UNITED STATES
COURT OF FEDERAL CLAIMS

HAZLEHURST,	)	
	)	
Petitioner,	)	
	)	
v.	)	Docket No. 03-654V
	)	
SECRETARY OF HEALTH AND HUMAN SERVICES,	)	
	)	
Respondent.	)	

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IN THE UNITED STATES COURT OF FEDERAL CLAIMS

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	)	
	)	
Respondent.	)	

Courtroom 6330
North Carolina Superior Court
832 East Fourth Street
Charlotte, North Carolina

Tuesday,
October 16, 2007

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

BEFORE: HONORABLE PATRICIA CAMPBELL-SMITH
Special Master

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C O N T E N T S

E X H I B I T S

PLAINTIFF'S EXHIBITS:	IDENTIFIED	RECEIVED	DESCRIPTION
1	296	--	Dr. visit report

P R O C E E D I N G S

(9:00 a.m.)

THE CLERK: All rise. The United States Court of Federal Claims is now in session.

THE COURT: Good morning. Please be seated. We are back on the record in the matter of Hazlehurst v. the Secretary of the Department of Health and Human Services, Case No. 03-654V. Mr. Webb, your next witness?

MR. WEBB: We will call Dr. Jean-Ronel Corbier.

THE COURT: Dr. Corbier? Dr. Corbier, did you want to pour yourself a cup of water? Dr. Corbier, would you raise your right hand, please?

Whereupon,

JEAN-RONEL CORBIER, MD,

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

THE COURT: Thank you. Mr. Webb?

DIRECT EXAMINATION

BY MR. WEBB:

Q Doctor, can you give us your name and address for the record, please?

A Yes. My name is Jean-Ronel Corbier, and my address is 990 Leann Drive, Concord, North Carolina.

Q Could you spell your name for the court reporter, please?

A J-E-A-N, hyphen, R-O-N-E-L. The last name is Corbier, C-O-R-B-I-E-R.

Q What is your profession?

A I am a board-certified child neurologist.

Q Would you describe the nature of your current practice?

A Yes. I practice in Concord. I take care of children with neurological disorders, all types of neurological ailments, including autism. I work full time as a clinical neurologist.

Q Are you board-certified in any areas?

A Yes, I am board-certified in neurology, with a special qualification in child neurology.

Q In your current practice, do you treat children with autism?

A Yes, I treat many children with autism, and have done so for the past several years.

Q Are you Yates Hazlehurst's neurologist?

A Yes, I am currently Yates Hazlehurst's neurologist.

Q When did you first see Yates?

A I believe the first visit was back in September of 2002. He had recently been diagnosed.

Q I filed a current copy of your CV as Exhibit 36. Does that CV accurately describe your education and work experience?

A Yes, that should be an updated copy of my CV.

Q Doctor, what is autism?

A Autism is a neurodevelopmental condition that presents with three core areas of deficits. One is problems with communication and language. Children with autism are unable to express their needs. The second area is that of social interaction. Children with autism have impaired social interaction.

And in the third area, it is behavioral. They have very restricted interests. They tend to have self-stimulatory behaviors, such as handflapping. And these three core areas constitute what we label as autism.

Q When do children usually suffer the first symptoms of autism?

A That is highly variable. Some children may show manifestations very early on, a year, sometimes even less, although signs before a year can be quite subtle. Some can start showing signs at 15 months, 18 months, two years, and there have been even case reports of individuals after three, which is unusual,

showing signs. So it is really very variable.

Q In your opinion, do the differences in the timing of the onset of symptoms mean that there are important differences between the autism of those who never develop correctly, those who regress, and those who regress later in life?

A Yes, I think the timing is important, and underlines a point that autism is not really one disorder. There are a lot of different subtypes.

For instance, there are some children that start showing signs very early on, and there are other children that appear quite normal. I think Yates would be a good example of that. And then later they regress, what we call regressive autism.

And so we're dealing with, even though we're dealing with a condition that behaviorally is labeled as autism, there are a lot of underlying etiologies with different times of onset, and different implications therefore.

Q Is the prognosis, or say the likely outcome of autism different in children whose symptoms began at different times?

A It's highly variable there again, because there are some children that start with early signs that may do well. Although in my experience, some

children that have problems from the very beginning may go on to have severe symptoms.

But the reverse may also be true. You may have someone with regressive autism that does well, or you can have a person with regressive autism that may go on to have chronic problems. So here again, it's very variable.

Q In your opinion, the differences in timing of the onset of symptoms suggest that there are differences in what causes autism with an early onset, and those who suffer regressive autism?

A Yes. If someone starts manifesting symptoms very early -- for instance, in early infancy, someone who presents maybe not just with core autistic symptoms, but let's say seizures or other problems -- I would be more likely to think of an antenatal etiology, or genetic, or metabolic, versus someone who is perfectly normal until later on, I would think that other factors, including environmental factors, may have a greater role in these individuals.

So I think the timing can help us sort out the different etiologic factors that are present, as we know that there are various factors that play a role in different cases of autism.

Q What causes a child to develop regressive

autism?

A The belief is that with regressive autism, that, first of all, the child must have an underlying genetic tendency. We think that genes play an important role in most kids with autism. But because they are normal and doing well, we feel that environmental factors also play a role. And so in regressive autism, it's very likely that we're dealing both with genetic influences and external environmental factors.

Q Is it widely accepted that environmental factors are a substantial contributing cause to most cases of regressive autism?

A I think if you read the literature of what has been written, many articles point to a multifactoral etiology, including environmental factors.

For example, Martha Herbert, who is a child neurologist at Harvard, and various others point to environmental factors as playing a role. We don't at this point know all of the environmental factors that play a role, but there's a general understanding that environmental factors contribute to the development of autism.

Q In your opinion, is MMR vaccination an

environmental factor that can cause a genetically susceptible child to develop regressive autism?

A Based on studies that have looked at children with regressive autism, I think MMR has been implicated in a subset of children. It's very important for me to mention subset. I don't think that one can generally say MMR causes autism in general. But if you look at a very specific subset of children with regressive autism that regress after the vaccine has been given, and who have a particular clinical profile with gastrointestinal problems, several, several studies strongly suggest that that is one environmental causative factor.

Q Could you describe for us the clinical profile that implicates the vaccine?

A A typical clinical profile would be a child who is developing normally, doing well, is vaccinated, and subsequently starts having problems: autistic symptomatology, loss of interest, the child becomes withdrawn. But in addition, the child also has significant gastrointestinal manifestations: diarrhea, bloating, malabsorption, so-called leaky gut. Sometimes the diarrhea may alternate with constipation. So a lot of gastrointestinal issues.

On top of that, the child may have evidence

of immunological problems, the child who is sickly. So that profile is highly suggestive, MMR being implicated in regressive autism in that setting.

Q Why would the combination of autism and gastrointestinal symptoms suggest a relationship between vaccination, or MMR vaccination, and the autism?

A Can you repeat that again?

Q Why would the combination, if you will, of regressive autism which begins after vaccination and specifically gastrointestinal symptoms, why would that implicate the vaccine?

A Okay, that's a good question. Before we even look at the vaccine, just to answer your question, we have to ask can MMR, or I should say can the measles virus or other viral infections for that matter, can they cause regression, can they cause neural behavior, neurologic and gastrointestinal problems.

Several, several studies have implicated the measles virus itself as a contributing factor to both gastrointestinal problems and neurological problems, including autism and developmental delay.

But we can go a step beyond that. And if you see a group of children with autism and

gastrointestinal problems, you can look at their gut and try to isolate a measles virus, which has been done by Uhlman, who actually looked at a group of developmentally delayed children with gastrointestinal problems.

But others have taken it a little bit further. For example, Dr. Kawashima has found that the persistence of measles virus in the gut of individuals with autism, he was able to tell that that strain was not a wild strain, but was actually a vaccine strain.

I think another good study was by, I believe it was Bitoun, who actually found, there was a case of a child who was vaccinated at 12 months with the MMR, did well, and then eight and a half months later that child developed seizures, started to regress, had a lot of problems. Unfortunately, that child eventually died. But in the process, a brain biopsy was done, and it was very revealing. It was found that that child had persistence of measles virus in the brain. And then when they tried to see exactly what type of strain this was, it was a vaccine strain.

And so I think there is ample evidence in the literature to suggest that the MMR is associated with persistence of measles virus in the gut, and also

in the brain. Bradstreet and others have shown that the cerebral spinal fluid of children, of certain children that were looked at with autism had the measles virus. We also know that the measles virus is very virulent and very immunosuppressive. So it would make a lot of sense, based on a biological framework, to suggest what I'm saying.

Q Now, this child you described where they did the autopsy and found presence of measles in the brain, did that child have autism?

A The child had autistic symptoms, but he actually developed a lot, he developed a lot of other symptoms -- seizures, he later became comatose. And so he was diagnosed with inclusion-body encephalitis.

Q Does the measles virus cause a variety of different central nervous system illnesses?

A Yes. Yes, I think what is very interesting with the measles virus is that we have in front of us a natural history in the environment of what happens with, when someone gets infected with the measles. Although most individuals in the past who were infected with the measles virus developed symptoms that were temporary and then resolved, there's a group of individuals that developed acute encephalitis; that is, after they were injected with the, or after they

received the illness, they then developed encephalitis, neurological problems, seizures, confusion.

But then there's another group that did not show any manifestations after they were infected with the measles virus until later, until anywhere from one to nine months later. These people developed a type of encephalitis we call subacute encephalitis, or inclusion-body encephalitis. And that's very interesting, because frankly, the majority of individuals in the past who had the measles infection did well recover. But there was a subset that went on to have postinfectious encephalitis.

In fact, in the group of those who had the inclusion-body encephalitis, there's a subset of them that had clear evidence of immunosuppression. And then there's a third group that, after they were infected with the measles virus, did well not for a week or two, a month or two, but several years, and then developed an acute encephalitis. And most neurologists in this country, especially child neurologists, know this entity as SSPE: subacute sclerosing panencephalitis. It's a condition that is preceded years in advance by measles, and then something happens which causes that individual, due to

persistence of the measles which may be transformed in the brain, to then develop a very serious condition that we call SSPE.

So I think if we look at these models that are right there in nature -- you have an illness where you have a virus that can cause different types of postinfectious problems -- we have a perfect model for looking at measles, mumps, and rubella vaccines, which aren't that live attenuated vaccines.

So the question is, if I may elaborate a little bit, can the MMR vaccine cause measles infection? Well, we can also look at other vaccines that have been used in the United States to answer that question, such as oral polio.

Oral polio was discontinued in the U.S. because there were several cases of individuals that were vaccinated with the oral polio vaccine to try to prevent the illness, when they were, you know, not necessarily very likely to get the illness. But then they came down with polio. So the decision was made several years ago to not give oral polio, but instead to give an inactivated form of polio, which I applaud that decision. I think it's a lot safer.

But with measles, with MMR, it's very likely, and there are a lot of evidence and reports in

the medical literature of individuals persisting with the measles virus, both in the gut and the brain. And perhaps later we'll talk about brain-gut connections. So to answer your question.

Q Do we know why some individuals develop SSPE years after they are exposed to the measles virus?

A I think the best explanation, although there are things that we still have to learn, is that everyone is different. Perhaps people have different genetic makeups, and that's one reason I think genes are so important, such that some people may develop an infection early, others later on, and others, most people, not at all.

And I think the same thing applies with MMR. I think most people that are vaccinated with MMR have absolutely no problems. I received my MMR vaccine a few weeks ago for my booster, because I didn't suspect that I would have problems. I have not in the past. But that does not mean that everyone has the same immunological makeup as I do. There are individuals that have a particular makeup that make them vulnerable.

So I think genes play a role in the person's constitutional makeup that would determine if someone tolerates it well, and in most cases people do --

(Electronic interference.)

Q You described at least three different kinds of central nervous system illnesses caused by measles vaccine, measles virus: the acute measles encephalitis, subacute measles inclusion body encephalitis, and SSPE.

A Yes.

Q Is there any reason to believe that that's the whole universe of central nervous system diseases caused by the measles virus?

A Well, I think these are the three entities that have been best described, but it's likely that there's a whole shade in between. In other words, you may have individuals who may have something that's a cross between acute encephalitis and the typical subacute encephalitis or inclusion body or you may have someone that has a clinical profile that's not quite similar to that SSPE.

So I think it's very reasonable that there's a whole spectrum of problems, and those three just represent what's been studied and identified in most of the prototypical cases, if you will.

Q Turn back to the significance of evidence of measles virus persisting in the intestines. In your report you cite a report by -- Petitioners' Exhibit

37-E -- Uhlman?

A Yes.

Q Potential viral pathologic mechanisms for new variant inflammatory bowel disease. And you cite that for the proposition that measles virus is found in the gut of children with autism. Do you feel that the Uhlman findings are reliable?

A I believe that Uhlman's findings are reliable, based on the fact that he used a lot of controls and up-to-date techniques to try to verify the persistence of measles virus in the group of children that he studied: namely, children with developmental delays. And so, to answer your question briefly, yes, I think that that was a well-done, valid study.

Q Have other people duplicated any part of that study?

A Yes. I think there are several labs that have tried to look at Uhlman's study, and I think some labs used techniques that were a little bit different. For example, I believe that Uhlman looked at the gut tissue, while other labs have looked at the blood to try to see if they could replicate the virus. Some of those labs, not surprisingly, came up with different findings. In fact, one of the labs did not even evaluate children who actually had gastrointestinal

problems.

But I think Dr. Stephen Walker's lab has done a better job at replicating the findings than other molecular biologists, Dr. Hepner and others. So I think that that study has been, or that finding has been replicated and validates the findings of Dr. Uhlman and others.

Q And Dr. Stephen Walker, is that the report that Dr. Hepner testified about in the Cedillo case?

A Yes, I believe that is the one.

Q Would the measles virus need to be present in the brains of children for it to cause central nervous system injuries?

A Yes and no. Let me explain. Various viruses, and not just the measles virus, someone can be infected with a virus. They may get rid of the infection, but then subsequently the immune system may overreact and create what we call an autoimmune disorder.

When that occurs, the antibodies may travel everywhere, including the brain, and cause problems. So that would be one case scenario where a virus, including the measles, without attacking brain cells, may result in a neurological problem.

But of course, in many other cases, as has

been shown by brain biopsies and cerebral spinal fluid, which is a better way to assess what's going on in the nervous system, have shown the persistence of measles virus in the brain, which more directly implicates the virus as causing neuronal damage.

The measles virus comes in a variety of changes, toxic changes, that can damage neurons. So that mechanism also exists.

Q Can autoimmune responses in the gut cause central nervous system injuries?

A Yes. I think there are several good examples of that. I think it was Maroudi who did a very nice article on celiac disease. Celiac disease traditionally has been considered a gastrointestinal problem. Celiac disease is a condition where some individuals do not tolerate gluten. Gluten is a protein that's found in wheat and rye.

And when individuals that have celiac disease partake of gluten products or wheat, the immune system elicits a very abnormal reaction where it attacks the gut. And the resulting problem can include chronic diarrhea in small children, weight loss, malabsorption, just a lot of symptoms.

Well, Maroudi showed that in addition to gastrointestinal problems, some individuals have

brain-related defect. There's Maroudi, there's Goodwin, and several authors that have shown anything ranging from cerebella ataxia, where the immune cells attack the cerebellum, which controls balance, in individuals with celiac disease. Goodwin has shown that you can develop strokes from celiac disease.

So there's a whole range of seizures. Actually, I think Maugrouder or Macken talked about epilepsy. There was a case reported in one of our journals, the Pediatric Neurology Journal, where an individual presented primarily with seizures, refractory epilepsy. And they happened to be doing a study, and that individual was diagnosed with celiac disease.

So I think there's clear evidence. There's celiac disease, there are inflammatory bowel disease like Crohn's disease, ulcerative colitis that are well known to cause extraintestinal problems, including neurological problems.

So I think it's very clear, undisputed, that many immunological problems that affect the gut also affect the brain. No one can deny the brain-gut axis.

Q Did some of those studies actually look at the antibodies doing the damage in the gut and the brain?

A Yes. Some of these studies have found that

the antibodies had receptors, not only to the gut, but also to the brain. And this is something that we're finding with a variety of chemicals, even secretin that has been looked at in autism, there are receptors for secretin in the gut. But there are also receptors in the brain.

That should not be too surprising, because we have millions and millions of nerve cells in the gut. I like to think of the gut sometimes as a secondary brain. So there are a lot of neurological cells in the gut. And so again, that supports the relationship of gut and brain.

Q Now, when someone suffers central nervous system symptoms of celiac disease, like ataxia or seizures, is the celiac disease or the intestinal aspect of the celiac disease always apparent when the person suffers the central nervous system illness?

A Not necessarily. There have been several cases, for example in gluten ataxia, so-called gluten ataxia, someone may present with an unsteady gait. That's what the term "ataxia" means. And the gastrointestinal symptoms may be very mild.

In the other case I talked about with celiac disease and refractory seizure, here was a child who presented with hard-to-control seizures. And it's not

until it was recognized that the child had celiac disease, had these antibodies directed to the gut, but presumably to the brain, only then was that child taken off of gluten, placed on a gluten-free diet, and guess what happened to the seizures? The seizures got better. The child was on antiepileptic medications as well, but the point is that child was refractory to treatment with the medication until an autoimmune process, such as celiac, was discovered. Gluten was removed, the seizures stopped.

Q In your opinion, is exposure to thimerosal as a result of vaccination an environmental factor that can cause a genetically susceptible child to develop autism?

A My answer to that is yes, I believe that thimerosal is among the environmental factors that are implicated in autism and related problems. And I base that statement on the fact that, first we have to understand what thimerosal is.

Thimerosal is a preservative that had been very poorly studied in the 1930s. I think there was one case where they looked at toxicity in an individual just for one day, and concluded that it was safe.

But thimerosal is 50 percent ethyl mercury.

Ethyl mercury is a type of organic mercury. There are

different types of mercury. There is elemental mercury, there's inorganic and organic.

Well, ethyl mercury has been found to be neurotoxic, highly neurotoxic, like other organic mercury, such as methyl mercury, which has been studied in various populations. But the question is, it's one thing to say that thimerosal is neurotoxic; it's yet another thing to say that it's implicated in conditions such as autism, or developmental problems, for that matter.

