. It's a very complicated study to do, very expensive. It's going to take us five or six years to get done. We've started, and hopefully we'll have data in four years that we'll be able to tell you.

I would certainly hope that the Special Masters don't wait for us because I think these families need an answer and they need to be able to move on with their lives.

MR. POWERS: No other questions, Special Master. Thank you.

THE COURT: Thank you, Dr. Leventhal. You

may step down.

THE WITNESS: Thank you.

THE COURT: And safe travels.

(Witness excused.)

THE COURT: Ms. Ricciardella, does the Respondent have anything further to offer?

MS. RICCIARDELLA: We do not, ma'am.

THE COURT: Okay. Mr. Powers, how about Petitioners? Mr. Ferrell?

MR. POWERS: Special Master, we have no additional witnesses to present. I think all we have at this point is closing, so if we perhaps took a brief, say 15 minute, break we'll be ready to close. Will that work?

THE COURT: Government, is that adequate for you?

MS. RICCIARDELLA: That's fine.

THE COURT: Okay. We'll reconvene at 25 to.

(Whereupon, a short recess was taken.)

THE COURT: We're back on the record then in the case of Dwyer v. Secretary, HHS.

Mr. Williams, you're going to close for us?

MR. WILLIAMS: I'm going to do the closing on general causation, and Mr. Powers will address the case-specific issues here today.

First, I want to thank you and Special Master Hastings and Special Master Campbell-Smith for the attention you've given this case. I think this is the most important case any of us have ever worked on if you think of the public health implications of your decision on general causation here.

There are still millions of kids getting thimerosal in vaccines around the world, and what you decide is going to be very important in what happens in the future not just in this country, but to those kids.

I think that we have established that there is a biologically plausible mechanism of how thimerosal-containing vaccines can cause regressive autism in some children. First of all, neuroinflammation can lead to regressive autism. I'm going to show you later diagrams from -- that show this is a generally accepted fact among the leading scientists doing research on the subject.

We know from the adult monkey studies that inorganic mercury can cause neuroinflammation. There's no question about that. There may be a question about how much it takes, but there's no question that inorganic mercury in the brain can cause neuroinflammation. That means there's no question

that inorganic mercury can cause neuroinflammation and autism.

We know that thimerosal-containing vaccines deliver inorganic mercury to the brain. We know that from the Burbacher infant monkey model. We also know from all the studies on humans and primates that there's wide variability in the blood and brain levels of mercury after exposure and therefore there must be some infants at the far end of what Dr. Brent admitted as a bell curve of susceptibility and the ability to handle autism -- the ability to handle mercury that some children are going to have very high exposures.

Next slide, please? Now, the two world leading experts on mercury toxicity were going to come and talk to us and give us their information on this important public health matter. For reasons we don't know, they're not here, but had they come, this -- we would have been able to use their own writings to show these facts.

MR. MATANOSKI: Your Honor, I very rarely object during argument, but I do think that this is actually beyond what the scope of argument should be in a specific causation case.

I understand that we were going to hear some general causation, but a slide entitled What Magos and

Clarkson Would Have Confirmed? If they wanted to put on evidence on that the time to do that was --

THE COURT: Well, I think we have some of those items in evidence.

MR. WILLIAMS: All of these are in evidence, Your Honor.

THE COURT: You didn't list -- again, the Burbacher infant monkey study is in evidence. You didn't list the exhibit number.

I'm going to permit Mr. Williams to argue what he wants to argue.

MR. MATANOSKI: Very well.

THE COURT: I would take exception that we don't know why they're not here. I think it's pretty clear from our status conferences that we understand why they are not here.

MR. MATANOSKI: Thank you, ma'am.

THE COURT: Go ahead, Mr. Williams.

MR. WILLIAMS: We did -- also just to respond to the objection, we specifically reserved closing on general causation until today at the last hearing.

Clarkson is a co-author of the Burbacher paper, and in that paper there is a statement that the microglial reaction to the inorganic mercury in the

brains of those adult monkeys -- we're talking about the adult monkey studies within the infant monkey paper, and they say it is not protective mechanism. It is a toxic mechanism. That's Clarkson who says that.

We also know, and this is also out of the Burbacher paper, but it's on one of Magos' own review papers in his CV, that the human brain to blood ratio is six whereas in the monkeys, in those infant monkeys, it's only 2.6, which means that in a human infant 2.3 more times -- two and a half times -- more mercury will be deposited in the brain, given the same dose into the arm. That's established in Magos' own writings, and it's established in the Burbacher paper. No evidence to the contrary.

We also -- it also says in the Burbacher paper that the infant macaques had blood levels that were comparable to the human infant levels in the three studies we have, two by Pichichero and one by Stajich, of human infants who got thimerosal-containing vaccines and then had their blood levels measured. So it is reasonable to conclude that brain levels of inorganic mercury in some human infants are in the same range that ignited neuroinflammation in those adult monkeys.

I think we've established that thimerosal-containing vaccines belong on the list of potential environmental triggers of ASD, specifically regressive autism. We already know from the literature and from the testimony that there are several well-recognized environmental triggers -- thalidomide, valproic acid, terbutaline.

And although Dr. Leventhal didn't know it terbutaline has actually been studied in a mechanistic way in that rat study that we gave you -- Zerrate is the first author -- again by the same group at Johns Hopkins that established it is a neuroinflammatory mechanism that seems to be how terbutaline causes autism.

We know that certain viruses can cause autism again through neuroinflammation, and we know inorganic mercury belongs on the list because we know it causes neuroinflammation in monkeys.

Now epidemiology for a minute. The existing epidemiological studies are uninformative on the question of regressive autism. Everybody agreed with that. There is not one study that has actually tried to isolate regressive autism and compare it to kids that have taken thimerosal-containing vaccines. There's not one study on that subject.

Not one study has ruled out an association between thimerosal-containing vaccines and regressive autism, and both Goodman -- Goodman agreed with Greenland on that and said he's technically correct that the numbers would allow regressive autism to be associated with thimerosal-containing vaccines even within the studies we have, so the existing epidemiology does not rule it out.

Now Fombonne. He contradicts himself. I think we showed that during the cross. First of all, he attacks all the studies that purport to show an increase in the rate of autism over time on the grounds that they inadequately detected it and that the real rate, we don't know what it is, but we know it's always much higher than what the old studies detected.

Then he turns around and cites studies that purport to show an increase after removal of the vaccines and says that's evidence that the vaccines didn't cause autism. He can't have it both ways. He's saying any study that finds an increase is unreliable, and then he turns around and says these studies that show an increase after removal of vaccines are reliable. It just doesn't make sense.

There is epidemiology in favor of causation.

Respondent has not refuted the Young VSD study, and I know you in particular, Special Master Vowell, distrust the Geiers, but the Geiers only provided access to that data. It was Dr. Young who did the analysis. She's a full-fledged epidemiologist here at George Washington.

She did the analysis. Her letter clearly states that it could be duplicated easily. Just run the program again. The government has it. If her analysis was wrong, we would have heard about that by now. We haven't.

Fombonne tried to critique it, but he clearly didn't understand it. On the chart that he made and showed you, he confused the two lines. He confused the one that was charting mercury exposure with the line that was the increase or change in the rate of the ASD diagnoses, and then he also claimed that the study looked at linear correlations when it actually used nonlinear rate ratios.

That's all explained in Dr. Young's May 30 letter to the Court, which has not been rebutted or refuted by HHS. Again, if her analysis was wrong HHS could easily rerun that program and prove it. That didn't happen, so I think that the only epidemiology we've got is in our favor.

Now, the standard of proof in the program, as I understand it, does not require Petitioner to have epidemiology to support causation, but if you Special Masters decide that under the facts of this case that's the one thing we're lacking -- that we needed to have an epidemiological study that links thimerosal-containing vaccines to autism -- I think you've got to presume that those two VSD studies would come out in our favor.

These are the studies that we tried to get access to do and were denied. These are the studies that a special panel of experts convened by NIH said should be done, and these are studies that Dr. Goodman -- not only the defense epidemiologist in the case, but the epidemiologist on the IOM Safety Committee, the Vaccine Safety Committee. He told us on the stand in this trial he thought they should be done.

This Administration won't do them. This Administration currently running HHS seems to want to decide this case without that data. I think you've gotta -- we're going to file a formal motion on this later, but we think we're entitled to a presumption, that these kids are entitled to a presumption that those studies would come out in support of causation.

Or, there's still time. We think you've got

the authority to issue an order authorizing the vaccine trust fund money to be spent to do those studies. Now, if you find that we must have epidemiological evidence then you also ought to wait for the results of Respondent's two autism thimerosal-containing vaccine studies that are underway right now.

We're very suspicious that the government wants to rush this case through a causation decision when it's got -- the two most expensive studies it's doing on the question are going to be published later this year, not in time for you to have them. Julie Gerberding, who is the NIH director today, said in a report to Congress -- it's one of our exhibits -- that both of these studies are finished and will be out in September or so of this year.

I think you got to at least wait until we have those studies because they're specifically on autism and thimerosal-containing vaccines. One of them is a case control study within the VSD, and one of them is this fortunate Italian randomized trial where a bunch of kids got different doses of thimerosal and then they've gone back and examined them to see if there's any differences.

We don't know what the results are. I

presume HHS does because at least one of these manuscripts is done, but they haven't offered that evidence here.

If you decide that we have to have a primate brain study that shows TCVs ignite neuroinflammation in the infant monkeys, we've got uncontradicted evidence that inorganic mercury causes neuroinflammation in adult monkeys. We have uncontroverted evidence that TCVs deliver inorganic mercury to the brain of infant monkeys, but we don't have the brain pathology work yet from that Burbacher study. That's still coming. They're working on it. And again, this is a study funded by Respondent.

And, if you're -- now, you may agree with us that we've already put on enough evidence and you can decide in our favor on general causation, but if you think we need this evidence you should wait for it. It's coming. It's partially within the control of the Respondent as to when it gets delivered to you.

So let me summarize again our biological plausibility argument. We know that the vaccines deliver inorganic mercury to the brain. We know the wide individual variability. We know that inorganic mercury persists in the brain for years.

Some human infants will have inorganic

mercury levels comparable to those that ignited the neuroinflammation in those adult monkeys. Neuroinflammation has been found in almost all the brains of human autistics when it's looked for, and persistent neuroinflammation can explain the symptoms.

The Pardo group has two diagrams, one of which we showed Dr. Leventhal. This is one we didn't show. This is from a second article by the Pardo group reviewing this. I wanted to pull this one up because it specifically shows that both infections, such as measles virus, and toxins, such as inorganic mercury or terbutaline, can affect postnatal brain development and brain maturation.