There have been several studies first that confirm that thimerosal was toxic. There have been some outbreaks in Japan, so-called Minamata disease, and Iraq, which led the United States to do further studies and look at certain islands, such as the Faroe Islands in the Seychelles, where they were noticing that a lot of children had neurodevelopmental problems that were unexplained.

Well, further studies showed that they were exposed to methyl mercury: the parents were, the mothers were. And it affected the children much more than they did the parents, which led researchers to note that the developing brain is very susceptible to methyl mercury.

Some studies have looked at methyl mercury,

and compared methyl mercury with ethyl mercury. And more and more studies are being done, and there is good evidence to suggest that ethyl mercury, although it's a different organic mercury, it's also very toxic. In fact, if or when ethyl mercury reaches the brain, it converts to inorganic mercury seven times faster than methyl mercury.

There are further studies that have been done to answer the question of exposure. After all, many children are vaccinated; not everyone comes down with autism or neurodevelopmental problems. So I don't think it's necessarily an exposure, per se, but studies show that it's a problem with excretion. Many children with autism do not excrete mercury very well.

Amy Holmes, in a very recent study done, looked at mercury in the teeth, and confirmed that children with autism, many of them have a much higher burden of mercury than children who do not have autism.

So really, all of these factors strongly point to the fact that in some individuals, particularly those that are genetically susceptible, can, as a result of thimerosal, develop autism. Perhaps along with a few other factors.

Q Do you believe that Petitioner's Exhibit 48,

Thompson, et al, early thimerosal exposure and neuropsychological outcomes at 7 and 10, that was published September 27 in New England, 2007, in the New England Journal of Medicine. Do you think that the results of that study hurts your case for thimerosal causing neurologic injury?

A I actually think that this case helps to support that thimerosal can cause a neurologic injury. I don't think that study focused on autism.

But what's an interesting finding is that children were noticed to have increased tics, both motor tics and a type of tic where sound is produced, that we call vocal or phonic tics.

Many children, in fact, with autism, or a subset of children with autism have tic disorders. That's a well-established fact. So that leads me to believe that thimerosal can cause other neurological problems, as well, such as tic disorders.

I might add I had one child that I saw several years ago who was vaccinated, and a day or two started having tics. And that child had multiple tics he started having. He was not diagnosed with autism, but he had behavioral problems. And that child did well after mercury was removed from his system; the tics stopped right away.

(Nearby interference.)

So I think thimerosal is neurotoxic in many ways, and of course can affect other organs, as well, including other organ systems, like the immune system.

Q Doctor, does Yates Hazlehurst suffer regressive autism?

A Yes. I was here yesterday, and I heard the excellent testimony of several family members. And to me it was very clear that autism, or that Yates was developing very normally up until about the first year of life.

And from what I saw and heard, we were able to look at some videos, he did regress afterward. I was very interested to hear various perspectives from family members, parents, grandparents, and I don't think there could be a better description of regressive autism. So Yates does have regressive autism.

Q When, in your opinion, did Yates suffer the first symptom of autism?

A From what I heard in the testimony yesterday -- and I'm very glad that I was able to listen to the testimony, because it gave me even bigger insight than I had when I first saw Yates and in the several years that I followed Yates. But what

I heard fairly consistently is maybe a couple of months or so after his 12-month vaccines, he started showing signs of what several family members have referred to as running wild. He was wild, which means that he was very hyperactive. He had a tendency to wander. He had a tendency -- he was just a different child. When family members, he was not as much a joy to be around as he had been before.

And then he started having other symptoms, as well: loss of interest with others, a decreased social interaction. He started becoming a picky eater. There were so many changes that were noted, but from what I heard, I would say probably about a couple of months or so after his 12-months vaccine. So by, let's say, April or March, in that timeframe, as best as I could gather.

Q Can you tell us which of those things that were described yesterday by the family members you think are aspects of his autism?

A Yes. I think, first of all, autism is best viewed as a collection of symptoms or signs. There are a lot of different things that fall into this spectrum of autism.

So the first thing that I saw in Yates's case that suggested to me a change pertaining to his

autism was his activity level, going from a fairly normal child, going from an active child to a hyperactive child who was described as very wild. So I would say that that would be the first autistic symptom, listening to the testimonies, that I could pinpoint.

Q How about the description that we heard that he lost interest in playing with his cousins, for example? Was that a symptom of his autism?

A Yes, most certainly. In fact, loss of interest with individuals, your surrounding, would be one of the core features of autism. So initially he played very well with his cousins: again, we saw videos of him interacting with his two older cousins. They were happy. He appeared happy.

But then that changed. To a point where the cousins did not want to interact with him as much. Even the young cousins had noticed a difference. I think if young children can see a change, it must be pretty obvious.

Q The description of him losing interest in toys or not playing with them in the way he had before, was that a feature of his autism?

A Yes. Children with autism view the world, view the universe in a completely different manner

than individuals that do not have autism. It's not that children with autism do not play with toys, but they do not play with toys in an appropriate manner. And the toys or the objects that they use is very restricted.

I think it was a very nice scene where Yates was playing with the bowl, a glass bowl. He took the bowl, he turned it around, and he started to roll it. In fact, rolling that bowl was a very typical type of activity that a child with autism might engage in, sometimes for prolonged periods of time.

Q Was that the scene from Amsterdam, where he's in a diaper?

A He was in a diaper. I can't remember if he was in Amsterdam or not, but he, I think he took the bowl from the table, he put it down, and he started to -- yeah, I think he was in a diaper -- he was rolling the bowl back and forth. He put it sideways and started to roll.

Likewise, a child with autism who had a car, for example, instead of playing with the car in an appropriate manner might flip the car or truck upside-down and spin the wheels. That's a very typical autistic behavior.

Q So his aunt I think described that his

favorite activity, I think in

late summer, would be to turn over, I think it was a stroller, and spin wheels, was that a feature of his autism?

A Yes. That's another typical, spinning wheels, stacking up objects, lining up objects. That gives you a sense of stereotypic repetitive behavior, which tends to fascinate children with autism.

Q You mentioned a moment ago that he became a picky eater. Do you see that as a feature of his autism?

A We see a fairly good subset of children with autism that become very picky eaters, even if they were eating very well prior to that. Now, let me say that nonautistic children can also become picky eaters temporarily, to a certain extent. But children with autism, especially those that fall under, or those that has gastrointestinal problems, immunological problems, can become very picky.

And I think Mrs. Hazlehurst gave a good description of some of the physical features of Yates after a period of time. He was not eating, and it showed. He had a protuberant belly, which most people would think of a malabsorption type of problem.

So we're dealing with very significant picky eating, which we know can be caused by a lot of

problems. Not only neurological impairment. By the way, mercury, one of the things mercury can do, mercury toxicity is a cause of anorexia.

But beyond that, there are gastrointestinal problems, reflux, esophagitis. Yates was later diagnosed with inflammation of the gut, of the colon, due to biopsy. So all of these things can combine to cause picky eating.

There are also sensory problems. People, kids with autism sometimes have what we call oral defensiveness. So the mere fact of putting food in their mouth could be something that's very hard for them to tolerate. So all of these explain why children with autism are picky eaters.

Q The parents describe a -- change in the way Yates used language from, I don't want to -- from "by," and "please," and "thank you," to basically numbers and letters. Is that a feature of his autism?

A Yes. If you look at all of the changes, all of the linguistic regression that Yates underwent, we see that there are several patterns. One is he had loss of some of the words that he had mastered before. He also had loss of what I would call pragmatic language. It's one thing to say words. We see kids sometimes that may have a huge vocabulary, but they

don't use it in a meaningful way, like conversational manner. Yates had reached a point where he couldn't express his needs. So I think there are various ways in which Yates's speech and language were impaired and altered.

Q And in the video we saw a very brief scene where Yates was getting off the bus. And we had a brief scene where he might have been flapping his hands. I guess I'm asking, is that, in your opinion, handflapping, is that an autistic characteristic?

A Yes. One of the common findings or signs in autism is what we call self-stimulatory behaviors. Self-stimulatory behaviors are behaviors that are partially or mostly involuntary, where some behavior is repeated over and over.

A self-stimulatory behavior could be motor, so handflapping would be a good example, actually a very common example that we see in children with autism. You can have visual stimming. Visual stimming is where you tilt your head a certain way just to kind of see things in a particular way that is soothing and pleasing to your visual field. Or in other words, your visual field allows you to perceive things in a way that soothes your brain. So instead of verbal-motor-stimming, you can turn your head a

certain way.

Some kids will just kind of line an edge and see things. Or I think a perfect description was given of Yates looking at the video or TV, looking at credits, and things rolling, scrolling up and down. That was a form of visual stimulation. So yes, the handflapping is definitely a form of autistic behavior.

MR. WEBB: If I might, yesterday at the end of the proceeding, the Respondent inquired into Dr. Corbier's most recent, records of Yates's most recent visit to Dr. Corbier. And I have provided a copy to the Respondent, and I will file it. But I thought I would like to hand you a copy, as well.

THE COURT: Please.

MR. WEBB: Because I'm going to ask a couple questions about it. I have multiple copies; I made five. If you need a couple -- would you guys like to take another one?

MS. RENZI: Yes.

MR. WEBB: So more than one person can look at it?

MS. RENZI: Thank you.

THE COURT: Okay, give me a moment. I'm going to look at our docket sheet, anticipating, as we refer to this, what --

MR. WEBB: We could either identify it as a trial exhibit or --

THE COURT: I was going to say it might be --

(Away from microphone.)

MR. WEBB: -- it could be filed subsequently. I know in Cedillo a lot of things were filed as Trial Exhibits -- I haven't even thought through which is the best approach to take.

THE COURT: Let's do this. Let's start our trial exhibit list, although I think in an ordinary exhibit it would be Exhibit 58. But let's start with this marked as Trial Exhibit 1.

MR. WEBB: Petitioner's Trial Exhibit 1?

THE COURT: Right, PX-1.

(The document referred to was marked for identification as Petitioner's Exhibit No. 1.)

BY MR. WEBB:

Q The first question I'm going to ask you about this is under "developmental history."

A Yes.

Q Is the history you provide there in your record inconsistent with your testimony that you've given today about when Yates's regression began, or

occurred?

A Yes, that's a good question. This, when you

read it, it says he developed normally until the age of 18 months to 24 months. What I saw yesterday indicates that he actually started to regress sooner than that. It's not uncommon sometimes when we're getting a history to, you know, list something, and then to go back and get further detail.

So there's no doubt in my mind that, based on the testimony that I heard yesterday, which was a little bit more detailed, I was able to get than in the office, because frankly in the office we didn't really focus too much on the development. And I think I wrote something similar in my initial visit in September 2002.

So I would say that the most accurate picture of what happened to Yates is what I saw yesterday, corroborated by several family members.

Q When you try to determine the cause of a child's neurologic disorder, what do you do?

A The neurological -- autism, or just neurological?

Q If you have a patient, and you, for some reason, are trying to determine what caused the child's neurologic illness, what process do you go through in trying to do that.

A When I see a child for the first time that I

want to evaluate neurologically, the first thing I do is to get a history, good history, the best I can, from the parents. That history-taking can evolve over the course of several visits. So the history is very important, because, based on the history, I can have a sense as to what the diagnostic possibilities are.

Then I do a general and neurological examination, mostly to test my internal hypothesis, the differential diagnosis that may be present. If I still don't have an answer, and if it's indicated, I will do certain testing. The evaluation that I do will depend on the symptoms that present, and problems, and these may include neuroimaging, electrodiagnostic testing, such as EEGs, lab work. And so these are tests that we may do.

I may have a particular presumptive diagnosis that may get another piece of information down the road that may alter or fine-tune my thinking, and may cause me to refine my diagnosis. So that's the general approach that I take.

Q When did you first develop the opinion that Yates Hazlehurst's February 8, 2001, MMR vaccination and the thimerosal contained in vaccines he received then and earlier in his life, contributed substantially to his autism?

A As I recall, with the first visit I was struck by a few things. I don't have that first visit in front of me, but I can basically remember that I was struck by the fact that he had regressive autism, based on the history. I was struck by the gastrointestinal problems that he had had, and I was struck with his recurrent infections.

And so basically, I looked at the possibility that the vaccines could have played a role. I did not necessarily make up my mind. And in fact, my approach was to try to rule everything out first, and to use that as a diagnosis of exclusion, because there are other factors that can cause regression.

For instance, if I have an 18-month-old in front of me who looks dazed, is not responding, that child may be having a type of seizure we call nonconvulsant status epilepticus. And we see this from time to time.

There are other metabolic conditions that can present superficially like autism. So even if I had a history that suggests a possible vaccine injury, I would first, not just relying on the history, use that information, but try to rule out other factors first. And if I cannot come up with a better

explanation, then it's to do some testing or see what tests are done to support the notion that vaccines might play a role.

Q From the description you give us, I take it that you were aware of the proposition that MMR vaccines, or thimerosal, might cause autism before you saw Yates.

A Yes. Yes, I was aware of that, yes.

Q Did you apply the process you described two questions ago when you tried to determine what was causing Yates's autism?

A Yes, I did. We had done several tests, not only right after that visit, but with subsequent visits, just to try to get a sense of what might be present, what underlying problems might be present with Yates.

I also encouraged the parents to do everything they can in terms of other specialists, because I look at it as a team approach. No single physicians has all the answers. And I encouraged them to keep me informed of some of the tests.

And so, I think I did a prolonged EEG, a 24-hour EEG and several labs along the way to specifically try and find out what the underlying factors might be.

Q In your opinion, was the MMR vaccination

that Yates Hazlehurst received on February 8, 2001, a substantial contributing factor to his regressive autism?

A With all the evidence, both in Yates's case and also what I know in general based on studies, I would say that the MMR played a significant role. In the case of Yates, I could only reach that conclusion after several other tests had been ruled out. He's had a very extensive genetic workup. He's had a karyotype (phonetic) high resolution where his each chromosome was combed very carefully to see if he might have a specific genetic disorder, as various genetic disorders can cause autism.

He's had what's called subtelomeric deletion testing; it's one thing to rule out chromosomal disorders. He had another thing to look with a fine comb, if the ends of the chromosomes might have a little mutation that's called subtelomeric deletion.

He had fragile X, which is a fairly common, relatively speaking, genetic cause of autism. That was ruled out. He had methylation studies for Praterwilly and Angelman's Syndrome, which is located on chromosome 15. That was ruled out.

He's had a variety of metabolic testing, including amino acids. There's a condition called

phenylketonuria, or PKU, which can, untreated, present as autism. That was checked, and in fact, all the organic gases were normal. And a variety of metabolic testing. He has had an MRI of the brain that was also normal, ruling out a structural abnormality. There are some structural problems that could mimic autism.

So all of these things having been ruled out. And he did go to Harvard, and he saw Dr. Tim Buie, who did a scope. He also had a biopsy of the gut, which showed that he had colitis. He had also a finding of a nodular hyperplasia, which has been described by various individuals: Wakefield, Uhlman, and various others.

And along with his profile of regression, and also not only neurological deterioration, but gastrointestinal problems following the vaccine, I must say that I cannot find any better explanation, as a regular child neurologist seeing patients like Yates and others. I must conclude that that played a significant role in Yates's case.

Q In your opinion, would Yates Hazlehurst have developed regressive autism if he had not received the MMR vaccination?

A I really don't have any basis to say that he would not have, based on the evidence that I have at

hand. You know, we see a child who is developing very well, who I think was actually advanced in many ways, getting vaccinated. And then subsequently, a couple of months later, a slow regression into the world of autism.

Because I don't have any better explanation, with all of the labs that have been done, not just by me, but by two other neurologists. He saw Dr. Zimmerman at Johns Hopkins, and I think he saw another, I think Guggelheim or someone else in Tennessee. He's been seen by immunologists, he's been seen by Tim Buie. So I could not find any alternative explanation for that.

I just want to state that, you know, initially if you have a child who regresses after a vaccine, it could be coincidence; that's a possibility. Or the vaccine may play a role. And I think if you rule out all the other causes, and if you have a good explanation that supports the persistence of measles virus in the gut and the brain, then it's a very fair assumption to say that that plays a role.

We do not have tissue biopsies that show the measles virus in Yates, although I think that that was a goal to have that done, and I would welcome that if, you know, that were made available. But knowing that

children with, many children with lymphonodular hyperplasia who have gastrointestinal problems and autism, that it's been shown through several studies that the measles virus is present. We can then infer that, since he has the same findings, although we do not have the measles test done in Yates, that that's what is going on in this case.

Q When you introduced that answer, I'm not sure you answered it in a way that I understood. I'm trying to ask you, do you believe, well, do you think that Yates would have developed autism had he not received the MMR vaccine?

A No, I don't think he would.

Q You've mentioned some of the facts specific to Yates's regressive autism that made you think the vaccine contributed to this, was a substantial contributing factor in his disease. How important in your analysis was it that he was normal before the vaccination?

A I think it's very important. In fact, I don't think I would have considered at all this possibility if he was in the other group of children who have problems since early infancy.

What made me even consider this possibility is that he fit a particular profile. It's my belief,

having worked with hundreds of kids with autism, that we're not dealing with one disorder. Instead, we're dealing with a variety of subtypes of autism. And it's really a little bit problematic that we don't have any particular laboratory tests at this time today to diagnose autism. It's a clinical diagnosis.

So we have to do our very best to categorize the subtypes. Yates fits very well in the subclass of children with autism who not only has regressive autism, but also has gastrointestinal problems, who has been sickly with recurring infections. So that was the first step that led me to a consideration or inclusion in my differential diagnosis that that could be Yates's problem.

Q In order to fit this profile, how soon should a child's first symptoms of autism begin?

A As far as the regressive or the postvaccination?

Q I didn't make that clear enough, because I want to make it clear. You mentioned several times that Yates fits the profile that suggests the MMR vaccination contributed to his autism. And you said that regression after the MMR vaccination was a part of that.

And I'm trying to ask you if there's a time

period in which the regression should occur after the

MMR vaccination for a child to fit this profile.

A I see, yes. My answer would be based on multiple other cases that have fit a similar profile, and also what I know about the natural course of a post-measles-virus encephalitis.