It can explain all these neurobiological trajectories here and eventually lead to the result of autism spectrum disorders. This is a generally accepted model of how autism can be caused by both viruses and by toxins such as inorganic mercury.

And then finally, the last diagram, the one we showed Dr. Leventhal. This specifically talks about the neuroglial activation being at the center of all of this leading eventually to the regressive phenotype of autism.

And I think under the standards of the program we've proven that these vaccines, by

delivering that inorganic mercury to the brain, in some kids can cause neuroinflammation that can cause autism.

THE COURT: Mr. Powers?

MR. POWERS: Thank you, Special Master, and thank you to the other Special Masters.

I echo Mr. Williams' sentiments in understanding the time and the energy and the effort that's gone into preparing and presenting and listening to these cases and also from Respondent's side the effort that it's taken to get these cases to hearing in the first place, to develop the evidence and present the hearings.

I also do want to thank the families, the people who have been here. Not just the folks who volunteered to be the test cases, but those families in the program that really have given Mr. Williams and myself and other members of the PSC the honor and the privilege of representing them here and the huge amount of trust that they've placed on us and they've placed on the experts that have been presenting evidence here, so I want to acknowledge them and thank them and let them know what a privilege it is to be here on their behalf.

I am going to talk about, as Mr. Williams

said, individual causation here, and I'll be brief. If this was a civil trial, Special Master, I believe that -- and we were the Plaintiffs rather than the Petitioners, would be entitled to a directed verdict.

What you heard from Dr. Leventhal was wild speculation about things that were not in Colin Dwyer's medical records and opinions about his view of causation that are completely unsupported by the record. Let's first talk about the issue in Colin's case, as Dr. Leventhal said in his report, that he believes Colin Dwyer's autism is likely caused by a mix of genetic abnormalities.

Now, he says that there's no genetic testing that's available in the record to confirm that or refute it, but I would explain to you, Special Master, that even if the genetic testing had been done his own testimony was just a tiny minority of cases of autism have any sort of genetic identifiable abnormality associated with them.

And so in the absence of any genetic testing, and you've heard the testimony from experts in the other test cases that only about 10, perhaps 12, percent of cases of autism have known genetic causes, so all we can assume at most, even if he is right, that if that genetic testing had been done

there's only about a 10 to 12 percent chance that Colin would have been -- would have been seen to have an identifiable genetic cause.

So it's wild speculation at two levels. First, assuming, as he seems to do, that if the testing had been done it might have showed something, but also assuming that in the absence of that testing, which is what we have here, that it's more likely than not that it was genetics. No -- not a scintilla of evidence in support of that notion that he expressed in his report that this condition is caused by a genetic abnormality.

Secondly, there is no evidence in the record that he was able to point to that showed evidence of early problems. And he went on at length about it's so subtle it might be missed. Well, sometimes it's so subtle that it is -- doesn't exist. It doesn't exist. And to -- again, wild speculation unsupported by the record, explicitly contradicted by the parents' testimony.

This was a normally developing boy, a completely normal progress of development with nothing in the record indicating anything related to autism had gone wrong before that first note in the chart at about 20 months of age when he had a language delay

that was identified.

And there was some debate about that 15 month chart note. You can debate that back and forth, but before 20 months that literally is the only shred of evidence, and that tiny shred of evidence would not support a verdict again if this was a civil case. Everything else that he said on the issue was complete speculation and explicitly, explicitly contradicted by the record and by the parents' testimony.

Dr. Leventhal also talked about his understanding or belief -- he would not want to use the word belief, but his understanding -- that there is no such thing as a regressive phenotype.

Well, Special Master Vowell, you have heard in other of these cases and in the general causation testimony that peer reviewed, published medical journals recognize if not a diagnostic phenotype a symptomatic phenotype of regression.

You recall that Dr. Rust, when he testified in the King and Mead cases, said that even aggressively retrospectively analyzing medical records, interviewing parents, looking at videos, he sort of ran up against the wall. There are always about 20 percent of children with autism that as hard as he looked retrospectively appeared to have

perfectly normal progress.

And we offer and the evidence supports, particularly the Dwyers' testimony, that Colin fits squarely in that 20 percent. And Dr. Leventhal's testimony to the contrary, again completely speculative and without a basis in the evidence or the record, and it's based on assumptions about a percentage in his patient population that clearly doesn't include Colin in this instance.

Dr. Leventhal's testimony is a classic example of what I think it was Dr. Rust described with Tycho Brahe where you're so fixed on an idea that you interpret evidence with such a strong bias towards what you think the ultimate answer is that all the evidence looks like it answers that question the way you want to answer it, sort of looking through the telescope and even through the wrong end of the telescope.

And as I said in the closing in the Mead and King cases, Dr. Rust was so fixed on the idea that autism looks like Rett's, Rett's is genetic, that therefore all autisms must be genetic, that his fixation on that Rett's syndrome idea blinded him to the possibilities in the peer-reviewed scientific literature that suggested that there are environmental

factors that may play a role, environmental factors that have been identified as playing a role.

And you see that same sort of approach here with Dr. Leventhal where even with the widely recognized, as Mr. Williams described, prenatal and some postnatal exposures that contribute to the appearance of autistic symptoms, Dr. Leventhal stubbornly refused to entertain the notion except in the most narrow hypothetical manner that environmental exposures interacting with genetic predispositions could result in the appearance of autism, particularly of regressive autism.

And so you have yet another expert from the Respondent's side so fixed on a rigid idea that it's all genetic that they miss evidence that would support an alternative theory of causation.

It's also important to mention if you're looking at this genetics issue really for 10 years now if you go back through the literature there are indications that we're discovering new genetic abnormalities, and as we get more sophisticated and do better scans we're going to find more and more. Dr. Leventhal testified about that today. We're finding new anomalies all the time.

Well, these folks who are looking for those

anomalies have been doing it for at least a decade, studies all over the country, and as you heard it's still just a tiny, tiny percentage of autistic disorders are associated with an identifiable genetic contributing cause.

There's something else out there, and these doctors, particularly Dr. Leventhal in this case, ought to be open to the idea that there is something else out there, and they should be open to that idea because it's supported by the literature.

So because Dr. Leventhal's testimony consisted almost entirely of assumptions, a priori conclusions, speculation contradicted by the record, contradicted by the parents' testimony, his testimony on causation in this specific case ought to be given very, very little weight, and you ought to decide, Special Master Vowell, that Colin Dwyer is entitled to compensation.

And finally, in integrating the theory of general causation that Mr. Williams described and how you might approach applying that general theory to this case, one way to approach it, I suggest, is that you could look at the evidence right now points to, and the testimony, and everything that's come in in these hearings points to sort of three possible models

in explaining the etiology of autism and particularly the etiology of Colin Dwyer's autism.

The first is that nothing caused it. Well, scientifically it's hard to accept and probably impossible to accept the idea that autism or any other medical condition is literally caused by nothing, but that is intellectually at least or logically at least a possibility that his autism and the autism of these other children has no cause, but we can rule that out because science won't accept that as a plausible conclusion.

You're then left with two other possibilities. One is the general causation possibility that Mr. Williams described and is supported by the evidence in these cases that thimerosal-containing vaccines in a child such as Colin Dwyer can contribute and they're on the list in the differential diagnosis and can be a substantial contributing cause of the autistic symptoms, particularly the regressive autism presentation that we see in Colin's case. That's a conclusion that's supported by both general causation evidence and case-specific evidence.

There is the alternative theory, if you will, that seems to be suggested by Dr. Leventhal,

which is that it was caused by something, but we don't know what that something is. And as you heard him testify in this case, the only thing that he could describe that would have contributed to Colin Dwyer's autistic regression was some genetic component, but you also heard him testify that that identifiable genetic component is only present in 10 to 12 percent of cases of autism.

So his theory of causation in Colin's case is 10 to 12 percent likely to be true, but 88 to 90 percent likely to be untrue and so under the standards of the program there is no alternative theory that's viable supported by the evidence presented here by the Respondent.

So on the one hand you have one of the competing theories ruled out legally by the standard in the vaccine program; another theory, which is that nothing caused it, ruled out as a matter of scientific principle, and that leaves you with an evidence-based model and mechanism of causation in this case that associates thimerosal-containing vaccines with Colin Dwyer's symptoms and his autistic regression.

It's supported by the general causation testimony and evidence, by the case-specific testimony and evidence, and it's that record that supports an

award of compensation in this case for Colin Dwyer.

THE COURT: Thank you, Mr. Powers.

Mr. Matanoski, are you closing for Respondent?

MR. MATANOSKI: Yes, ma'am. First I'd just like to thank the Dwyers -- I see Mrs. Dwyer is still in the courtroom -- for allowing their case to go forward to help the Court decide this important issue, to be the third test case.

I'm sure it wasn't an easy time, but probably the couple of days in the courtroom don't compare with what dealing with children with autism is all about, and I'm sure that that's shared by families everywhere, those challenges, and in fact also no doubt some joys that are associated with that too.

I am giving the closing. I drew the short straw. Although by the time we get up here it must seem like we really enjoy ourselves doing this, we really don't. I'm sure the Court appreciates when we're brief. I had hoped to be a little briefer than I'll have to be, but I still hope to make it fairly short.

First, I want to point out on the specific causation lawyers are kind of slick. They move things around and kind of play a shell game. When I heard

the comments about the specific causation case it made it sound like Respondent has the burden here to show what actually caused it. Actually, the burden is on the Petitioners to show the vaccine caused autism. And Respondent doesn't have to show that it's genetic in origin.

I think that the comments about Dr. Leventhal's testimony on that point are a little off the mark. What Dr. Leventhal was saying is essentially most practitioners, most folks who study autism as a profession, believe that it's largely genetic in nature, and that's where the research has been directed, and in fact it's been fruitful in that regard. There's still much more to do, but everything that has come out has pointed to genetics as very strongly associated with autism.

And most of the research that's been done has shown that autism would have a prenatal course, that it can essentially be seen, that the preconditions, if you will, for autism are in place beginning before birth in most instances.

I think there is also a little bit of a misconception about what the force of Dr. Leventhal's testimony was. He basically was saying that Colin's case really is sadly no different than many of the

cases that he sees where there's a gradually emerging picture of difference, perhaps delays, but at least difference in the quality of behavior in a child as the child develops.

It's not necessarily apparent right from the start. That's very rare. In most of the cases it's apparent later, and it may seem that a child who has just made -- reached certain milestones, has subsequently had trouble keeping those milestones, as the condition progresses there often is an improvement. That's the natural course of the condition.