We know that with postmeasles encephalitis, especially the subacute type, you can start having symptoms in the first month, or you may not have symptoms until nine months later. There is the case I mentioned earlier, I think the author is Pitnum, who demonstrated a case of postmeasles, post-MMR-measles encephalitis that occurred about eight and a half months after the child was vaccinated with MMR.

Based on all of that information, I would say that the range would be anywhere from eight to nine months, based on the factors that I have mentioned. And I think it would, of course, vary with the individual, and based on what other factors might be present, as I think the best way to look at these cases is to look at it in terms of contributing factors.

Q In your opinion, is the child's case stronger where the first symptoms of the autism are a month or two, than at eight or nine months?

A Well, not necessarily. Not necessarily,

because clinically, variation is the name of the game. Some things occur early, some things occur late.

I think that one has to, I think that if you're in that one to nine months, that's pretty good. That's a good suggestion that you're dealing with a postinfectious problem, or a postmeasles vaccine problem.

But whether it occurs at one months or three months, I don't think that we have enough information to say that it's more likely at one month than four months, or at least I have not seen that in the literature.

Q In order to fit this profile, was it necessary that Yates had gastrointestinal symptoms?

A Yes. That's one of the other factors that I think from the very start caught my attention, is that Yates had a lot of gastrointestinal problems. The fact that he had gastrointestinal problems raises the issue of could he be in the subset of children that have this so-called lymphonodular hyperplasia colitis, or inflammation of the gut.

And frankly, I was not surprised when the testing from Dr. Buie showed that he did have colitis, he did have lymphonodular hyperplasia. And although the findings from Dr. Buie were called mild, they

weren't mild, they weren't so mild that he did not feel that medication was necessary. He placed him on medication, and he started doing a lot better.

So I think that the gastrointestinal symptoms, just based on what we know in the literature based on studies that have made this association of autism, developmental delay, lymphonodular hyperplasia, that that played a significant role in my diagnostic impression.

Q What kind of gastrointestinal symptoms would you need in order, for a child to have, to fit this profile?

A I would say you'd have to have a child who has diarrhea, who has constipation, but mostly the diarrhea. A child with reflux possibly, though I would say diarrhea would be a bigger component. Having evidence of a malabsorption would be an even stronger case.

Because what the underlying pathology shows is basically a gut-related problem that's immunologically based. So there's something attacking the gut, causing swelling. That swelling can cause leakiness of the gut. That leakiness of the gut can cause certain nutrients that should properly be absorbed to get inside the bloodstream; hence, can

cause the multiple food allergies that we tend to find.

In fact, Dr. Buie's report mentioned that there was an eosinophilic component. I have spoken to several pediatric gastroenterologists to ask them what is the significance of the eosinophilia in the gut, and they say that it is an indication of some type of allergic response. So I think all of these things are quite significant.

Q In your opinion, was the thimerosal that Yates Hazlehurst was exposed to through his vaccinations a substantial contributing factor in his regressive autism?

A I think thimerosal played a role, but I don't think that it played a role that was as great as the MMR. And this is based on the following findings.

If you look at his vaccines, he had DTaP -- diphtheria, tetanus, acellular pertussis -- of the infanrix type, which did not contain thimerosal. He did have hepatitis shots, which I think did contain the thimerosal.

But I was able to find in his labs some indicators that the thimerosal played a role. And these indicators were his glutathione, which is an antioxidant, one of the most important antioxidants in

the body. The glutathione is a ubiquitous molecule that counters free radicals throughout the body and the brain. And it is well known, very well known through the work of Dr. James and colleagues and various others, that children with autism have a very low, or have a lower glutathione compared to nonautistic individuals.

If your glutathione is low, of course, that can contribute to your recurrent infections. His glutathione level was low, and of course, that can also lead to oxidative stress.

The other finding in his labs is that he had significant elevation in one of his porphyrns. Porphyrn is a substance that is in our heme, which, when synthesized, becomes hemoglobin. And that heme is what gives the blood the redness of its color.

Well, his copoporphyrn, one of the porphyrns called the urocopaporphyrn (phonetic), the rate was significantly elevated. The problem is mercury has been shown, through a study from France with Dr. Nataf, and later replicated with a perspective study with Dr. Geier, that the mercury can literally inhibit the synthesis of the porphyrns.

With this inhibition of the synthesis, the porphyrns, some backup products will rise in the body

and will be excreted in the blood. You can check that in the blood. And so porphyrns are a good way to test for mercury toxicity, and his was elevated.

So we do have some laboratory evidence that the thimerosal played a role in Yates's case. Although in fact between MMR and thimerosal, I would say that the MMR had a larger contributing role.

Q Do you believe that the thimerosal's role was a substantial contributing factor to his autism?

A Well, I think, based on the lab tests, that it did contribute significantly enough to cause impairment in his porphyrns, and also significantly enough to cause his glutathione level to be lower than normal.

(Pause.)

Q Let me ask you a question about, you mentioned that you think that the MMR vaccine's role was more than the thimerosal -- if the Special Master were to, say, for one reason or another, not accept your testimony concerning the MMR vaccination, do you think the evidence concerning thimerosal is sufficient to stand by itself as a cause of his autism?

A From what we know, again with various studies looking at thimerosal, we know that thimerosal is linked to autism through various studies I pointed

to, studies from Dr. Holmes, Bradstreet, and others, that show that children with autism have a bigger burden of mercury, not just exposure, but in terms of excretion.

And so, based on the labs, if I did not have any labs the answer would be no. But because we do have some labs, I think that that's sufficient.

I did mention that the DTaP was not the type that contained mercury; but again, we can't just look at the amount. We can't look at the level of mercury.

So although I think that, comparatively speaking, the mercury may have had a lower case -- and I'm making that argument just based on all of the evidence that I have -- I do think this significant event would have played a significant role.

Q If, on the other hand, the Special Master were to, for whatever reason, reject your testimony and the other evidence available that thimerosal contributed to Yates Hazlehurst's autism, are you of the opinion that the evidence concerning the relationship between the MMR vaccination and Yates's regressive autism is sufficient to stand on its own?

A Yes. I think, based on the testing that he had that demonstrated lymphonodular hyperplasia colitis, his entire profile, and what I would call

pertinent negatives -- that is, exclusion of other possible causes for his autism -- I would say yes.

Q Do you hold the opinions that you have expressed today to a reasonable degree of medical certainty?

A Yes, to the best of my knowledge, the opinions that I expressed were correct, based on my way of thinking.

MR. WEBB: That's all the questions I have.

THE COURT: Thank you. I have a few questions, Dr. Corbier. I wonder if counsel is interested in a brief break before we move into cross?

MS. RENZI: Actually, Special Master, if we could take maybe a half-hour at this point, then I know we could probably finish up before lunch for the end of the day.

THE COURT: Okay. All right. I just have a couple of questions, Dr. Corbier, before we do this.

You have referenced several times the profile. As I go forward, would you tell me in your view, what is the profile that you believe Yates represents?

THE WITNESS: He represents that profile of a specific subset of children with autism who, number one, have regressive autism. So children who were

1 developing normally, and at a particular point

start to regress. That's number 1.

Number 2 in the profile are children with regressive autism, that in addition have significant gastrointestinal problems; so the chronic diarrhea, malabsorption problems. And included in that would be children who, along with the gastrointestinal problems, could be shown to have this nodular hyperplasia, which is a finding that Wakefield in England, acknowledged almost 10 years ago, recorded others, Uhlman, other people recorded. So the lymphonodular hyperplasia, in conjunction with the gastrointestinal problems.

And then a third, which is not necessarily present in the profile, but often is, is immunological disturbances. I say immunological because children that have this profile of regression and gastrointestinal problems have gastrointestinal problems that have been linked to immunological deficits.

THE COURT: What immunological deficits would you be looking for that would fit this profile?

THE WITNESS: I think the best would be, for instance, finding autoantibodies. Autoantibodies are antibodies where the immune system secretes antibodies, that instead of being directed against

viruses and bacteria, are directed against certain tissues, including brain cells.

Yates did have an immunological workup that was normal, at least the things that were tested. But I didn't see any mention of testing for antibodies to the brain, for example, which would show a, what we call skewing of TH1 to TH2, meaning going from a cellular immunity to one that's based on antibodies. So basically, these children may have a higher susceptibility to developing autoimmune problems.

THE COURT: Do you believe that, what in Yates's record supports your view about his immunological disturbance?

THE WITNESS: Yes. What makes me think that is that he was sickly. I believe that the infections that Yates had, his recurrent, not just, you know, viral infections, but also yeast infections, and his need for chronic antifungals. I feel that all of these things suggest that he was sickly, and was not just the average intermittent illness, intermittent infection, that type of child. He was a sickly child. So that suggests that his immune system was impaired.

Now, a lot of people, when they talk of immunological disturbances, a lot of physicians think of immunodeficiency the same way you would think of

AIDS or someone who's on chemotherapy. These people are severely depressed and show certain signs with their labs.

However, someone who is sick all the time, you can infer that there's something with their immune system; their immune system is impaired. The same way that I would say, you know, Yates's MRI of the brain was completely normal, but we know he has a neurological problem. He's lost the ability to speak the way he was talking before. He's not interacting well. So we know he has a brain problem, although I can't prove it with the MRI.

So I think that the fact that he was sickly, sick all the time, means -- and even the lymphadenopathy that several members have pointed out. Lymphadenopathy is just swollen lymph nodes that it's usually a reactive sign that there's an infection going on.

If the lymph nodes are swollen all the time or for a long period of time, it means that the body is still reacting to viruses. In fact, it would actually make me think of a persistence of some type of virus. And of course, we talked of persistence of measles virus.

If I may just elaborate a little bit

further. Once someone is normally vaccinated with the MMR, pediatricians tell parents that you may, after a few weeks, start experiencing some symptoms. Rash, you might experience flulike symptoms. When they say this, they're really saying that the measles virus may kick in a little bit, not too much, you don't actually want to create the illness, but just elicit an immune response. That tells a body that the body will not have memory for the virus, and then the clinical symptoms go away.

And later on, maybe two months later, you may have a few other symptoms referable to the mumps, rubella, et cetera.

But what happens if you have these symptoms in a persistent manner? Chronic lymphadenopathy, chronic not feeling well for a variety of processes. The parents talked about feeling warm. You know, a lot of times pediatricians would ask for the actual temperature. You know, was there a thermometer that was used.

So we don't know what the temperature was, but you know, let's say Yates felt warm intermittently. I would say that may not necessarily be significant, you know. But if he feels warm all of the time, even in the absence of the natural

measurement, although I couldn't prove it, but I would suspect that, along with the chronic lymphadonitis and all of the other symptoms, that there was something going on with his immune system.

THE COURT: Do you think that if, in your opinion, absent his autism diagnosis, with the number, Yates did have a number of ear infections and ultimately ended up with ear tubes. So there is evidence that he was reacting to something, true.

Absent a diagnosis of autism, would it strike you that the number of his ear infections and illnesses and that sort of thing, you would still characterize this as someone who was sickly? This is out of the ordinary for small children?

THE WITNESS: Well, I see a lot of children with neurological problems that I can see different children that are occasionally sick, and other children that are sickly; that is, they're sick all the time.

You know, I refuse to believe that children that are as sick as Yates was was just the norm. I do, however, accept the fact that it is normal, or it is acceptable, I should put it that way, to have a few infections when you're young, especially if you're exposed.

But if you keep getting sick recurrently, to the point of being on multiple, not just antibiotics, but multiple antifungals, then I must conclude that the immune system is impaired. Not necessarily immunodeficient. Most physicians, when we say immunodeficient, you're thinking of severe abnormalities that you can document with labs, and like I say, conditions like HIV, chemotherapy, certain immunological disorders. But I'm arguing for immunological impairment of some sort.

THE COURT: Let me ask you, as well, Dr. Buie has submitted a letter and found colitis, an allergic colitis, in Yates. Yates apparently does have some food allergies that have been established.

Absent any finding, because there hasn't been the presence of MMR that has been at least established by tests, that has been found in Yates's gut, do you think that it's sufficient for his food allergies to have established the colitis that was present for him? And if it were just attributable to food allergies, would that change your opinion about the MMR persistence?

THE WITNESS: If he did not have the clinical signs that I saw, the recurrent vomiting -- or the recurrent, sorry, diarrhea; if it was just

that, with none of the clinical symptoms; I would yes, say that that's a possibility. I would say that the colitis, the allergy component or the eosinophilic could be due to food allergies.

But putting it together with the clinical profile of recurrent diarrhea, in fact if you look at his gastrointestinal symptoms, they almost seem out of proportion to the gut findings, which is not necessarily unusual with medicine. Sometimes you will see a lot of clinical symptoms, and then you may not see a lot of, or a significant -- or I shouldn't say significant -- a severe finding to match that.

So I think in order to answer that question, I'd have to look at the context. If he did not have any bloating, any diarrhea, any protuberant belly, and just that, I would say yes, there's a good possibility that the food allergy could do that.

THE COURT: One more question. You referenced a patient of yours who has developed tics following a vaccination, that were ameliorated or eliminated after he went through the mercury detoxification program. Did his behavioral problems improve at that point?

THE WITNESS: Yes, yes, they did. In fact, if I may explain this a little bit further. There has

been a lot of talk about chelating children with autism that were shown -- who have evidence of mercury toxicity.

As a neurologist, I was not trained to think of chelation that way. I had chelated kids with, who are in the ICU with lead toxicity, or in a coma, for example. I had chelated children like this. But that was the first case I saw of a child who was not that sick, in terms of, you know, coma, seizures, but just developed significant tics postvaccine.

That was the very first time I chelated someone, because I try and find evidence, laboratory-wise, that a child did have mercury postchelation. If I give a small dose, I was able to collect some mercury in the urine.

And so I used that, if you will, as a test case, which was very successful. Not only did the tics stop, but so did the ADHD symptoms and other conditions that the child had. Which gave me the impetus to consider chelation in other patients, including with autism.

THE COURT: Was that the most pronounced behavioral problem? The attention deficit?

THE WITNESS: The attention. He was aggressive, too. Intermittently he was aggressive,

but he was, yes, very hyperactive. And the tics were interfering with his daily routine.

THE COURT: At what age do you think normal development needs to occur? And then there's the taper-off before it can be termed regressive.

THE WITNESS: Regressive. Well, the regressive, the regression in a general sense, in a neurological sense, the regression may occur almost at any age.

For example, patients with Retts syndrome, which is another condition that presents with autistic features, the regression in that particular condition can start anywhere from six months to 18 months, on average. So --

THE COURT: I'm talking absent any of those; just with the profile that you were talking about.

THE WITNESS: Oh, with my profile, yes.

THE COURT: Your profile.

THE WITNESS: Yes. What I have seen with others and with personal experience, I would say the regression is often seen somewhere between the 12th month to 24-month period. And I really can't pinpoint very specifically where, you know, in that group, but usually children that have regressive autism can regress typically between 12 months to a couple of

years. But there are exceptions. There are some kids that do not regress until after three years of age.

THE COURT: Thank you. 10:50?

MS. RENZI: Thank you. That will be great.

THE COURT: We will recess until 10:50.

(Whereupon, a short recess was taken.)

THE CLERK: All rise.

THE COURT: Please be seated. We are back on the record. Respondent's counsel?

MS. RENZI: Thank you. Good morning, Dr. Corbier.

THE WITNESS: Good morning.

MS. RENZI: My name is Linda Renzi, and I represent Respondent in this case.

CROSS-EXAMINATION

BY MS. RENZI:

Q Dr. Corbier, throughout your report you used the term "biologically plausible." Do you recall that?

A Yes.

Q Could you please define "biologically plausible?"

A By biologically plausible, what I mean is if someone is going to make a hypothesis, it's always helpful to have an example that makes sense

biologically.

For example, when we talk of MMR, any possible complications of MMR, it helps to have a model that exists that's undisputed, a model that is well accepted. It's well accepted, for example, that people who have measles infection can go on to have either acute, subacute, or the SSPV encephalitis. So that would be an example of a biological model that explains what a postinfectious process is.

So that's what I mean by -- or biologically plausible would be something that fits with a pre-existing understanding of a condition.

Q So biologically plausible then can be used within a hypothesis. It doesn't have to be proven.

A What's that?

Q To be biologically plausible, we can still be talking about a hypothesis; it does not have to be proven.

A Well, a biologically plausible factor or point is a starting point. So I'm suggesting that you go from something that's biologically plausible, and then you can confirm that through research and studies.

Q So it just means possible. It means possible.

A Possible, yes. Well, it means what it means -- yes, it means something can exist in the way that you're seeing, based on what is known.

Q To be biologically plausible, does there have to be reliable scientific studies to support the hypothesis?

A It, yes, you should have at least if not studies, you should have some type of elucidation of the mechanism. For example, if someone makes an observation, even if studies are not yet done, biological plausibility with someone who explains, someone who, a knowledgeable person who explains a mechanism that's observed, would qualify for biological plausibility, even if several studies are not done yet.

Q I know in your testimony earlier you talked about Yates's profile. But could you - your report also contains a number of hypotheses as to the cause of autism. Could you explain in detail what you think Yates's vaccinations did to cause his autism?

A What I see in Yates's case as far as profile, and as far as the vaccines -- and we'll just pick MMR for this discussion -- is that we see a child who fits under the category of a child with regressive autism. Normal development, he receives a set of

vaccinations, and within a couple of months or so,

give or take a few days, he starts to regress. Then we have a child who has a lot of gastrointestinal problems.

That is a set of conditions that have been found with many, many, many other children in a very similar fashion. And putting Yates in that profile is very important because, as I mentioned earlier, autism is not one disorder. There are so many causes and contributing factors for autism. Some, for example, if I saw a female who was degenerating and whose head size was small, who was losing ability to use the hands, but also had autistic symptom, I would think ah-ha, this was Rett syndrome. Or if I saw a child who had autistic characteristics, but had a lot of seizures and had some abnormal patches on his skin, I would say ah-ha, this most likely is tuberous sclerosis.

So basically, the profile that I talk about is basically a set of symptoms that collectively fit a pattern that's reproducible; a pattern that has been reported widely, and whose laboratory investigations fit also within that profile.

Q But do you know how the vaccines cause the autism? He fits a profile, but do you know what role exactly the vaccines played in that profile?