And what Dr. Leventhal was saying is as time has gone on more and more of the researchers have realized that if you look back in the cases that apparently seem to have a normal trajectory and then there seems to be a loss that you see earlier signs and symptoms that all was not on a normal trajectory from the beginning.

That was the force of his testimony, and that testimony was backed up by other testimony this Court has heard before he took the stand. Dr. Lord, who specifically studied regressive autism, made that point quite clear that as this has progressed the concept of regressive autism has become more

encompassing, that autism itself seems to have a progression where it appears that there's a loss, but when one goes back one sees that there's unusual or differences in development earlier on in almost every case.

What Dr. Leventhal was saying is as they've gotten better, the folks who do this for a living, the folks who make their lives about studying autism, they have realized that more and more of those cases they can see the differences earlier on and that in very few instances when they've studied quite closely do they see that there isn't some sign that the trajectory or the course is not the same as other children.

Dr. Mumper's testimony, which really wasn't much of a focus during the closing argument here, she seems to be relying on isolated lab results to come up to the conclusion that vaccines are the cause here. She's been asked in this case and in other cases what would that pattern be? What do we need to look at?

And in fact, there doesn't seem to be a particular pattern. In the King case certain test results were relied upon to draw the conclusion that vaccines or thimerosal in vaccines were associated with autism in that case or a cause of autism in that case.

In the Mead case, other results were looked at and thought to be by Dr. Mumper indicative that vaccines were causing or evidence that vaccines were causing autism, and now in Colin's case we see yet a different pattern of test results being relied upon to reach that conclusion. In fact, those test results, with really no pattern, how can one say that there is any kind of clinical evidence from these test results that one can rely on to make that kind of -- to draw those kind of conclusions that Dr. Mumper is relying on?

And as you'll see when you go through the testimony, we believe that she largely moved away from relying on any specific test result when questioned about each specific one. She said that essentially the mercury test result, the positive provocation, was really the only test that she had that showed that mercury was there and that she was relying on to implicate thimerosal as a cause in this case. But then she admitted that she really didn't know what the normal range would be for that test. How can one say that this is an abnormal result when one doesn't know what normal is?

Her testimony seems to be formed largely by the Defeat Autism Now world view, which is that toxins

and heavy metals are implicated in autism, and to use the example that Mr. Powers used of Tycho Brahe, I think that comes to bear with her testimony as well.

It doesn't matter which test results she's looking at. It always comes back to a heavy metal or a toxin when it could be that the acidosis, that the lactic acid buildup, was because the child was crying, for example, when the test was taken.

The scenario that we see played out with Colin, his course, is sadly played out too often amongst children with autism. It is played out not only in this country, but in other countries. It's played out across the globe in fact, this gradually emerging picture of a child who seems to be slipping into autism, seems to have had some positive or normal development, but then gradually having more trouble speaking, gradually -- a gradual awareness, if you will, that the child is not developing in the same way as one would expect.

This doesn't seem to be influenced by the presence of vaccination or not. As I said, it's repeated around the globe whether the children are vaccinated or not. It doesn't seem to be influenced by whether thimerosal is in those vaccines or not.

As you've already seen in some of the

studies that have come out, vaccines being in, or thimerosal being in the vaccines or not being in the vaccines isn't changing the prevalence of autism. It continues to rise. It doesn't seem to have an impact on it.

It doesn't change. Vaccination in the child who has autism doesn't change the clinical presentation of the case at all. It's the same clinical presentation whether the child is vaccinated or not and whether that vaccine that the child received had thimerosal or not. It simply is not having an impact at all. Again, this is unfortunate. It's played out far too often, but it is not being influenced. The condition is not being influenced by vaccination.

I want to touch now on the general causation because that was a matter of some discussion by Mr. Williams. I see that the glutathione theory, which is where we started with this general causation case, seems to have dropped out. It wasn't in the opening statement. It wasn't in the closing statement. It seems that the theory of causation now is neuroinflammation and largely seems to be neuroinflammation alone.

That was a theory that Dr. Kinsbourne

recently advanced in this case that obviously wasn't present until just a couple of weeks before the trial in May. This is something that after six years in the making this seems to have come up kind of at the very end.

Mr. Powers and Mr. Williams have focused on the causation burden and say that the information they've given on neuroinflammation meets that causation burden. That would be their focus is on the causation burden under Althen and Grant, the specific criteria that they need to meet under that test that the Court has articulated, the Federal Circuit has articulated.

Respondent starts a little earlier than that, if you will, in the calculation, and that is about what evidence feeds into Althen and Grant. We start out with an analysis under Daubert about whether there's good scientific evidence to even meet that burden.

So obviously if the evidence that you have or the evidence that's being offered does not meet the criteria of good scientific or reliable evidence then you'll have nothing at all to test about whether you've met your legal burden under Althen. Our position has been throughout this that the

Petitioners' evidence that they've offered, the testimony they've offered, fails to meet that standard of reliability that is set out under Daubert and that this Court applies.

Daubert stands for the proposition that there are not multiple kinds of scientific evidence, a kind for scientists to use and a kind for Judges to use. There's only one kind of scientific evidence. It is the kind that scientists use. That's the kind that Judges are supposed to be looking for as well.

When scientists or when witnesses come and present something that is not -- does not meet the standards that general scientists would use then the Court must reject that testimony, cannot use it. It cannot feed into a legal standard of causation. That's the problem Petitioners face right now. The testimony they've offered doesn't meet the Daubert standard.

Now, the Court has permitted PSC six years to gather evidence and to find competent experts. They've permitted the formation of a large group of attorneys with essentially the resources of hundreds of other attorneys at least theoretically to help them advance their case. And they've permitted discovery. In short, they've permitted a -- a fairly, they've

given ample -- the Court has given ample opportunity to develop a causation case here.

What you ended up with, however, was speculation rather than good scientific evidence, and it was speculation that really came in, speculation that was kind of last minute, if you will, in the way it came up in terms of the neuroinflammation case.

I'm going to just touch briefly on some of the things that Mr. Williams discussed, some of the specific pieces of evidence. He discussed the Burbacher study and the adult monkey studies that were extensively discussed in the May trial.

If you kind of distill that down to its kernel, what do the adult monkey studies tell us? That mercury given to these monkeys in amounts way exceeding those in childhood vaccines produced glial activation to some extent and no clinical symptoms.

If you look at the Burbacher study, the Burbacher study essentially told us that ethyl mercury leaves the body quickly, but some is converted to inorganic mercury. The amounts involved in that study were far below those in the adult monkey studies.

But let's assume for a moment that after these autopsies are done Dr. Burbacher, who, by the way, is not conducting studies at the United States

Government's behest. I'm not sure at whose behest he's conducting the studies that were mentioned by Mr. Williams, but it's not at the United States Government's behest.

Let's assume for a moment that these -- that he conducts his study and comes back and it shows some sort of glial activation similar to what was seen in the earlier adult monkey studies. Well, we know in those studies at much higher levels of inorganic mercury and seeing glial activation there was no clinical signs.

What should we expect in terms of these studies then, these autopsy results from the Burbacher study? There would be no clinical signs. Well, certainly autism has clinical signs because the families deal with that all the time.

They also relied on the studies by D.B. Pardo, and we heard of the -- the Lopez-Hurtado studies. The interesting thing about those is it wasn't a small group of patients of clearly regressive autistic children that we've heard premised as the phenotype for vaccine-related autism. It was these findings of markers, of neuroinflammation, in these individuals was found across the board. It was not a specific phenotype. It was everyone.

Now, certainly the Petitioners are not coming in here and saying that the 50-year-old people in Lopez-Hurtado, that every, every case of autism is caused by thimerosal in vaccines. This was a nonspecific finding that the Pardo group and Lopez-Hurtado had seen in the autopsy studies. It was everyone.

Dr. Pardo provided a letter trying to explain some of these results because of this sort of late development of the neuroinflammation theory, and, as you know, he was ready to testify if the Petitioners desired.

Interestingly, although neuroinflammation is seeming to be the sole focus of their case now, they didn't want to hear from him; although -- they didn't want to cross-examine him as it were, although they did -- they are relying on his study, which he of course explained a bit in his letter that was provided to the Court.

There was also some discussion by Mr. Williams of epidemiology. Again, I think the notion of trying to say that the epidemiology that's come out since this allegation first came up can be dismissed because now the Petitioners are changing their focus to clearly regressive autism is certainly not borne

out by the evidence. You can't dismiss this.

The interesting thing about their theory, and which was a little bit frustrating I think when we were going through the general causation, is as you ask each expert is your theory, is your mechanism, whether it's glutathione or it's neuroinflammation, is that going to be limited to clearly regressive cases and they said no. So why would the epidemiology then have to be limited to clearly regressive when the mechanism isn't? The mechanism that's being offered wouldn't just come up in a clearly regressive case.

But apart from that, you've heard from Respondent's experts who have studied autism that there isn't this phenotype of clearly regressive autism, and that was the whole premise for throwing out, as it were, the epidemiological studies.

There was some discussion about Petitioners' Trial Exhibit 17, a letter from Dr. Young, and also the discussion of the epidemiological study that was introduced I think it was the fifth day of trial from -- with the authors of Young, Geier and Geier. That epidemiological study was sponsored by the PSC, as I'm sure the Court is aware from reading the acknowledgements. It was essentially done for litigation purposes.

Dr. Young's letter, which is Petitioners' Trial Exhibit 17. I think there is a little housekeeping we have to do with respect to that. There was -- when that was first introduced during the trial I can explicitly remember saying that whether we would agree that it could be considered by the Court was a matter that needed further discussion.

But if it is considered by the Court, I think it bears mention that there is actually some criticism of the study that's out there now. Mr. Williams said the government hasn't spoken as if the government immediately turns around and can do an epidemiological study responding to what is a clearly flawed study in the course of a month.

That's not how science has evolved. Good science actually takes a little while, and good epidemiological studies take a little while to do. I'm not suggesting that there would be any study done to reply to that. If one canvasses the comments that have come out since the PSC has released this study, they have uniformly criticized the methods used in that study, including criticisms by one of the Petitioners' own experts, Dr. Greenland.

So to suggest that the silence of the government in the face of a litigation-generated,

clearly flawed study means the government has no answer is simply not true.

To hopefully wrap up here, again the government's case is essentially saying that the Petitioners have no good scientific evidence. Good scientific evidence isn't a hypothesis generated by a phone call from Petitioners' counsel to an expert who's appeared before the Court in vaccine cases well over 100 times on myriad issues. That's not good science.