A The role that I believe the vaccines play, based on the research that has been done, is that children with MMR who regress tend to have changes in the gut: the lymphonodular hyperplasia, which several researchers have found measles, persistence of measles virus in the gut. Studies in these similar groups, or some reports have also shown viruses in the brain. So the way that I believe that the autism was caused by the MMR is the vaccine impaired his immune system.

Q I'm sorry, which vaccine?

A The measles. The measles. It probably also either went to the brain potentially, or he developed a postimmune reaction, where the antibodies themselves went to the brain. And one of the two caused the regression, leading to the autism, along with the gastrointestinal symptoms.

Q You don't know which one? Whether it was the antibody or --

A I don't know which one because we, I didn't do a brain biopsy, so I can't say for sure.

Q Have you read the reports in the Cedillo hearing that were submitted in the testimony of the Cedillo hearing of Dr. Griffin, the virologist?

A I've read several, but I don't know if I read that one in particular.

Q Have you read Dr. Ward's?

A I may have, but I don't remember. I looked at several of the reports, but I don't remember the names very well.

Q I'll ask you just a couple more, if you're familiar with them. Dr. Bustin?

A I've heard of his report, yes.

Q Dr. Brent?

A I read some of his work from previous situations.

Q And did you read the report of Dr. Michael Gershon?

A The name is familiar. I think I did, but I don't recall the details.

Q Doctor, you've been a practicing neurologist for five years?

A Seven years.

Q Seven years. And how long have you practiced in the Charlotte, North Carolina area?

A I started practicing February 13 of this year, 2007.

Q And in your seven years, how many autistic children have you treated?

A I haven't counted specifically, but it's probably several hundred patients.

Q How are your patients referred to you?

A A variety of ways. Either through their pediatrician; a lot of patients have come through therapists, psychologists, or sometimes a patient will have an outside evaluation, say, by a neuropsychologist, and then that therapist or that specialist will refer to me. So various specialists. And some patients will come on their own. They've heard of me through, you know, different means, and then will come.

Q Have you heard of Defeat Autism Now Organization?

A Yes, I have.

Q Do you get referrals from, and I'll call it DAN, do you get referrals from DAN?

A I have in the past, yes.

Q Do you know what percentage of your practice are DAN referrals?

A I'm not sure what percentage. It's a little hard to say, because some parents, when they're researching a physician, they go online. And they, let's say it's a patient who's interested in looking at biomedical things, they may come across DAN on the internet, and then come that way. You know, they may see a name of a DAN practitioner, and then may come.

So the referral could be made that way, so to speak.

THE COURT: Pardon me, counsel. I'm sorry. It's been drawn to my attention that we have to speak a little louder into the mic, that we're all apparently soft-spoken. So if we could try to speak up a little bit, that would be great.

MS. RENZI: I will.

THE COURT: Thank you.

THE WITNESS: So have I answered your question? What I'm trying to say is there are some parents that do their own evaluation. For example, they say we want to find a particular type of doctor, so they go on the internet or they talk to people, and then my name comes up. And then they come that way.

BY MS. RENZI:

Q So if they go on the DAN web site, they would find your name?

A My name is not there right now, no. It was there up until, when I left, it was there up until the time that I came here.

Q You left, you're talking about --

A Up until I left Alabama to come to North Carolina.

Q Of the children you treat with autism, how many have been diagnosed for the regressive autism?

Q I don't know the number, I haven't kept track of the number. But my guess would be a third or so. And the number could be higher, but the third would be those that probably truly have -- there are some kids that superficially may present with regressive autism, but when you look at the history you see that something else was going on. Maybe they had seizures, weakness, fussiness, or some other problem, so I don't count those.

Q Of those children that you believe have true regressive autism, how many of those do you believe developed regressive autism as a result of vaccinations? Either MMR or thimerosal, or both.

A Several. I haven't counted, but I do, in terms of profiling, I hear very similar stories over and over with certain types of patients. And all of those would be representative of what we see with Yates.

Q Would you say more than 50 percent?

A Fifty percent of the regressive autism?

Q Yes.

A Maybe, but I'm not sure. I've really not kept track in that way.

Q And of those numbers, how many do you believe developed regressive autism just as a result

of the MMR vaccine?

A Not very many. Let me elaborate. The reason is, I prefer to say contributed, because there are so many factors that I think play a role.

Other vaccines, for instance. Thimerosal, for instance. Other environmental factors, some of which we still have yet to identify. So I wouldn't say just, in most cases.

Q Doctor, you stated that you were on the DAN web site while you were practicing in Montgomery, Alabama.

A Yes.

Q But you are no longer on it now?

A Yes.

Q Why is that?

A I've attended I think at least two or so, or more, DAN meetings. And their requirement in the past was to attend at least a meeting, and to demonstrate a willingness to follow some of the principles of DAN, which I did. So I was listed as DAN.

And then I think recently they came up with a rule, they sent a letter to everyone, that in order to remain on the list, you have to attend a DAN meeting I think at least once a year. And by attending the yearly DAN, that shows that you're

still, you know, following some of the treatments and recommendations.

I've not been able to attend the past, oh, I don't know, two or three years, because I attend a lot of conferences. Although a lot of my practice is devoted to autism, I attend a lot of conferences for epilepsy, spasticity, cerebral palsy, so a lot of conferences. So I've had to balance those, so I've not been able to attend regularly.

Q Although you no longer attend the DAN meetings, do you still subscribe to the DAN protocol that is required?

A I don't look at DAN as a rigid protocol, but I look at DAN as a movement. I've looked at a lot of the different therapies with DAN, and if I might answer your question by stating that the way I got involved with DAN is because before I ever heard of DAN, I was very involved in nutritional therapies myself, and then heard of DAN and thought, ah-ha, here's a group of physicians that subscribe to biochemical and nutritional things. So I joined DAN.

I think the DAN approach is very diverse. There are a lot of principles. For example, I believe that the gut and the brain immune system are related.

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So there are a lot of things in DAN that I still adhere to.

I don't adhere to everything necessarily. I don't follow a rigid, if a patient comes in do this, do that first, necessarily. So I use a lot of the principles because I think that they're justifiable, yes.

Q When did you learn that Yates began to regress at 12 months of age?

A The first I saw Yates initially, September of 2002, as far as I recall. He had been diagnosed at Vanderbilt I think in July, and my initial history was obtained at that time.

Q And what does that initial history say? Do you know? As to the timing of the regression.

A I think -- I mean, I don't have it in front of me, but I think I might have reported 18 months, just like I did with this last case.

Q So when did you learn, then, that the regression began at 12 months of age?

A I think I probably learned about it, I don't recall exactly when. But I can tell you that over the course of several visits, we refined the history and findings. And I must say that the biggest refinement was yesterday; I got the clearest picture I've ever

had

with Yates listening to the different family members, looking at the videos. I did review some videos beforehand, but you know, everything was put in a very clear manner today, or yesterday.

Q But your report which you filed in July, the premise is that you have regression started after the vaccination to 12 months of age, correct?

A That's what I put in my report, that's correct.

THE COURT: For the record, the report to which you're referring is Petitioner's Trial Exhibit No. 1?

MS. RENZI: No, it's Dr. Corbier's report, which is --

THE COURT: His expert report?

MS. RENZI: His expert report, I'm sorry.

THE COURT: Okay.

THE WITNESS: I actually, if I may correct, here's what I wrote on the July 2 report. I wrote, "He developed normally up until the age of 18 months to 24 months." But I then wrote, "Just prior to that, he started to have regression."

So the just prior I admit is not very clear. But you know, this is saying that it actually occurred before, and I'm not very specific as to before. But

like I say, yesterday I was able to, you know, put the whole picture together and understand.

BY MS. RENZI:

Q But your expert report, which is dated, was filed in July, Petitioner's Exhibit 26 -- the basis for your expert report is that regression began at 12 months, is that correct?

A Well, I think I put in my report, I believe I put 12 to 18 months interval. I don't think that it's easy to just give a specific, you know, time when the regression -- I put it in an interval in my report, I believe. If I can refer to that.

Q You also state, Doctor, in your report that timing is paramount to your diagnosis, is that correct?

A Yes.

Q And the timing from your testimony today, the paramount timing is one to nine months following vaccination?

A Yes. Putting all of the information that I have found in articles -- we talked about biological possibility -- putting all of that information together, and based on known reports of post-MMR encephalitis, I would put it in the one- to nine-month period.

Q It seems like a broad range to be a paramount consideration, would you agree?

A Sure, it's a broad range. A lot of things tend to be very broad.

Q What vaccinations did Yates receive at 12 months?

A He had the MMR. Let's see.

Q You're looking this up? What do you have in front of you, Doctor?

A I just have my report.

Q You have Exhibit 26, your expert report?

A Yes.

Q Okay.

A I have noted here that he had had the Hib, Hep B, MMR and Prevnar.

THE COURT: You're referring to page 5?

THE WITNESS: Oh, I'm sorry, page 5, yes.

BY MS. RENZI:

Q Which of those vaccines contained thimerosal?

A I believe he had the hepatitis B.

Q That's the only vaccine that contained thimerosal?

A That I know of.

Q That you know of.

A Right.

Q Do you know how much thimerosal is contained in the hep-B vaccine?

A Not off the top of my head, no.

Q Is there, your timeframe is one to nine months, correct? For you to determine that there's a causal vaccine relation, is that correct, one to nine months?

A Yes. And not only vaccine, but also the live measle virus, as far as an acute or a subacute encephalitis.

Q What is the timeframe, absent the MMR, for thimerosal-containing vaccine, following a thimerosal-containing vaccine, for regression to begin that you would attribute the regression to the thimerosal-containing vaccines?

A I'm not sure. I'm not sure. I don't have a good paradigm for that one. And the studies that I have seen have not really addressed that question, so I cannot address that scientifically.

Q So you don't know.

A I don't know.

Q If Yates's regression had occurred prior to 12 months, prior to the receipt of his MMR vaccine, would you have attributed his regression to his six-

month vaccinations?

A The lab indicated some problems with thimerosal, so I would have, you know, included, as I do even now, the thimerosal as playing a role.

Is that your question? I'm sorry.

Q No. I'm saying absent the MMR vaccine, would you -- I'm sorry. What I said is, had he started to regress prior to his 12-month vaccination, would you have attributed the regression to either his six- or nine-month thimerosal-containing series of vaccinations?

A I would, what I would be able to say is that the thimerosal in the vaccines up to that point potentially played a role, contributed. That's all I would be able to say with any certainty.

Q Would that certainty, would -- what weight would you give to the vaccine's playing a role in the autism? In the onset of autism or the development of autism.

A Are you talking which --

Q What percentage?

A I'm sorry, which vaccine? I don't understand your question --

Q The vaccines at six and nine months.

A Okay.

Q You say could potentially cause the autism.

A There's no way for me to give a percentage, because that's not been studied scientifically.

Q So you can't say more likely than not.

A I could say more likely than not that that was a contributing factor. Yes, that I can say.

Q So greater than 50 percent, you can address, you can attribute a percentage to it.

A That that played a role, yes.

Q What is the basis for that opinion?

A Well, the basis is I was very struck by the fact that at six months, or after his six-months shots, he had a screaming episode which I think, to the best of my ability, was probably linked to the pertussis. He screamed unconsolably for several hours, according to the testimony we heard yesterday.

For a neurologist or a pediatrician, if a patient screams unconsolably, we think of irritability. We think that there's something wrong. And so the only thing that I could link, based on the history, to that screaming episode would have been the pertussis. And therefore, I think that that, you know, played a role. That's an indicator, in other words, that the vaccine played a role; that he had some type of reaction.

Q And is the six-month reaction reflected in

the medical records? Or is that from the testimony you heard yesterday?

A I've heard it before, as well. I've heard it in greater clarity yesterday, in terms of the duration. But in fact, I think that might be in my report here, so I must have heard it before.

Q Is it, to your knowledge, reflected in the contemporaneous medical records? A reaction to the six-month vaccination.

A Are you referring to the pediatricians?

Q Yes.

A I don't recall, but I can tell you that I reviewed the pediatrician's record and didn't feel that it was very thorough. So it wouldn't be too surprising if that was not included.

Q Did Yates exhibit signs of an encephalopathy due to his pertussis vaccine?

A The screaming for several hours in an unconsolable way is what many people would call an encephalopathy, albeit temporary, but an encephalopathy. In other words, he was in an encephalopathic state. He was crying; he could not be consoled. So likely, if you try to, you know, look at Yates, he probably would not, you know, interact in any meaningful way, so we could use the term

"encephalopathy" in that state, in that condition.

Q And how does pertussis cause an encephalopathic state? Because there were no sequelae of that encephalopathy. He didn't regress at six months, is that correct?

A I don't think he had encephalitis. I think he had encephalopathy. If you have an encephalitis, that can cause damage to neurons, neuronal lots, and that can lead to sequelae. For example, if you have a herpes encephalitis or any type of encephalitis, you have death of brain cells. An encephalopathic state can occur with a variety of things. Certain drugs can make you encephalopathic, head trauma can make you encephalopathic temporarily, and you can recover.

So encephalopathy, all it means is a diseased state of the brain. And it could be due to any number of causes. It can last any number of time, or any length of time.

Q Why is it your opinion that it was caused by the pertussis?

A I just don't have a better explanation. And I know, based on the CDC, that crying for several hours is one of the things that should be instructed to parents to look out for. That, along with other manifestations.

So based on that, I think that that's likely what happened. I can't prove it, but that's likely.

Q And you don't know how pertussis would do that, do you? Or do you?

A Do I know how pertussis would cause the encephalopathy?

Q Yes.

A I know how regular pertussis would. Regular pertussis -- and I'm not talking about the acellular pertussis, but the whole-cell pertussis -- has three neurotoxins that can affect neurons in a very adverse way, and can cause a lot of neurological symptoms.

The way the acellular pertussis can cause a child to scream, I don't know that that's been elucidated, but it is something that pediatricians are instructed to warn parents about.

Q If we accept that Yates had a reaction at six months of age, how critical is this event to your opinion that Yates's autism was caused by his later vaccinations?

A To me, his reaction, or I should say possible reaction to be precise, to what I think was the pertussis, just tells me that he may have some type of underlying predisposition that caused him to scream for several hours. After all, not every child

who receives a pertussis vaccine would scream. But I don't think that that -- I think that's one among several factors. And that's why I always like to use the term "contributing factors." I think that could have played a role, thimerosal could have played a role, and the other vaccines could have played a role. But it just adds to the notion that he likely had a reaction. He was prone or susceptible to reactions.

Q On page 4 of your expert report you indicate that you performed a 24-hour EEG on Yates?

A Yes.

Q How is a 24-hour EEG taken? What is your role in administering that EEG?

A My role in obtaining that, maybe I should just, for clarity, kind of explain. First of all, an EEG is electroencephalogram. It's a brain wave test that allows us to determine several things. It allows us to determine whether a child is likely having seizures. It tells us what the status of the brain cells are. It actually looks at the cortex of the brain.

The reason why sometimes we'll do a 24-hour EEG is that a routine EEG, which can last 30 minutes, or even a little longer EEG that lasts one hour, can miss certain things going on in the brain. It's not

unusual for a patient who has a lot of seizures to have a normal routine EEG, because the abnormal signals in the brain, the abnormal electrical activity occurs periodically. So it's a snapshot picture in time. So the 24-hour EEG just enhances our chances of finding abnormalities that you might miss on a routine EEG.

Moreover, when I do a 24-hour EEG on a patient with autism, I'm looking for some specific problems that can present as autism that I'd like to rule out, such as static epilepticus, that show in sleep. That is a condition that may not show on routine EEGs, but when you enter deep stages of sleep, the EEG becomes continually abnormal in the parietal occipital areas.

If I saw such a patient, whether Yates or someone else, that would lead me to a very specific direction. When we talk about profiles -- in EEG, that indicates a particular seizure syndrome. Let me treat the seizure and see if I can get the child to speak or improve, based on that diagnosis.

Q And what did you read on this EEG?

A I found that he had intermittent bifrontal, central, and generalized epileptiform discharges.

Q And was this EEG ever read by a neurologist

who is board-certified in clinical neurophysiology?

A No, but I don't think that's necessary, because I'm a board-certified child neurologist. And I've trained with some of the best epileptologists. So it's not necessary for the EEG to be reviewed.

Now, if I do have a question, I'll never hesitate to confer with a colleague or an epileptologist. If I'm not sure, if I think that there's a questionable finding, then usually I'll be the first one to call or send a copy of the EEG for review. But if I'm confident with my findings, then there's usually no need to have an epileptologist confirm the EEG. That's part of our training.

Q So if you had -- what epileptologist have you studied with? And where?

A I studied with Dr. Prevetara at the University of Cincinnati, when I did my year of adult neurology. And Dr. Tracy Glaucer, who is one of the most renowned pediatric epileptologists in the country, perhaps the world. And he was at the Children's Hospital of Cincinnati, where I did my fellowship training.

Q What is an epileptologist?

A An epileptologist is a neurologist that does further training in the field of epilepsy, whether

epilepsy surgery, whether epilepsy treatment, or a variety of aspects of epilepsy. So an epileptologist has completed their neurology training, and then they do a year or two of extra training in epilepsy-related things, treatment.

Q Are you an epileptologist?

A No, I'm not an epileptologist. I'm a general child neurologist.

Q Has Yates ever been diagnosed with clinical seizures?

A There was a suspicion, and I can't remember if it was at nine months -- let me see. There was a, he had an episode of staring at one point, and was seen by a neurologist in Tennessee. Let me see if I can -- he saw Dr. Mark Bruggerman. This was on May 13, 2002. I'm looking at my, on page 3, under the neurological profile.

And I don't recall seeing what his final impression was, but his exact words were, let me see. He wrote, let's see, he wrote several, he basically was staring off for several minutes, and "still not back to himself." He did an EEG at that time that was unremarkable.

I don't recall what the report says, but most neurologists, when they read an EEG, will put, as

a disclaimer, "a normal EEG does not rule out seizures."

To me, that episode looks like a seizure. The only way to truly confirm that that episode was a seizure would be to obtain an EEG while he's staring, which most of the time we don't have that luxury. So you have to go by clinical findings, and then your level of suspicion based on your expertise.

Q But was there a diagnosis of clinical seizures? There was a possibility of seizures. Was there a diagnosis?

A You mean by Dr. Bruggerman?