An expert or a witness who will come before you just a couple of weeks before trial ginning up essentially a hypothesis strung together by little pieces of information -- a study of adult monkeys many years ago, a study of infant monkeys more recently, a study of autistic patients a couple of years ago -- that's not how good science is done.

It's not courtroom driven science. It's done by researchers, the types of witnesses the government presented, the ones who are researching in the field of autism, that are making their profession, their lives, about researching autism.

Daubert makes clear that the courtroom isn't the place for speculation and spurious reasoning passed off as science simply because the witness who

appears before you has a Ph.D. or an M.D. after his or her name. It's about the scientific method and scientific process.

After six years and countless opportunities, PSC has failed to offer any such evidence. Instead it's offering essentially the same thing that you've seen in far too many vaccine cases, which is the same experts who seem to think that science in the courtroom is different than science that's practiced by the researchers outside it.

And ironically, and I'm sure this was not lost on the Court, the proponent of the hypothesis that's driving this litigation right now, the proponent of that hypothesis appeared before you, Special Master, only several months before he came up with that hypothesis telling you that he could not conclude, based on the evidence, that thimerosal caused autism.

And yet after a phone call from the PSC he did come up with that hypothesis just a couple of months later. And the irony I'm sure is not lost on the Court either that this witness who came up with this hypothesis of how vaccines are causing autism does not treat children with autism; in fact, does not even treat children for any neurologic conditions at

this point.

Petitioners have failed to meet their burden. They have no -- they have no scientific evidence period. Without scientific evidence they cannot meet their burden under Grant and Althen. They can't prove general causation, and obviously if they can't prove general causation they cannot prove specific causation.

Thank you, Your Honor.

THE COURT: Brief rebuttal?

MR. WILLIAMS: Very brief. First, on the allegation that this is a recent invention, if we had been forced to try this case in 2004, two years after we started, we wouldn't have had the Burbacher infant monkey data. We wouldn't have had any of the neuroinflammation data at all. All of that was published in 2005 and later.

Frankly, we have tried to resist the rush to decide this case because we know how many other important studies are going on that will inform the Court on the very questions where there may still be some doubt like on the dose level of how much inorganic mercury it takes to set off neuroinflammation in infants. We don't think there's any need to decide the case in a hurry when we have so

much important science still going on, but we are here and I think we've proven our case.

Now, as to whether there's a regressive phenotype or not, I think the best evidence you've seen is not created by our experts. It's from the medical literature, and it's the CHARGE study out of California where a group of independent epidemiologists, none of them connected to us, looked very hard at whether or not there was a true regression of both language and social interaction in some kids.

And they found that, yes, in 15 percent of the autistic cases in California that they looked at there was true regression of both of those features. Now, it makes sense from a timing point of view. Neuroinflammation may well explain most autistic cases. It may well be that that's how thalidomide works. We have really good evidence that's how terbutaline works. And it's just a question of timing. It's when are you exposed to the agent that can trigger the neuroinflammation.

In the case today we heard that this child had early insults of mercury, earlier than most children got, but it could well be that the development of these regressive features happens as

the neuroinflammation gets going, and it can't start until they get enough mercury in their brain to trigger it. It makes sense that it would be regression, but that doesn't mean that neuroinflammation doesn't explain other types of autism too like the terbutaline model.

And Dr. -- it may well be that we didn't have Dr. Kinsbourne available to give the opinion in this that we have to have ultimately from an M.D. that gives us causation, but Dr. Aposhian, who is only a Ph.D., has cited all those neuroinflammation papers in his report way back in August of last year, so it wasn't that big a surprise that it was coming. And it frankly, it's the one theory that makes the most sense when you look at all of the medical literature.

I can't believe that they suggested that we were the ones that didn't want Pardo to come. We withdrew -- after we saw his letter and understood how it helped us, we withdrew our objection to his testimony and looked forward to it. And then the government realized that his letter didn't help them too much, and we didn't hear from Dr. Pardo.

The accusation that we're the ones that tried to prevent his testimony, frankly if this Court wanted to really get the facts you've got the power to

ask Dr. Pardo to come independent of us. You can call witnesses on your own. You don't have to wait for one of the parties here to call a witness. If you want to hear from Pardo, let's schedule a time and do it.

That's all I've got.

THE COURT: Any surrebuttal, Mr. Matanoski?

MR. MATANOSKI: No, ma'am.

THE COURT: All right. Well, this then concludes the taking of evidence on the specific case of Colin Dwyer.

What will happen now, for the benefit of those who are not familiar with this procedure, is that we'll generate transcripts of today's hearing. We'll probably have corrections to those transcripts along the model that we've been applying on the other cases, and at that point we'll establish a briefing schedule that will allow the parties to submit posthearing briefs. We'll be scheduling that in one of our status conferences that we have routinely in the Omnibus Autism Proceeding.

Before we conclude today, however, I want to take this opportunity once again to thank the Dwyers for coming forward with their case. It is difficult to sit here and listen to experts analyze what happened and why it happened, and it takes

extraordinary courage to not only live with an autistic child, but to allow the facts and circumstances of his birth and development to be publicly discussed. We appreciate you and your husband's willingness to do that.

Mr. Ferrell, we thank you and your law firm for advancing this case to give us that third test case when circumstances deprived us of the other third test case. I thank you and the Dwyer family on behalf of my colleagues, as well as myself.