Q Yes.

A I don't recall seeing it. I didn't write that in my report. I don't remember what he put, what his diagnosis was.

Q Where are you currently employed, Dr. Corbier?

A I'm employed in Concord, North Carolina, at Concord, North Carolina, CMC Medical Center. CMC Northeast Medical Center.

Q Is that a hospital?

A It's a hospital, multispecialty group practice. I think we are probably 200 physicians and multiple pediatric subspecialists in this group.

Q Do you have hospital privileges in the

Charlotte area?

A Yes, I do.

Q What hospital?

A It's called CMC Northeast Medical Center.

Q And did you have hospital privileges when you were practicing in Montgomery, Alabama?

A Yes. Since I started practicing up until now, I've always had hospital privileges.

Q Why did you leave Montgomery?

A I left Montgomery for several reasons. When I was doing my training in Cincinnati, I was approached by the medical director, who asked me to join the faculty to do research in pediatric stroke. I was going to do it, but I decided instead to go into private practice.

I'd been in private practice for six years in Montgomery, and worked well, enjoyed the clinical setting. But I was kind of missing the intellectual side of things, in terms of the possibility of doing research, possibility of doing medical education, teaching residents and others. In Montgomery I did not do a lot of these things, and wanted to kind of change that a little bit.

The other factor is that I was in solo practice. And if you know anything about solo

practice, it means long hours, working all of the time. My wife, who is a pediatrician, was helping me run the office. We have a 10-year-old son that we were both trying to see patients, work all the time, and home-school in the office. So that became a little stressful.

So I decided to seek employment somewhere where I could be employed, and not have to have that amount of stress, and still continue or pursue my interests in academia, teaching, and other areas. So that's why I left.

Q But you say 100 percent of your time is devoted to your clinical practice, correct, at this point?

A At this time, it is. There's a very large research campus that's being built not too far from my hospital. And one of the reasons I chose Northeast is to hopefully have the opportunity to be involved in research.

I also chose this particular hospital because it's associated with Duke. There are a lot of collaborative things that we would like to be able to do in the future.

Q What is Mannatech?

A Mannatech, there is a company out of Texas

called Mannatech. Mannatech is a company that produces a type of nutrient called glyconutrient. The flagship product is Ambrotose.

Ambrotose is a product that contains eight sugars, including cylose, manose, fucose, neuraminic acid. And the company basically has found a way to patent these eight sugars into a particular product. They've gone on to market other nutraceuticals, all of which contain these eight sugars: multivitamins, digestive enzymes. So that's what Mannatech is.

Q And you prescribe it to your patients?

A I'm what I call myself, I call myself a nutritional neurologist, which means that a lot of my practice, almost from day one up until the present, I spend a lot of time talking about nutrition, as I believe that dietary interventions are important.

I became interested in Mannatech based on prior interest in a specific neurometabolic disorder called congenital disorder glycosylation (phonetic).

When I was in Cincinnati, I took care of a young child who was about eight or nine, and kept having recurrent strokes. I mentioned earlier that I was going to join the faculty to do pediatric stroke, and here I was. I saw a little girl having strokes for no apparent reason, and multiple other problems.

We were able to diagnose her with a specific problem with glycosylation, which means that she, herself, had a substance called glycoprotein, which are proteins with a sugar moiety And her body, for some reason she was lacking a particular enzyme. She was not able to make one of the sugars, called neuraminic acid.

I became very interested in that disorder. At the time there were about 50 cases described in the country; I took care of three of them, and interacted with several kids throughout the world with this condition.

I've given several conferences on this particular disorder. And my conclusion or thesis, especially to neurology conferences, was that this problem was probably underdiagnosed. I felt that many individuals might have a glycosylation disorder.

Getting to your question. Well, I called Mark Patterson, who is a child neurologist at Mayo Clinic. I did part of my training at the Mayo Clinic. And he was having success treating some patients with this disorder with a particular subtype called congenital disorders of glycosylation type 1-B. This is a type where children present with a lot of gastrointestinal problems. They are bloated, they

have massive diarrhea, they have blood in the stools.

So I said to Dr. Patterson, you know, have you thought of using this manose, which is a sugar that is implicated in the immune system, with other disorders? In particular, other congenital disorders of glycosylation. He said he had tried; it was not effective.

I thought of the glycosylation disorders for a long time. And when I became involved with autism, I thought that maybe a subset of children, perhaps a large subset, might have problems with glycosylation.

People in Montgomery and in the area knew that I was involved with nutrition. So there was a lady that came to me and said I see that you're involved with nutrition; have you heard of glyconutrients. I told her no, I've never heard of that. In other words, I'd never heard of the product that she presented.

But I told her, I said, I usually do -- I'll listen when people come to me and they present new ideas. I try to be open-minded. So she explained to me about the eight sugars.

I said, ah-ha, I know about manose and these other sugars. And she says you know, people are doing //

quite well with this. I said well, let me see the research. And it dawned on me that this was something I could add to the list of things that I recommended beyond vitamins, minerals, the essential fatty acids.

But I told her before I'd put anyone on this, I'd need to do research. I spent several weeks, if not months, researching everything I could on this particular supplement, and then decided to go to Texas, where Mannatech Company is located.

I spoke to leading physicians, researchers, pathologists, neurosurgeons, a lot of people who were and biochemists involved with this; and made up my mind that this is something that I needed to recommend to my patients, along with other things that I recommend.

So I started to recommend Mannatech. And I must say that a subset of children who have taken the glyconutrients have done quite well. Not everyone, but I've found no therapy unit that works for everyone, but several people did respond to treatment with glyconutrients in terms of their symptoms.

Q What research did you review?

A For what?

Q To conclude these substances would work?

A I reviewed a lot of studies by, there is a

biochemist -- I'm blanking on his name. He is the

first one to look at the role of manose. I looked at research from a pathologist; his name is Dr. Reg McDaniels, O'Malcheck and several others who had written on the use of glyconutrients.

I also reviewed, in looking at how this might work, I reviewed work from Dr. Hudson Freeze, who is in La Jolla, California. I had called Dr. Hudson Freeze because when I was researching congenital disorders of glycosylation, he had collaborated with me and sent me some of his slides for some of my presentations. So I looked up some of his work, as well, in my research of the glyconutrients and how they might work in outpatients.

Q You've given presentations. Have you ever published on this?

A On what?

Q Glycosylation.

A I'm not published, but I've given several grant rounds to neurologists and pediatricians and medical individuals. Probably the reason I'm not published on this, though I would like to, is that when I moved from Cincinnati to Montgomery, my emphasis gradually shifted from congenital disorders of glycosylation to autism. So I kind of not necessarily lost interest, but most of my focus and

energy was diverted to autism.

Q Did any of the studies you looked at look at the glycosylation in autism?

A Did any -- I did not see any direct studies, but I did see several things that might implicate glycosylation to autism. Such as, there have been several studies with the glyconutrients that have looked at gastrointestinal problems. As I mentioned, children with congenital disorders of glycosylation, especially type 1-B, have a lot of gastrointestinal difficulties, which I thought was applicable to autism.

Also, some of the most robust studies have been done with the immune system, with glyconutrients for other immune disorders, such as lupus, HIV, and other conditions, knowing what I know, say from the studies with Dr. Ashwood and others who have looked at the immune system and Dr. Zimmerman and others in autism, I thought that this would be a very good fit, in terms of applicability. But I didn't see any study, in fact that's one thing I was starting to work on is I was starting to maybe develop some informal studies that I might pursue down the road.

Q Is Mannatech FDA-approved for the treatment of autism?

A It's listed -- oh, for autism?

Q Yes.

A Unfortunately, the only thing that's FDA

approved for autism is the drug Risperdol, and that, FDA approval just came one year ago. So in essence, all of the physicians, myself included, we were using unproven therapy as far as Risperdol. But it did finally get FDA-approved.

So no other drug that is used in autism, whether it's a stimulant or SSRI, nothing has at all been FDA-approved for autism. In fact, most drugs are not even FDA-approved for kids. Most of the drugs we use, we know they work, we know that they have a safety record for other conditions. So many pediatricians, neurologists, doctors feel safe using them in kids.

A good example is epilepsy. Very few drugs for epilepsy are approved in the very young children. So if we have a one-month-old who does not respond to, say, phenobarbital, instead of letting a child have a seizure until they die, we'll use a drug that we think is safe, although there is no approval. So to answer your question, since most drugs are not approved by FDA, it's even harder for a supplement to get approval from the FDA.

THE COURT: Pardon me. Dr. Corbier, please help me to make sure I'm oriented. You referred to SSRI?

THE WITNESS: Oh, yes. That's selective

serotonin reuptake inhibitors. These are antidepressants. They're used a lot in some children with autism, because some children with autism have mood disorders.

In fact, talking about profiling, I'm a big believer in profiling. There's a group of children with autism who, they look very depressed. And if you treat their depression properly, often their autism symptoms will disappear. We call that pseudoautism. A very important disorder. Because if you can identify that a child's autistic symptoms are actually due to some neurochemical defects that really cause a lot of emotional symptoms, mood problems, depression, and you treat it correctly, that child with that specific antidepressant treatment will resolve the autistic symptoms. So that's what an SSRI is.

THE COURT: Thank you.

THE WITNESS: You're welcome.

BY MS. RENZI:

Q What is Mannatech FDA approved for?

A What's that?

Q What is Mannatech FDA approved for?

A Well, I don't know that it's FDA-approved, but it's then looked at. It's received a patent, and it's in the PDR, Physician Desk Reference. So if you

look at the PDR, you'll see Ambrotose.

I don't remember the specific indications, but all of the attributes of Ambrotose are listed, like other things that are listed in the Physician Desk Reference.

Q I want to move on to page 6 of your expert report, which you call the pertinent factors in Yates's case.

A Yes.

Q And I want to look specifically at the first five bullets.

A Yes.

Q Do you rely on all of these bullets in combination for your conclusion that Yates's autism, the development of Yates's autism was vaccine-related?

A You're saying do I rely on all of them collectively?

Q Yes.

A Yes. Each one I think is important, but each additional bullet point adds further credence or further support to that belief.

Q I'd like to go through these, then, one at a time. And if you could tell me, I'll ask some questions following up.

How important is it, in your opinion, that

Yates was normal prior to the development of his autism?

A In terms of the MMR vaccine?

Q Yes.

A It's only important based on the timing. In other words, if the regression had occurred at 11 months, obviously that would not be of any significance. But the fact that there was regression noted some time after 12 months, I think is the starting point.

We have an event or an intervention in this case, a set of vaccines. Subsequently we have regression. So the starting point is to ask myself, is it possible that the vaccines played a role? And until I have further information with that particular first bullet point, the answer is maybe it's coincidence, or maybe there's something to this association.

Q And how important is that factor in your opinion that thimerosal-containing vaccines caused Yates to develop autism?

A As I've noted throughout my report, I'm a believer, based on all of the studies and research, that we're dealing with, in many cases of autism, with several factors. Such as, as far as vaccines,

thimerosal

may play a role. I mentioned my belief that Yates was sickly, so if thimerosal contains mercury, that can further weaken your immune system that can have an adverse role not to mention that mercury can cause autistic symptoms. So we have to look at all of the factors involved, and there are many.

I might add that I think that his addiction to milk at one point probably had something to do with it, as well, or at least contributed to some of his symptoms. The parents reported that there was a time, I think it was in Norway -- I don't know if it was a little bit before, as well -- where he was addicted to milk.

Well, some researchers, like Dr. Paul Shattock and Pensup have looked at opioid therapies or opioid theories for autism. Some children with autism are unable to digest casein. Casein is a protein that is found in dairy products. And there are a variety of reasons why children, some children with autism, are unable to digest the casein. And when they try to digest it, it gets converted to a peptide called caseomorphine, which is similar to morphine, and that can make the child have a variety of symptoms.

Notice in the report, though, that when they went on a gluten-casein-free diet, he did get somewhat

better. Not completely better, but he got a little bit better. So I would say it's a fair assumption that by going on a gluten-casein-free diet, they removed that particular contributing factor. He got a little bit better.

But I think it's not until he saw Dr. Buie and was placed on some medications that he had some real relief from his gastrointestinal symptoms.

Q So milk is one of the contributing factors?

A Milk was a contributing factor, in my opinion, to some of the symptoms he developed. Not necessarily causing it, but it probably made some of his symptoms, aggravated some of his symptoms.

Q The second bullet is, Yates had evidence of a compromised immune system.

A Yes.

Q How critical is that factor to your opinion that Yates's vaccines played a role in his development of autism?

A I can't give you a percentage. It helps. It's not a necessary component, but it does help. I say that it helps, because immunological problems are widely reported in children with autism, including children with gastrointestinal problems.

One mechanism by which vaccines such as, or

vaccine adjuvants like thimerosal, causes problems is by altering the immune system. So the fact that Yates was sickly adds to the picture, the profile.

Q So assuming all other factors, but Yates had a normal immune system, would that change your opinion that his development of autism was vaccine-related?

A That's a difficult question, because how would I prove that his immune system was normal? Do you mean if he was not as sickly, perhaps? Normal children can get six to 10 infections per year when they're very young.

So are you saying if Yates had, say, six infections per year, for example, and was not as sickly as he has been, would that change my opinion? Is that what you're saying?

Q Yes. Let's say he only had six infections.

A Yes. Let's say he had just a few infections, as it's recognized that you can have a few infections early on, that would not alter my opinion. What I've mentioned in terms of the MMR, if the other factors were present, some of the other critical factors.

Q How critical is the third factor that Yates was sick at the time he received his MMR vaccination?

A Based on CDC recommendations, that if a

child is moderately ill, that vaccine should be postponed; I think that that is yet another contributing factor.

Yates, I argue, had a compromised immunological system, and perhaps had an underlying genetic vulnerability. So it was very risky, in my opinion, to give a child vaccine who was sick at the time.

What I would have maybe recommended would have been to postpone the vaccine, and not to give it in a state where he might be further immunologically compromised by having an infection.

Q So if we take all of your other pertinent factors, and assuming all those factors, but he was perfectly healthy at the time that he had received his MMR vaccination, would that have changed your opinion that the MMR was related to the development of his autism?

A It depends what type of information I had. If, for example, his gastrointestinal workup did not show that he had lymphonodular hyperplasia and colitis, it would -- I'm sorry.

Q I'm sorry, Doctor. I'm saying all other circumstances the same, okay?

A Okay.

Q Except that Yates was perfectly healthy at the time he received his MMR vaccination. Would that change your opinion?

A No, it wouldn't. Because we do see a subset of children who seem healthy, and after vaccines they get really sick. So that would not change. I just think that in Yates's case, it demonstrates the coexistence of underlying genetic predisposition and environmental etiology coming together to cause problems.

Q So it's not a pertinent factor in your decision.

A I didn't say it wasn't pertinent. But the more points you have --

Q It's not necessary.

A Yes, that would be a better characterization.

Q Yates had no family history of autism. How important is that factor to your decision, to your opinion that MMR vaccine played a role in his developmental autism?

A It's important, in the sense that if Yates, for instance, had had multiple family members with autism, I would really strongly think that he had a specific either genetic or neurometabolic problem,

perhaps that I might be able to identify.

For instance, there are, at this time, approximately 100 candidate genes that have been looked at with autism. And there have been familial forms that have been identified.

So if Yates had multiple family members that had autism, I would think you know, maybe genes are playing a bigger role. Now, when I say genes, it's important to understand that some children with autism have identifiable genetic abnormalities that can be proven with the lab: for example, fragile X or neurometabolic disorders that are genetically based, such as PKU.

But there are other children who have a genetic predisposition, but detailed genetic testing, as in the case of Yates, does not reveal anything. So the fact that he did not have any family history that we know of just is yet another point to suggest that we're dealing with a problem where environmental factors play a significant role.

Q What percentage of children with regressive autism have no family history of autism?

A Studies show that autism in general, whether regressive or nonregressive, there are many families that don't actually have a family history of autism.

There is, however, a higher concordance rate if you look at twin studies, for example.

But what has been shown is that there are a lot of family members that may have traits. For example, you may have a child with autism, and there's a fairly good chance that a member may have speech delay, dyslexia, some other symptoms.

When you look specifically, though, at regressive autism, I'm not, I don't know what the numbers are in terms of family history aspect. I'm not sure what that is. It's probably, I would suspect, higher than the general population, but not necessarily very high.

Q On page 9 of your report you have a section called "Where is the Lesion."

A Yes.

Q And you state that the most important point that can be made clinically is that all parts of the brain can be involved with autism, is that correct?

A Yes.

Q And you believe that all parts of the brain can be involved in autism?

A I do.

Q What is the basis for that opinion?

A First of all, if I may back up just a little

bit, I want to explain why I wrote, "Where is the Lesion?"

I'm an ordinary child neurologist, and based on my training, what all neurologists in the United States are taught to do when confronted with any neurological problem is to ask that very question: where is the lesion. Asking that question can help you to arrive at the proper diagnosis. So that's why I chose to put this in this form.

When we ask the question as neurologists, where is the lesion, before we get any type of labs, the first thing we try to do is, first of all, get a good history. And I mentioned earlier that history-taking is important in arriving at the right diagnosis.

The next thing we do as neurologists is do a good neurological examination. Most of the times, with a good history and a good neurological examination, if you've done the job correctly, you should be able to arrive at a very reasonable diagnosis, or at least a reasonable differential diagnosis. And then if you're not sure, you may get supportive tests, such as labs, EEGs, and MRIs.

Recall that I also said that autism is not one disorder. Autism, there are a lot of different

causes for autism. And what has been shown through

both neuropathological studies, case reports, volumetric MRI studies, is that various parts of the brain are affected.

I did mention the case, I think it was Ghazinddin, the first report in my list, of a person that presented with autism due to lesions in a typical area. He had herpes encephalitis that affected the frontal lobe. Usually herpes encephalitis affects the temporal lobe. He went on to develop classic autistic symptoms.

While there are some studies that say well, you must have temporal lobe involvement, well, this person did not have temporal lobe involvement. If you just look at clinical manifestations of children with autism, they may have visual processing problems; that points to the occipital lobe. They may have mood problems; that points to the lymphatic system. They may have executive problems, motor problems; that points to the frontal lobe. They may have handflapping, involuntary movements; that points to the basal ganglia. And many studies have implicated the cerebellum as being involved.

When I review the literature to see what parts of the brain are likely involved, I see contradictions. Not because the studies are

inadequate or because they're bad, but because probably they're looking at different groups of individuals.

I suspect that some individuals may not have any visible changes at all if you did a biopsy. The problem is when you diagnose someone with autism, there is not a single laboratory test that you can do that confirms the person has autism. In fact, you do not even have to be a medical doctor to make an official diagnosis of autism. A psychologist can make the diagnosis.

I mentioned this pseudoautism, which is caused by depression. Someone like that would not have any lesions at all; not in the temporal lobe, not in the, you know, lympic system, and so forth. So my assertion that any and all parts of the brain can be involved is based on different reports of different areas being involved.

Also knowing any good neurologist who applies the rules that we're taught as neurologists to ask where is the lesion should come up with the fact that this is a diffuse problem, which involves various brain structures. So it is in that sense that I say any and all parts of the brain can be implicated.

Q Is it your opinion that both thimerosal-

containing vaccines and the MMR vaccine worked in concert to cause Yates to develop autism?

A I believe that both of them had an impact. When you say in concert, I don't know that one caused the other, but I know that both of them, based on the laboratory evaluation that I have, likely, very likely, could have played a role. In the case of the thimerosal, I see evidence of oxidative stress due to his low glutathione. I see his porphyrns, urinary porphyrns, copoporphyrns being very elevated.

So that -- all that tells me is that there is evidence, laboratory evidence, that there is an insult caused by mercury causing these laboratory changes. They don't get there -- studies have shown, Dr. James has shown that if you compare kids with autism versus kids who do not have autism, if you look at their glutathione level, intracellular glutathione, the glutathione is lower in children with autism, which makes sense -- it fits with everything we know about autism, mercury toxicity, other problems -- than those that do not.

So in that sense, I would say that yes, the mercury component of the thimerosal, and then the MMR. I see evidence, you know, laboratory wise, that strongly suggests that this was not just a

coincidence, this regression, post-MMR.

Q But you don't have a hypothesis as to how both of them combined, working in concert, to cause Yates to the development of autism.

A Well, in concert in the sense that if you weaken the immune system, we know that thimerosal can, is a neurotoxic. So if we have evidence that thimerosal is involved, which we do, and that we know that it can depress neuronal function or brain cell function; and if we know that measles can affect the gut and can also affect the brain; then, in concert, you can say that there's possibly a synergistic effect of the two.

Q What is the evidence that thimerosal causes neuronal, you said neuronal --

A Oh, yes, neuronal.

Q -- dysfunction? Is that what you --

A Yes, neuronal. There have been several studies that have looked at, and some are listed here -- I'm blanking on the name of the author. But what they've done is they've compared thimerosal and methyl mercury.

Most of the studies have initially been done with methyl mercury, which is an organic mercury. Thimerosal contains ethyl mercury, which is a different form of organic mercury. So what scientists

have asked is, could thimerosal cause similar

problems, as far as the brain, to that of methyl mercury. And the answer that was found is yes.

And as I mentioned earlier, and I don't remember the author's name, it's been found that when thimerosal goes to the brain, it actually converts to inorganic mercury, which binds very strongly to brain cells at a rate that's seven times faster than methyl mercury.

Also, scientists have said well, let's look at thimerosal. After all, thimerosal contains not only ethyl mercury, it also contains thiosalicylates. Thiosalicylate is just yet another component in thimerosal, so one researcher asked how do we know that it is specifically the ethyl mercury that's causing problems in the brain. Maybe it's the thiosalicylate.

What that particular study showed is that the thiosalicylate did not affect the immune system, but the ethyl mercury did. I mean, it affected the brain as well as the immune system it suppressed the T-cells.

So I think there is growing evidence to support the fact that ethyl mercury is neurotoxic. And I think that was part of the basis for the AAP and Human Health Services and other groups coming together

to recommend the quick removal of thimerosal from the vaccines.

Q In what dose is ethyl mercury neurotoxic?

A Yes. I don't think --

Q Are these papers talking about ethyl mercury at the doses that are contained in thimerosal-containing vaccines?

A I think some studies are a little bit misleading, or I think not designed properly. Because we should not necessarily be talking about dosages or doses. Now, we know that the EPA said that if you have 187.5 micrograms of thimerosal, that exceeds the limit that they consider safe -- not for kids, but even for adults.

Q Who said that?

A The EPA.

Q Said that about ethyl mercury?

A What's that?

Q The EPA said what about ethyl mercury?

A No. They said that the level of mercury that is considered above what is considered safe is a cumulative dose of 187.5 micrograms. It was based on that recommendation, in large part, that the recommendation was made to remove thimerosal from the vaccines. However, I don't think looking at -- and

that was for I think methyl mercury, but they applied it as well as ethyl mercury.

Q The EPA applied it to ethyl mercury?

A Yes, they did. Because ethyl mercury is also an organic mercurial, just like methyl mercury. And a lot of the data, most of the data, the initial data was with methyl mercury. All the epidemiologic, Seychelles Island and Faroe's Island, Minamata disease, these were all based on methyl mercury.

But some researchers have shown that ethyl mercury is also not only neurotoxic, but also it impairs the immune system.

Q What studies are you discussing that say that thimerosal suppresses the immune system?

A There is a study, let me see if I can find it. It's in one of the --

(Pause.)

A It's in one of the lists -- I don't know if I have it. It's basically a study, that's the same study that I'm referring to that looked at thimerosal and the subcomponents, ethyl mercury and the thiosalicylate. And I can find that for you for the record.

Q I'll ask you a few questions.

A Sure.

Q Was it an in vitro or in vivo study?

A It was, I think, an in vitro, I believe. I believe.

Q And were the doses of thimerosal equivalent to the doses that are contained in thimerosal-containing vaccines?

A I don't remember specifically what the doses were. But regarding the dosage question -- which I think I kind of went on a tangent, I didn't answer your question -- the dosage is not the biggest issue when it comes to mercury, in the sense that if you look at all children that are vaccinated, if everyone receives the same dose of methyl mercury, or ethyl mercury for that matter, not every child will be affected.

Instead, what most of the researchers have focused their attention on is the ability to excrete mercury from the body. I think most people tolerate thimerosal. Not that it's necessarily safe, but people have mechanisms in place in their body to excrete the thimerosal, excrete the ethyl mercury.

However, if you're genetically predisposed to not eliminate the ethyl mercury, even if the dose is relatively small, you may have problems. So in other words, we're dealing with a sensitivity issue.

There's a recent study that came out just

this year, measuring mercury in baby teeth. And that was a good indicator of increased body burden of thimerosal in the body. I think Bradstreet did a study where he looked at over 200 kids with autism. He compared them with a control to see if, to look at how they're excreting the mercury, and found that the control, they were able to excrete the thimerosal, or mercury, I should say, but not the autism group.

Amy Holmes has done a study where she looked at first baby hairs, where it was found that children with autism were not able to excrete mercury. They were not able to eliminate it from their body. And if you have a high body burden of ethyl mercury, then it goes to the brain and other tissues, and can cause damage.

So I think it's very important to know that. We're really dealing with an underlying problem of elimination.

Talking about biological possibility, we do have other models. For example, one neurological problem where a heavy metal is not excreted is Wilson's disease. Wilson's disease is a condition where some individuals are not able to excrete copper. The copper goes to the brain, the retina, other conditions, and you have problems.

Q And that genetic condition has been identified, correct? We know the gene that causes Wilson's disease.

A Yes, we know the gene that causes Wilson's disease.

Q We can't identify the genetic causation of somebody's inability to excrete or not excrete mercury. There has been no identified genetic disorder, is that correct?

A Well, since 2003, fortunately we've been able to -- not me, but other scientists have come up with a way to complete the human genome project. And there's a lot of research underway to understand underlying mechanisms. What I think will happen in the future is we'll never, ever -- I can make that statement with confidence -- we'll never find a or an autistic gene. We'll never do that.

But what we will find in the future, or geneticists is, we'll find groups of genes and susceptibility factors that may account for different traits. For example, we may identify in the future particular genetic mutations or problems that interfere with the ability to excrete certain metals or other things of that nature.

Q But as of right now, that's pure

speculation.

A What's a speculation?

Q That there is a genetically susceptible class of children that cannot excrete mercury.

A No, that's not speculation at all. There have been several studies that have compared children with autism versus children without autism. And it's been shown that the children with autism are not able to excrete the mercury, meaning that they have a greater body burden than those who do not have autism and are able to excrete the mercury. So that part is not speculation at all.

Q And what are those studies, again?

A I mentioned Bradstreet did a study where he looked at over 200 children.

Q And do you know where that was published?

A It was a few years ago. I don't remember a specific date.

Q And that's --

A And then there's -- I'm sorry?

Q I'm sorry. And that was this hair study, is that correct?

A No, the hair was yet another study. Amy Holmes did a study, that was also a few years ago. Don't quote me on the date, 2002, 2001, or something

like that. Amy Holmes in Louisiana did a study where she looked at baby hairs.

Now, this was an interesting study, because she really did not anticipate the findings. She thought that the group of children with autism would probably have a lot of mercury excretion. But what she found was just the opposite: kids with autism had a very low amount of mercury in their hair, compared to other children with autism.

And so with the help of Boyd Haley I think, who was a toxicologist, they were able to work out the mechanism and show that there's a problem with excretion.

There's a study, and I apologize, I forget the name, but we can give it to you. I have it here. A study that came out just this year, in 2007, looking at increased mercury in the teeth of children with autism. Again showing this increased body burden concept.

Q That study was the baby teeth of autistic children, is that correct?

A Yes. I don't remember their age. I don't think they were young like the children in Amy Holmes's group.

Q Are teeth excretory organs? I mean, do

people excrete mercury through their teeth normally?

A I don't know too much about dental physiology in that sense. But it appears that the teeth is one way of measuring body burden of anything. In fact, in that study they looked not only at the mercury, but they looked at lead, and I think they looked at cadmium.

They specifically found that if you looked at both groups, there was not a problem with lead, there was not a problem with cadmium, I think, but there was a problem with mercury. So that was good, you know, selective proof of body burden.

So that has been looked at in a variety of ways. Teeth, hair, and other methods. And that just gives credence to the fact that we're not looking at a dosage problem. If you're able to eliminate, regardless of the dose -- I shouldn't say regardless. If it's an extremely high dose, you will be toxic. But if it's a reasonable dose, then you may be able to tolerate the effects, the toxic effects.

But if you don't have the underlying genetic predisposition to clear the toxin from your body, then you'll have problems. And I think that's what all of the studies that I have reviewed show, that we're dealing with a problem, genetically based susceptibility to clear mercury from the body, not

necessarily the toxin. And I can, if I may --

MS. RENZI: Go ahead.

THE COURT: Go ahead. I was just going to say for the record, I believe the article to which you're referring is the 2007 Adams article.

THE WITNESS: Yes, that's it. Adams, yes.

THE COURT: Okay. Exhibit, Petitioner's Exhibit 47.

THE WITNESS: If I just might add that I think between 1850 and 1950, there was a disorder called Pink's disease, which baffled the medical community for about 100 years. This was a condition where children would just become very irritable for no apparent reason. They would cry for long hours, they had acrodynia. The extremities turned red and so forth, and no one could figure out what happened with these kids.

Until I think it was in the 1950s or so they discovered that they were using teething lotions and other things that contained mercury, that was getting in the system, and that was causing various problems, hypersensitivity and other types of things.

Now, some people have said that Pink's disease is synonymous with autism. This is one area I do not quite agree, because the symptoms, if you look

at Pink's disease, there are some autistic characteristics with Pink's disease, but I don't think it matches up 100 percent. But I use that example just to say that many kids were using the same teething lotions, same concentrations, same dose, but only some came down with this Pink's disease. Suggesting that again, we're not dealing with a dose issue, but we're likely dealing with the genetically based underlying susceptibility to clear the toxin.

BY MS. RENZI:

Q In Pink's disease, the teething powder was administered by parents as needed, is that correct? To the gums of the children?

A I believe so.

Q Then how can you conclude that all the doses that the children received were the same?

A I don't mean to conclude or to say that they were the same. But if you look at an entire population, only some of the children came down with Pink's disease. But that is a valid point. We don't know exactly how much -- I don't think any study was done to look at, you know, how much they were given.

But the point I was trying to make is, you know, it's likely that some people came down with the disease because -- in other words, not everyone that

used it. You might have a family where, you know, say you had twins, for example -- this part is speculation -- when someone might come down with it, and someone else, no.

But with vaccines, that is not the case, because we know exactly how much mercury everyone is getting. And we know that some people are susceptible; others are not.

Q How do we know that some people are susceptible and some people are not?

A How do we know that some people are susceptible to mercury toxicity?

Q Yes, and some people are not.

A I'm using that based on studies where, you know, children are compared that have an inability to clear mercury, versus individuals who are able to clear it very well. And the ones that are able to clear it do not have symptoms.

Going back to Amy Holmes's group, not only did she find that individuals who were able to, who had a low hair mercury level, not only did she find that these children were autistic; she was able to stratify. That is, those with a lower level, those that were not excreting things at all, were more severely affected.

So I think what I'm trying to emphasize is the fact that dose I don't think is the biggest player. What is a susceptibility to clear toxins.

Q What is the Bradstreet study that you rely on?

A I don't remember the year, but it's referenced in the Cedillo case. It was I think a few years ago. He looked at 260 children or so, and he compared them with a control; the control was smaller. And he tried to measure, I believe, the excretion of mercury in children with autism, versus children without autism, and found a higher -- he found that, I think, that the children with autism had a higher body burden than the group that did not.

Q What did he study? It wasn't the hair. Was it urine? Was it feces? What did he study?

A I think he might have done urine. I think he might have done urine. Now, I'm not sure which direction this question is going, but if I may just kind of elaborate a little bit. There have been other studies. I think Fombonne did a study -- I'm not sure I'm pronouncing his name well -- where he looked at blood levels of mercury, and concluded well, there are no changes with autism and nonautistic children.

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If one is going to do a study, it has to be done correctly. If someone is exposed to mercury, that mercury does not stay in the bloodstream for very long. So if you want to look at body burden, you have to do a chelation challenge, which means that you give a chelator, you check the urine in a few hours, and then you can measure if something is coming out.

Q And to do a chelation study, don't you need both prechelation studies, prechelation levels and postchelation levels?

A That's exactly the way I would do a study. That's right.

Q And did Dr. Bradstreet do his study with prechelation levels?

A I don't remember exactly how he did his study. But you know, I know Dr. Bradstreet is very well aware of pre and post chelation issues. So it wouldn't surprise me if he conducted his study the proper way.

Q And if he did not? If he did not obtain prechelation levels, would you still rely on that study to show that there is a subset of children --

A If he did not rely --

Q -- who cannot excrete mercury?

A I think there would still be an indication

of something is wrong. If you don't do a prechelation, and you do a chelation challenge, and that one group is excreting a lot of mercury and a group is not, I think that already shows something.

But in terms of pre- and postchelation, that would be a, you know, that's something else that could be done, as well. But I think that even without doing the prechelation, just showing that one group has heavy metals that you are able to prove through chelation versus a group that you give a chelator, the same chelator, the same dose, and they're not excreting the mercury, you can conclude that there is a difference. That should be meaningful.

I think the purpose of the study was to show, through a laboratory test, that children with autism have a higher body burden of mercury. And the mechanism to explain that is inability to spontaneously excrete mercury.

Q How else can you get mercury in your body?

A Which type of mercury are you talking about?

Q Methyl mercury? Ethyl mercury?

A Methyl mercury can be obtained through a lot of different sources: fish, the environment. I think all of us are exposed to mercury in one way or another.

Q And did Dr. Bradstreet's chelation study differentiate between ethyl mercury and methyl mercury found in the urine?

A I don't recall. I don't recall.

Q Did Dr. Bradstreet's study take into account dietary concerns?

A Such as?

Q Fish consumption.

A I don't know.

Q Would that make a difference to you in the reliability of that study, that it's due to thimerosal-containing vaccines, the chelation levels?

A Not really. In the sense that assuming, well, the research that I have found is that the fish that we consume are regulated. There are guidelines. The amount of mercury from fish, unless you get a lot of shark and some other types of fish, the smaller fish are fairly safe, based on the normal American consumption of fish, which is not extremely high.

So in, you know, generally speaking, unless you have someone who is eating a lot of shark and someone who had no exposure to fish at all, that might potentially play a role. Potentially.

Q Do you know how much mercury there is in a can of tuna fish?

A I don't know, but I think that that's, you know, highly variable. Tuna is a bigger fish, and there are certain types of tunas that contain a lot of mercury. But the last reports that I saw is that the average can of tuna, with the regular consumption that most Americans would use, would not, you know, necessarily give a problem. But I can't tell you numbers.

Q But we don't know whether Dr. Bradstreet did take a dietary profile of any of these children to know their fish consumption, what type of fish consumption they were eating.

A Yes, I don't know.

Q Do you agree that methyl mercury and ethyl mercury are different forms of --

A Yes, I agree that they are two different organic mercurial compounds.

Q And would you agree that they have different toxicological properties?

A Yes. I think we still need to study them in more detail, but as I mentioned earlier, it seems that when ethyl mercury goes to the brain, it converts to inorganic mercury seven times faster than methyl mercury. So we know that there are differences.

Q What is the basis for that statement?

A You mean, how did they do the study?

Q No. What is the basis for your statement that ethyl mercury converts into inorganic mercury seven times faster --

A There was one study that looked at ethyl mercury in comparison to methyl mercury, and that's the conclusion, based on this study. Based on their methodology, that's what they were able to report. I'm not a toxicologist, so I cannot comment on the intricacies of that particular study. But that's what was --

Q Can you name that study?

A I don't remember the author, but I'd be happy to make sure that this is provided to the Court for the record.

Q Do you know how long it takes for ethyl mercury to clear the bloodstream?

A I'm not sure exactly how long.

Q Do you know how long it takes for methyl mercury to clear the bloodstream?

A I think methyl mercury can clear the bloodstream in, I've seen some reports that say weeks, I've seen some that say a little bit sooner. But it does, you know -- maybe a few weeks. And that's an important consideration. Because if you're going to

assess mercury levels in someone who has been exposed several months ago, that may skew your study results.