We'll endeavor to issue an opinion in this case when we conclude it's appropriate to do so. That applies to I'm sure my colleagues in the other two cases on this theory. I would expect, however, that we'll be issuing the opinion on the first theory test cases first, then to be followed by the opinion on the second theory that we've heard here.

With that, this concludes -- Mr. Powers, do you have anything? You look like you wanted to say something.

MR. POWERS: Yes, I did. I was not too subtle on that. And I would stand up, but my foot has been cramping, so excuse me while I remain seated.

THE COURT: Texas rules.

MR. POWERS: I just wanted to just say

something on the record in terms of a couple of things that the Petitioners anticipate happening after this hearing concludes, but likely before -- in fact certainly before -- the briefing schedule kicks in.

The first is that we do anticipate filing a couple of motions related to evidence. Mr. Williams, for example, alluded, and we've talked about this in one of the status conference calls, about inferences that the Petitioners are entitled to based on evidence that did not come in or that was unavailable. We anticipate getting motions like that filed pretty promptly here at the conclusion of this case.

We also anticipate at least one motion relating to some additional evidence in this case. It's another issue that we talked about in the status conferences, and this is about the toxicology evidence and the effect of Drs. Magos and Clarkson being withdrawn from the witness list and their reports being withdrawn.

We believe that there should be an opportunity for the Petitioners to submit some additional evidence essentially in rebuttal, and it would be a motion, asking -- understanding the orders of the Court on June 17 and July 3, asking the Court to reconsider those motions and potentially allow some

very limited additional evidence. We anticipate that motion coming in.

And then finally, Special Master, we mentioned at the conclusion of the King and Mead cases, but the Petitioners want to make it clear on the record that as new, important, relevant evidence comes into the scientific literature, whether it's HHS' population studies that Mr. Williams described, Dr. Burbacher's second phase of the monkey study, that as long -- up until a decision is reached in these cases we would want leave to introduce that evidence into the record, and if leave is not given and agreed to then again we would have additional motions to file on those issues.

We have nothing filed right now, Special Master, but I didn't want to leave here today blindsiding anybody. We do anticipate getting these teed up with you and serving them on Respondent promptly.

THE COURT: Anything further the Respondent wishes to add?

MR. MATANOSKI: Not to add, not anything further, but in response to Mr. Powers' suggestion that there's some motions coming.

It was I believe either the second or third

motion along that caught my attention in particular. All of them did, listening to all of them, but it was the suggestion that there's going to be essentially a motion to offer additional evidence on toxicology.

The purpose of our status conference and what we talked about, Dr. Clarkson and Magos not appearing, and then that was subject to some discussion and then we said okay, well then if the Court ruled in a certain way about allowing the record to remain open on general causation so we said all right, then in light of that we'll just pull the expert reports of Dr. Clarkson and Magos.

We didn't hear anything about a motion to continue the case at that point, to continue the record being open, and the Court issued its ruling that came out on July 3, I believe, saying the record on general causation is closed. Now, if this is going to be revisited either through motions and perhaps the record ends up being reopened, Respondent may then move to reintroduce Dr. Clarkson and Magos' report.

The idea was to be at an end. We're not in a rush to be -- I mean, if you say we're in a rush, after six years I hardly think that this trial is rushed.

THE COURT: We're rushing slowly.

MR. MATANOSKI: Yes. But there has to be an end at some point to allow you to make a decision. The inference that we're in a rush because we happen to know that there's information coming out that's harmful to us, there is obviously not. That inference should not be drawn.

We just know that the trial was supposed to end in May. There were certain developments that came up because of the availability of Drs. Clarkson and Magos that forced the record to remain open. We were addressing all these issues in May trying to know exactly what was going to happen in July.

With respect to that, it was only going to be Dr. Clarkson and Magos coming back for testimony, but we evaluated our case and decided we didn't actually need them to come back in July, and we felt the record would be closed at that point and we just want the record to be -- we understood it to be closed on general causation.

If it's going to remain open, then we may have to implore the Court, move the Court, to allow us to go back or to introduce other evidence in response to the new evidence that we would receive from the Petitioners.

THE COURT: Well, I'm sure that I'll speak

for my colleagues and say we're always willing to reconsider a decision we've made, and that would apply with equal force to decisions in favor of Petitioners and in favor of Respondent.

So if you give us good reason to reconsider we'll do it. If we don't think the reason is good, we won't.

MR. POWERS: That's what I would anticipate, Special Master. Thank you.

THE COURT: All right. So we do anticipate some motions. Just then as a further housekeeping matter, I anticipate that the Petitioners' master list will be filed into this case, the trial exhibits will be filed into this case, and the expert reports will be filed into this case prior to this record itself being closed.

At this point in this case I've heard that testimony and I've read those reports, but they're not filed into this case so it's important that be done as soon as possible.

MR. POWERS: And we understand that. We'll be working closely with Mr. Ferrell and his law firm to make that happen in Colin's case.

THE COURT: All right. Then the Court is adjourned. Thank you.

(Whereupon, at 2:40 p.m., the hearing in the above-entitled matter was concluded.)

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REPORTER'S CERTIFICATE

DOCKET NO.: 03-1202V

CASE TITLE: Dwyer v. Secretary

HEARING DATE: July 22, 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: July 22, 2008

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