Q You talked about the studies at Minamata and Seychelles and Faroe's I think in your testimony today, and also in your report, is that correct?

A Yes, that's correct.

Q Were those prenatal or postnatal exposures at Minamata?

A My understanding from the studies is that they were mostly prenatal, it's my understanding.

Q Of methyl mercury, correct?

A Of methyl mercury, that's correct.

Q Through maternal consumption of fish, is that correct?

A Yes. Yes, contaminated maternal consumption of fish.

Q And how does that study compare to the dose of thimerosal that children receive postnatally in thimerosal-containing vaccines?

A First of all, that study did look at methyl mercury, not ethyl mercury. And they were looking at prenatal exposures, that children who were affected had a different type of problem. A lot of them had a cerebral palsy type of picture.

I use that example just to suggest that

organic mercury in general, and specifically methyl mercury, does affect the developing brain. That's a starting point. We talked about biological plausibility. It is biologically plausible based on this model that organic mercury in the developing brain is toxic, neurotoxic.

At the same time, I think these studies show that the adults, as far as I recall from the study, were not affected, as the children were. Which suggests that if you have a young child who is having rapid brain development, which is the case for young infants, that they are particularly susceptible to toxins in general.

I think at Faroe Island, there was a differentiation between the Faroe Islands and the Seychelles, where I think there were more problems in the Faroe Islands, even though superficially they were both consuming, you know, contaminated fish. But they found that there were other environmental factors, such as PCBs and other toxins involved in the children within the Faroe Islands.

The other thing that was very interesting is the idea of pulsing. Instead of the one big bolus they were getting these exposures, you know, intermittently.

So I use this as a model, basically, to strongly suggest, based on epidemiologic studies, that

organic mercury is toxic to the neurodevelopmental brain. I'm not using that study to say that this study shows that thimerosal causes autism, no. I'm saying that these studies point us to the direction of looking at environmental factors. That's why I strongly believe that environmental factors play a role in autism and other neurodevelopmental problems, as demonstrated by these cases.

Q Are those studies dose-related? Do we know the dose at Minamata?

A At Minamata in Japan?

Q Do we know the dose of methyl mercury?

A I don't know if we have -- you're talking about Minamata. You're not talking about the Faroe Islands or the Seychelles Islands.

Q We'll go there first. Let's start with Minamata. They are different.

A Yes. I don't recall the dose. I'm not saying they didn't say the dose, but I don't recall that, that detail. All I know is that with Minamata disease, you know, many individuals were affected. Older individuals, not children.

In fact, with Minamata disease -- and also Iraq, where grain was contaminated with methyl mercury. This is what led the U.S. to go and look at

what's happening with the Faroe Islands and Seychelles.

So one group of epidemiologic problems led to further investigation. And I think that's the way science works. We look at all of the data, what I would call the natural history of things, and we try to apply what we know from nature, from past experiences, to what we see happening right now.

Q In the Seychelles and Faroe Islands, let's start with the Seychelles Islands. What was the consumption of fish in the Seychelles Islands?

A Do you mean what type of fish?

Q What type of fish.

A I don't recall.

Q What were the resulting neurological deficits that resulted from the prenatal exposure of fish consumption in the Seychelles?

A I think there was a range of problems, ranging from neurodevelopmental to motor problems, cerebral palsy-type pictures. So I think there was a range of neurological impairment, not just one particular problem. Learning deficits, things like that.

Q Cerebral palsy?

A Yes, cerebral palsy picture. In other

words, cerebral palsy is a condition where there is an abnormality in your muscle tone. So children with cerebral palsy can either be spastic, where they're very tight, or they can be very floppy. And there is an arrest usually, or a problem with their motor development. And of course, kids with cerebral palsy may have seizures. Some have mental retardation, though not all of them. So that was also one of the several neurological complications seen there.

Q Was autism, was there an increase in autism in the Seychelles as a result of the methyl mercury?

A In the strudies I've viewed -- I don't, at least in the articles I looked at, I didn't see autism. What I saw was a range of neurodevelopmental and, you know, motor problems.

Now, that does not mean that there was not autism, because I doubt very much that in the island, there were people who were very aware of autism and knew what to check. I think that there has been better recognition of autism over the years. So in fact, that's why some people believe that -- I'm one of them -- believe that better diagnostic or better awareness has led to better recognition of autism.

So I believe that if there were children with autism, and there's a possibility that there

were, that investigators probably were either not

aware of autism, and therefore did not ascertain whether that was present.

Q That's just speculation on your part, however, correct?

A Pure speculation, yes.

Q Faroe Islands? Do you know the consumption of fish?

A I don't know the type of fish. But what I do know with the Faroe Islands is that studies showed there was more involvement of neurological impairment than in the Seychelles Islands. And I think that that led to some comparative studies to see -- in fact, I'm sorry, the cerebral palsy-type picture. I think a lot of these things apply to the Faroe Islands.

With the Faroe Islands what was found is a lot of the children had more complications than the Seychelles Islands. At least that was statistically significant epidemiologically. And I thought that was important, too, because, you know, on the surface, they are both exposed to similar types of toxins. But later it was found that there was a different type of exposure, in terms of more toxicities in the Faroe Islands, not just the thimerosal, but according to what I've read, PCBs and other toxins.

And again, I want to emphasize, that's why I

talk of contributing factors. There may be other things out there in the environment that we do not yet know of, but hopefully in the future we'll have a better understanding of all of the various contributing factors to autism, both genetic and environmental.

Q Now, the Faroe Islands, you say thimerosal or methyl mercury?

A If I said thimerosal, I meant methyl mercury.

Q Okay. And then what was the other possible?

A PCBs.

Q Do you know if there is an increase in the diagnosis or incidence of autism in the Faroe Islands?

A I don't know. But the same thing I said about the Seychelles Islands would definitely apply to the Faroe Islands.

Q You state on page 15 of your report --

(Pause.)

Q It's the last paragraph. "Although signs of acute mercury toxicity are different from those of autism, it is very reasonable that ethyl mercury toxicity in chronic low doses with environmental triggers can contribute to the development of autism."

A Yes.

Q What is, could you please define "chronic low dose?"

A What I meant when I was writing this particular paragraph, chronic low dose is instead of a one-time large bolus, whether by mouth or intravenously, I was trying to say a repetitive dosing pattern. For example, children who were vaccinated at two, four, six months, a year, and so forth. If we're looking at mercury, they're exposed in pulses, as opposed to someone who has a large bolus of intravenous ethyl mercury, or methyl mercury for that matter. I suspect, and I think these epidemiological studies would also support that, that the changes would be very different. So that's exactly what I meant.

Q So your definition of chronic is every few months over the course of a specific time period.

A And there's no specificity. I was just trying to say recurrent doses, whether it's, you know, a month, three months, but just recurrent doses, as opposed to a big, large dose.

A good example is lead. I've taken care of kids with acute lead toxicity. If you have a very high level of lead exposure, that may land you to the ICU with coma, seizures, and other types of problems.

And by the way, you would need chelation with EDTA.

But if you're exposed to lead, for example if you live in a very old house and you're exposed to lead in a smaller level, but in a more chronic fashion, you may have other symptoms, such as attention deficit hyperactivity disorder. There may be changes in your blood in terms of small microcytic cells. So you may have a different set of clinical presentations.

Q So in that paragraph when you refer to chronic low doses, you just mean the normal vaccination schedule the children undergo.

A That would be one example. I wasn't just referring to that, but that would be one example of a repetitive small dose of toxin.

Q Chronic. You say chronic in your report.

A Yes.

Q What do you mean by "low dose?"

A Low is not very specific, but what I mean is a dose that in most healthy individuals should not cause sign of acute toxicity.

For instance, in that context, I did not include a specific value, because the only point I was trying to make is just a dose that's low enough to be considered tolerable to most people. Did I answer

your question?

Q And that would be -- so chronic low doses is just simply saying the vaccines that children receive in the normal course of proper medical care?

A No. I mean, you can infer that. But all I'm trying to say, or what I was intending to say when I was writing this, is that, you know, if you have a repetitive dose that's low, you can have symptoms.

I was, in my mind, contrasting that with a situation where, instead of having several doses over a period of time, you just have one large bolus. And I think that question is pertinent to, you know, other conditions that have been attributed to autism, with methyl mercury presenting different ways.

You know, I think that if a person has a very high exposure to mercury, and that they have verifiable mercury toxicity, depending on how the mercury is given, they may have a totally different set of symptoms.

For example, if you give a very high dose of mercury, let's say methyl mercury, or even ethyl mercury, as one of my articles point or show, you may have renal shutdown. You may have renal failure. You may not necessarily have that if you have a small dose given in small repetitive dosages.

Q So dose is important, would you agree?

A What's that?

Q Dose is important?

A It's important in the sense that it can dictate the way certain symptoms manifest. In that sense. Low, one-time dose toxicity versus small recurrent dose. But it's not important in the sense, or as important in the sense of stating that in order to have certain symptoms, you have to have a particular dose, because that varies, depending on the individual.

If an individual has a very good ability to clear certain toxins, for example let's say they have a healthy set of enzymes we call metallothionine, which is an enzyme that's responsible for clearing or detoxifying heavy metals, then that person may tolerate a higher dose of mercury; versus someone who is exposed to just a low dose of mercury, but because a particular enzyme whose job it is to rid the mercury is not functioning properly, that individual with the relatively small dose may run into problems, or may develop symptoms.

THE COURT: Is it not also true, Dr. Corbier, that dose is important when you refer to chronic low dose? You say you didn't know what that

was, but it was the

level at which a dosage would be acceptable, or that most people could survive, could tolerate.

A Could tolerate, yes.

Q So dosage is also important.

A Yes, yes.

Q For that purpose.

A For that purpose, absolutely. Let's put it this way. Another way to rephrase what I'm trying to say is I don't think that the thimerosal that children receive that do not have symptoms, I don't think it's because necessarily that that level is safe. But I think that at that level, most people's immune system, most people have a level of, how should I say, they have enzymes that are competent enough to, at that level, detoxify the particular chemical.

So another way to put this yet is if we were to give higher and higher doses, there would be more and more people that would show vulnerabilities to the thimerosal, or any other agent, for that matter. So it's in that sense that I think -- I don't know if I'm making sense. I have one thing in mind, but I don't know if I'm expressing it. Did I explain it to you?

Q I understand your point.

MS. RENZI: Is the inability to excrete mercury a recognized condition in the general medical

community?

A What do you mean general medical community? What is your definition of general medical community?

Q Is there a diagnostic code for it?

A I believe, well, I mean, that would go under a toxicological code. I can't tell you what that code is.

Q But there is a specific code for an inability to excrete mercury?

A I don't know that the code would just apply to mercury. It might, or it might just be a code dealing with heavy metal problems in general.

Q If I asked a medical toxicologist if he recognized the condition of a person's inability to excrete mercury, do you think he would recognize that condition?

A I have spoken to several, and know of several, very well-qualified professors in toxicology that would say yes. I also know others that would say no, but in medicine controversies occur all the time.

Q Can you explain to me how an inability to excrete mercury causes autism?

A You have to basically see what happens if you don't excrete mercury. The first thing that has been shown is if you do not excrete mercury, that mercury will leave the blood system -- so if you're

exposed, it will first go to the blood system. And as we said earlier, after a few weeks or so that mercury will travel, and will go to different tissues, including the brain. That's an established fact.

We know that mercury is neurotoxic. That I think is very well established. And we also know that several studies, which I mentioned before, have shown that children with autism, as compared with children who do not have autism, have demonstrated inability to get rid of the mercury.

If they're not ridding the body of mercury, naturally, that is on their own, then that mercury is in the body. And I don't think, most people would say that mercury is not safe. It's one of the, you know, most remarkable toxins known to man.

So if you're exposed to mercury, if you're not excreting it, it's going to different tissues, such as the brain. And if we know that it's neurotoxic, and if we know that children with autism are shown, through several studies, to have a higher burden of mercury; then it is, that's good evidence to suggest that mercury toxicity is linked to autism.

I say linked, because I'm not suggesting that that's the only factor present. If a child with autism is exposed to mercury, and that child has the

ability to properly excrete mercury, I don't think the mercury will necessarily contribute to that particular child's autism.

If, on the other child, it is a child with autism that has demonstrated inability to clear the body of mercury, then I think that that's good clinical evidence, better than some other evidence that we use all the time in medicine, to suggest that connection.

Q After mercury is injected, after thimerosal is injected into the body, what happens to that thimerosal?

A The thimerosal will, it will stay in the bloodstream for a period of time. And then what will happen to the thimerosal is, it will go to different tissues. And it's been shown that thimerosal can cross the blood brain barrier. So one of the places that the mercury will end up in the thimerosal is the brain. I believe other organs, it may also go to other organs. But as far as our discussion, the brain is the main concern.

Q What other organs does the thimerosal --

A I saw some studies that showed that it could go to the kidneys, for instance.

Q Do you know what percentage of the mercury

goes to the kidneys?

A I don't know what percentage, but I can tell you that it's interesting that many children with autism have a specific metabolic condition called, let me see, it's a transsulferation defect. It's a condition where -- I'm trying to remember the name of the enzyme. It's sulfa. It's phenosulfatetransferase deficiency, or PSD for short.

Phenosulfatetransferase deficiency is a condition where there is an enzyme that's not allowing proper excretion of sulfates, or there's a problem with excretion of sulfates. And that involves the renal system. That's important, because there are a lot of children that do not tolerate certain chemicals, including phenols. And that's attributed to this phenosulfatetransferase next to the renal system.

And so the kidneys I think can be a site of involvement in certain children with autism.

Q What else does the thimerosal go to?

A It I think can go to muscles. And I refer to a study that was done by Axton, where some individuals -- this was a little bit different case. They received a big bolus of thimerosal, and a lot of them had necrosis of the buttocks. So I assume that

you can have some, you know, local reaction from the

thimerosal.

Q Do you know the dose in that study?

A I don't know that the dose was identified, because the thimerosal was actually a contaminant of chlorine phenocoll, which is an antibiotic. And a lot of those studies were in Africa. So they did not even know initially that the thimerosal was present until later, until they started having problems.

It was not just children; there were adults, as well.

Q But you don't know whether dose was discussed in those papers?

A I don't know, but I know they were able to identify the thimerosal. But I don't know what the dose is. I would suspect it's a higher dose than what would be found in vaccines.

Q You state that the ethyl mercury crosses the blood brain barrier and goes to the brain, the thimerosal.

A Yes.

Q What percentage of that, what percentage goes to the brain?

A Versus other tissues?

Q Yes.

A I don't think we know that. Or at least I

don't.

Q And what happens to the mercury once it goes to the brain?

A Once the organic mercury goes to the brain, it is fairly rapidly converted to the inorganic form. The inorganic form then will bind to certain proteins in the brain very tightly, and then it will stay there for prolonged periods of time. And that's according to the study.

Q How does mercury in the brain cause autism?

A I don't know exactly how mercury in the brain causes autism, but because some studies that I have reviewed show a loss of brain cells in particular areas, such as the temporal lobes, the limbic system. These are all areas where if you have any type of lesions -- it does not have to be thimerosal, it can be an encephalitis or a neurochemical change -- you can then develop autistic symptoms.

So I think it's very reasonable to say that because the thimerosal will affect certain parts of the brain that are known to be particular areas that are neuropathologic studies, volumetric, MRI studies shown to be associated with autism, that that could be how it does.

In other words, it's a question of, in that

case, location, and not necessarily mechanism. Various impairments in the appropriate parts of the brain can result in autism sometimes, and it doesn't matter if it's a virus, it doesn't matter if it's a toxin. It doesn't matter if it's trauma. Some people with trauma have developed autistic symptoms. So that would be my answer.

Q So is the inorganic mercury then in the temporal lobe?

A What's that?

Q Where is the inorganic mercury stored? All over?

A Well, in different areas. In the temporal lobe is one area. I believe the cerebellum is another area. The cerebellum has also been implicated in, one location that's been implicated in autism, the -- of the cerebellum, through neuropathic studies, is one area that's involved. So that's one area where there's nerve cell damage due to thimerosal.

Q What studies are you relying on for that?

A For which one?

Q Where the inorganic mercury goes in the brain?

A I don't remember the authors, but I can get that to you. I reviewed quite a few studies, but I

can't remember all of the authors.

Q How does thimerosal or mercury cross -- does the thimerosal go to the brain, or the ethyl mercury that goes to the brain?

A Well, I don't know what happens to the thiosolicolate, and I don't know if that's been looked at very carefully. The only study that I'm aware of that looked at thiosolicolate, which is also part of thimerosal and ethyl mercury, what that study did was to specifically state that the ethyl mercury component had an effect on the immune system, but not the thiosolicolate. I think it was trying to look at the specific component that's toxic to the body. But that's all I know.

Q How does it cross the blood brain barrier?

A When you say how, what do you mean by that?

Q The mechanism.

A Well, I don't know exactly what the mechanism is. I don't think that that's been studied. But I would assume that it's the same way that any toxin that crosses the brain, that it would occur.

Q How does ethyl mercury convert into inorganic mercury?

A Ethyl mercury converts by a process called methylation. Methylation is an organic

compound, only you have a carbon atom and three hydrogen molecules. So if you attach a methyl group to an organic mercury compound like ethyl mercury or methyl mercury, then that will convert to an inorganic form. That's kind of interesting, because some studies have shown that children with autism in general have a higher exposure to antibiotics. So there are multiple antibiotics.

That's a problem as well as far as mercury is concerned, because in the gut we usually have a natural gut flora where we have friendly bacteria, so-called probiotics. And what these do is that they will methylate; they will convert the organic form to the inorganic form. And then what happens is that the inorganic mercury could leave the body very easily.

If the gut flora is affected such that you wipe out the gut flora, and instead you have a buildup of yeast, which several studies show that kids with autism have yeast; or if you have e. coli, then they do the reverse. So instead of having a methylation from the organic to the inorganic, they do not do this methylation, so you have a -- remnants of the methyl mercury. And that is allowed to absorb more quickly in the system, and that can cause problems.

I mention that because it's important to

look at the immune system, the gut, the brain. I think the more studies that are coming up, the more we're able to have a clearer and clearer picture of what happens to children with autism from a biochemical standpoint, from a gastrointestinal standpoint, from an immunological standpoint, and from a neurological standpoint.

Q What studies show that thimerosal causes brain damage?

A There is a study in Japan, and I don't remember the author. But he showed, I think that was one of the studies where they showed specific neuronal damage to brain cells with exposure to mercury. I mean to thimerosal.

Q Was dose a factor?

A I don't recall that detail. I don't remember exactly how that study was conducted, but that was found. And then, as I mentioned earlier, some other studies were able to find the specific location of methyl mercury.

Q Was the study in Japan an in vitro or in vivo study?

A I believe it was an in vivo, but I'd have to check.

Q I just want to go to the paragraph on page

15, the second paragraph from the bottom. A statement by the U.S. Public Health Service, Department of Health and Human Services, that paragraph.

A Yes, I see it.

Q Has the Department of Health and Human Services ever stated that there is evidence that thimerosal-containing vaccines cause autism?

A I don't think I've seen that statement. But this statement basically suggests that there was enough concern that something had to be done. So I assume -- and this is just an assumption -- that with all of the information that they had at hand at that time, there was enough concern to request that thimerosal be removed urgently. I don't think if there was no concern whatsoever, that they would have made that comment.

I am aware that some of the people that were involved here were probably aware that not adequate studies, or not enough studies were done when thimerosal was added to vaccines as an adjuvant. So I think with that, and you know, research showing that there are problems, that that was enough to suggest that thimerosal be removed. And I think that should be applauded.

Q Hasn't the American Academy of Pediatrics

specifically stated that thimerosal-containing vaccines pose no risk, increased risk, of autism?

A They may have, but that would contradict their position in this statement.

Q Has the EPA ever stated that thimerosal that's contained in thimerosal-containing vaccines causes autism?

A I don't think they were that specific, no.

Q Is it your opinion that Yates has persistent measles virus in his gut, which either caused or contributed to his autism?

A In his where?

Q In his gut.

A In his gut. I think it's more likely than not that that's the case. I can't prove it right now because I don't, labs were not done to prove it. But he does have similar findings, the lymphonodular hyperplasia, the colitis that children who have been shown to have the persistent measles virus were noted to have this persistent virus. So I'm kind of inferring, based on that, that that's a likely explanation.

Q Is it your opinion that Yates has autistic enterocolitis?

A Autistic enterocolitis is a term that Dr.

Wakefield introduced to the medical community. And I think what Dr. Wakefield was saying with that term is that in the subset of children that he looked at that had gastrointestinal problems, lymphonodular hyperplasia, and autism, that they had this new entity that he described as enterocolitis, autistic enterocolitis.

In the sense that Yates had the lymphonodular hyperplasia, or has the lymphonodular hyperplasia, and autism, then that term would be applicable, yes.

Q Could you please describe any advanced training you've had in the specialty of pediatric immunology?

A What do you mean by specialized training? You mean like a fellowship?

Q Yes.

A No. I specialized in child neurology.

Q On issues related to immunology, would you then defer to an opinion of a board-certified pediatric immunologist?

A Yes, and I think in my initial visit we talked to Yates about needing to do further immunological workup with an immunologist. And I meant by that board-certified immunologist.

In my approach to autism or any neurological problem, I look at it as a team effort. I'm trained in the field of neurology; I try to keep up as best I can with the other fields. But we do not hesitate to enlist the help of other specialists in other areas to make sure we have a comprehensive approach for the benefit of the patient.

Q If there were no evidence of persistent measles virus in Yates's gut, if the tissue samples came back negative, would you still assume that the MMR vaccine played a role in his development of autism?

A If that were done and there was no persistent measles, that would lessen my current position. But I would have to, before I, you know, made a definitive opinion, I would have to look at other mechanisms, such as autoimmunity. Yates did have an immunologic workup, but I don't think that included autoantibodies. So that would have to be included, as well.

Q How certain would you be of the MMR contributing to the development of autism without the finding of persistent measles virus? Would it still be more likely than not?

A Well, I would say yes. It would be less,

but still, I mean, unless I can come up with an

alternative explanation. You have a child who is doing well, who regresses after a very specific event. I would use the same approach for medications.

If I start someone on a new medication, such as Dilantin, and all of a sudden that individual starts having neutropenia or low blood count or some other symptoms, or ataxia, for example, yes, it could be coincidence. But I would say that unless I can find an alternative explanation, the drug I recently started that caused a change in a patient likely is the thing that may be the contributing factor.

Q You state that you're not arguing that MMR causes autism -- it's on page 14 of your report -- but can be one along with several other environmental triggers.

A Yes, that's correct.

Q And what I wanted to discuss next, what are the environmental triggers that you're talking about, environmental factors?

A One would be thimerosal. Another would be -- there are a number of viruses that have been implicated in autism. Certain herpes viruses. We mentioned herpes encephalitis, a few documented cases by not only the ones listed here, but Dr. DeLong.

So basically, there are other potential

causes, or other contributing factors in the environment. Rubella, you know, we talked about measles, but rubella has been implicated. Certain medications, certain anticonvulsants.

So although our knowledge isn't complete at this point, there are various environmental triggers that I think are widely accepted by many individuals as contributing to autism.

Q Is the list that you've just recited an exhaustive list?

A Not at all. I just mentioned the ones that, you know, most people you know, talk about, or are reported in the literature. But there's a growing number of environmental factors that are implicated in autism.

The ones I mentioned are pretty much accepted across the board.

Q Are there any other ones that you can think of?

A Some people have looked at environmental toxins from the emissions from certain factories. For example, there was an epidemiologic study at Brick Township in New Jersey, where they found that there was a very big cluster of children with autism. And they found that not too far from where these families

were staying, there was a factory. I don't remember

the specific factory, but there was a very, very high concentration of children in that particular township in New Jersey that could not really be explained readily by just genetics alone.

So that particular set of environmental factors, or these emissions from the factories were implicated. I do believe personally that there are a lot of toxins in the environment that can play a role, not only with autism, but with the growing number of children we see with asthma, allergies, and also neurodevelopmental problems.

Q With all of these environmental toxins, how do you identify the two that you're speaking of today -- thimerosal-containing vaccines and MMR -- as being the more likely causes of the development of autism?

A That's an excellent question. I go by what we have, the evidence that we have. We know that these children are exposed to thimerosal; we know it's toxic. Or MMR, for that matter.

I always leave room for other agents, other environmental triggers being present. I don't know what they are yet; I would not be surprised if in the future a growing number of environmental factors are discovered. And that's why I almost insist on saying

contributing factor, contributing factor, because there may be many. There may be many.

Q If there are many, do you know the percentage then, let's say, thimerosal-containing vaccines play amongst all of these contributing environmental factors?

A I don't know that answer, and I don't know if anyone does.

Q Could the exposure of these other environmental triggers that you have spoken of -- and I believe you said there are probably some out there that we don't even know about, is that correct?

A Yes, that's correct.

Q Could these other environmental triggers, in your opinion, cause the development of autism without exposure to either measles or thimerosal-containing vaccines?

A Are you saying if a child is not vaccinated with, say, MMR or thimerosal, could that child with those exposures develop autism? Is that your question?

Q Would you attribute it to other than toxicologic, or other environmental exposures?

A Oh, yes. Yes, I think that, I think that there are various different types of toxins that can

play a role. This is purely, I mean, we don't know what these are but, I believe that any neurotoxic agent that the developing brain is exposed to in the right individual, right individual meaning someone that has the right genetic predisposition, can result not only in the development of autism, but could also contribute to other conditions, neurologic or nonneurologic.

Q Can the exposure of thimerosal and thimerosal-containing vaccines without the other environmental factors cause a child to develop autism?

A I believe, based on the studies that I have seen, that the best I could say is it's possible. The problem is, you know, many children that receive DTP and other vaccines containing thimerosal also receive MMR. There are other environmental triggers that are present. So at least at this time, it's a little hard to separate or distinguish each different factor.

Q And I'll ask the same question with MMR. Can exposure to MMR, without the other environmental factors, cause a child to develop autism?

A I think it's likely. But again, I think that I don't know how many studies, I don't think I've seen some that have isolated MMR as an only

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environmental factor. I don't think that we're anywhere near the level of technique necessary to isolate one particular environmental factor compared to another. And I would say the same applies to almost any aspect of medicine.

Q In your report, you rely on two in vitro studies with high doses of thimerosal to demonstrate that thimerosal can cause oxidative stress and/or the depletion of glutathione.

A Yes.

Q Are there any in vivo studies that support this hypothesis?

A I believe, I believe Dr. James has done a lot of work, which I believe is in vivo, but I'd have to double-check.

Q Do the in vivo studies that you refer to show similar results with doses of thimerosal that are contained in thimerosal-containing vaccines?

A I believe so, but I'm not sure that the doses are exactly similar.

Q And I am almost done, so, you state in your report that you're the recipient of the Rock Award.

A Yes.

Q What is the Rock Award?

A When I did my training as an adult neurology

person-in-training at the University of Cincinnati, I distinguished myself academically in terms of my performance. So my program director gave me an award called the Rock Award that he named after me, for my academic performance in my adult year of neurology training.

Q Who is your program director?

A His name is John Quinlan.

Q Is that an award that's recognized by the University of Cincinnati Medical School?

A I don't know how to answer that question. I don't know that an award like that had been given before.

Q Has it been given since?

A What's that?

Q Has it been given since?

A I've not spoken to -- I've not asked that question to Dr. Quinlan. I have asked him to give me letters of recommendation when I needed a letter, but I did not discuss that particular finding. Actually, that's not too, too important to me, so I don't know. You know, having that continue, is what I'm saying, is not too relevant to me.

Q So the Rock Award was just something that was bestowed upon you by your program director.

A It was an award that was created, and that was named after me. I was the first recipient for that award, and it carried my name.

Q And you may have also been the last recipient of that award, is that correct?

A That I don't know. I have not tried to find out.

Q And he was, Dr. Quinlan was, worked in the adult neurology section?

A Yes. When you train as a child neurologist, you do a full year of adult neurology, and then you do -- first of all, you do pediatric training, and then you do a year of adult neurology, followed by two years of child neurology. My year of adult neurology was done at the University of Cincinnati, and John Quinlan was the program director there.

Q If I told you that we contacted the Adult Neurology Department at the University of Cincinnati Medical School and they have not heard of that award, would that surprise you?

A I would say that you probably did not talk to John Quinlan.

Q You also state in your expert report that you did extra training at the Mayo Clinic and at Johns Hopkins University?

A That is correct.

Q What extra training did you do in Johns Hopkins University?

A I basically applied, I contacted them and told them that I wanted to further my training. I had already, you know, I was in a program where I had a complete medical training, but I wanted to do some further training in what I considered, you know, a very recognized institution. So I decided to go various places. The first place was University of Michigan, the next was Johns Hopkins, and then I went to the Mayo Clinic. So it was general.

Q And you were at Johns --

A Well, at the Mayo Clinic I did adult neurology, further adult neurology training. And at Johns Hopkins it was further pediatric neurology training, if that answers your question.

Q And the further training, it was a year-long program that you went to?

A A month at each place. A month.

Q A month at Johns Hopkins, and a month at the Mayo Clinic.

A At the Mayo Clinic. Yes, that's correct.

Q On page 2 of your report you make the statement that given the discovery that immune

mechanisms are implicated in autism. What studies are you referring to that immune mechanisms are implicated in autism?

A I'm sorry, where are you reading?

Q I'm sorry. It's on page 2 of your report.

A Okay. Which paragraph?

(Discussion held off the record.)

A Oh, yes. Okay, I see. Yes, what is the question?

Q What studies do you rely on for your statement that immune mechanisms, there's a discovery that immune mechanisms are implicated in autism.

A There are actually several studies. There's Dr. Zimmerman at Johns Hopkins. There's Dr. Ashwood. There's Dr. Gupta. There's Dr. Singh. And these are just some of the researchers that have done a lot of work in the immunology of autism, but I'm sure there are several more.

Q Singh is the last one?

A Zimmerman.

Q No, you said --

A Oh, Singh. I believe that's S-I-N-G-H, Dr. Singh.

Q Thank you.

A And Dr. Ashwood is one of the articles that

we have listed.

Q And Dr. Zimmerman, he's a pediatric neurologist?

A That is correct.

Q He's not an immunologist.

A No, he's a neurologist, but he has studied the immunology of autism.

Q Do you consider him an authority on the issue?

A In the issue of the immunological aspects?

Q Yes.

A I respect Dr. Zimmerman, and I consider him, yes, one of the authorities, yes.

Q You state in your opinion that Yates suffered from having a compromised immune system.

A Yes. I believe that his, he shows clinical signs of immunological disturbance, yes.

Q And what is the basis for that opinion?

A Being sick all of the time.

Q It's the number of upper respiratory infections?

A Upper respiratory infections, yeast infections, swollen lymphadenopathy, or swollen lymph nodes that persist. I don't think I'd be that concerned if he was periodically sick, but a child who

is "sick all the time" I think, based on my experience, is significant clinically.

Q How many upper respiratory infections did Yates have that leads you to this conclusion that he had a compromised immune system?

A I list -- let me turn to my list. I list the different times, on page 4, under "Immunologic Profile," that he was, at least that I could find, that he went to the pediatrician for treatment. So I base my report in part on these visits.

But the sense that I get in talking to the parents is that he was not brought in every single time. We saw a report yesterday of Yates, for example, having a screaming fit, and I don't think he necessarily went to the doctor for that, or right away.

I think there are many cases where parents try not to go to the doctor if they don't have to. Sometimes they will, but a child can be sick and stay at home, or try home remedies. Several studies show that many individuals, you know, have infections that they try to handle themselves.

So if you look at all the infections, both the ones that are listed by the pediatricians and others, and the report that I heard that he was sick

all the time, that is the basis for my saying that his immunologic system was impaired.

Again, I want to make a distinction between impairment and immunodeficiency. If you have classic immunodeficiency, that should show up on some of the, in most cases on some of the immunological workup that the immunologist did. But that does not rule out an impairment of the immune system, any more than, as I mentioned earlier, that a normal imaging -- he's had normal CT scan, EEG, and other testing -- that that would rule out the neurological problem.

Also, I might add that not all of the immunological tests were done. There are a lot of other neurological tests that could have been a little bit more, I don't want to say pertinent in a way to minimize what was done, but that could have provided some more useful information.

Q How many episodes of thrush or candidis was Yates actually diagnosed with?

A Let me count, on page 4. He had one on March 7. Let's see. On January 17 he had thrush with possible yeast involvement of the skin and hand. And I put in quotation the actual mark, or the actual statement of the pediatrician.

On January 7, I think that would be the

third episode. Let's see. There is mention of that, or that he had had thrush on May 4, that he had previously had thrush. On May 5 there is that mentioned, as well, so what's that? I don't know, five, four, five, six, something. Whatever is listed in my report is what I was able to see from the medical records reported as thrush.

Q Did an examining pediatrician ever diagnose Yates with chronic swollen lymph nodes? On examination.

A I read a note from his pediatrician, I think Dr. Carlton Hayes, that from what I read from that note, or from what I gather from that note, he was, it seemed, concerned enough to investigate that possibility, that he was referred to an immunologist. As far as I can remember.

Q For chronic lymph nodes?

A No, for evaluation of the immune system.

Q Okay. Did Dr. Hayes ever diagnose Yates with chronic --

A With a chronic lymph node. I don't recall. I don't recall seeing that.

Q So that comes from, as far as you know, your statement that he had chronic swollen lymph nodes comes from statements of the parents.

A The pediatric visits, what they would ask the parents to do is to list symptoms. And so the record contains both the physician's impression diagnostically, and also in the chart is incorporated all of the concerns that the parents had. I can't remember if he specifically said if he, you know, examined the lymph nodes and diagnosed him with lymphadenopathy.

Q How do you reconcile your testimony today regarding measles virus with the testimony of the virologist, Dr. Griffin, in the Cedillo case?

A I don't recall specifically what she said about viruses and the persistence of virus. Can you refresh my mind as to what particular aspect of her report?

Q You have no recollection of that report?

A Well, I read a report, I remember. Oh, in the Cedillo. I'm sorry, I was thinking of a respondent to this case. I don't remember. I looked at some of these a while ago. But can you maybe point out a particular statement she made that I could discuss?

Q It was a very long testimony that I don't want to go through. So any of the experts that testified in the Cedillo case, you're not familiar

enough with their testimony to know whether your testimony conflicts or confirms any of the testimony of the experts in the Cedillo case?

A Well, I've looked at some of the, several of the reports a while ago. And some of them I know of from, you know, previous writings, so I kind of know the way people think. And I've read the respondents, the virologist, to my report, or the immunologist.

Q Are you familiar with the testimony given by Dr. Ward, Dr. Bustin, Dr. Fuginami's report, and Dr. Chadwick regarding the reliability of the works of Dr. Uhlman, Kawashima, Walker and Dr. Wakefield?

A A little bit more Dr. Bustin than the rest.

Q And how do you reconcile your testimony then with the testimony of Dr. Bustin?

A My testimony, first of all, the, I think there were some questions about methodology with PCR and the reliability of some labs, such as Eugenics with Dr. O'Leary in Ireland.

My assessment is made with some more recent reports, with Dr. Stephen Walker's lab and Dr. Hepner, who had reviewed not just Dr. Uhlman's work, but had reviewed some of the criticisms that had been made.

And what I got out of that is that some of the labs that have tried to replicate some of the

earlier studies -- for example, from Dr. Uhlman's lab -- did not quite match up, if you look at the methodology.

For example, there's one lab that tried to look at measles virus in a group of children that did not even have gastrointestinal problems. Also, I think one of the labs looked at kids that had gastrointestinal problems, but looked at blood samples instead of tissue samples from the gut.

I also looked at Dr. Hepburn's, the way she characterized the methodology of Dr. Uhlman, all of the controls that were used, to make sure that there was no cross-contamination, to make sure that repeating techniques or repetitive techniques, different techniques, were used to arrive at the same conclusion. And I felt very comfortable on, I'm not a molecular biologist, but based on my reading, based on the fact that she looked at it, Dr. Walker looked at it in a separate way. That they felt comfortable upholding Dr. Uhlman's work, and the validity of his conclusions.

So I, for me, reading that type of literature, I'm comfortable with that, as opposed to what Dr. Bustin would say about the reliability of those studies.

Q Have you ever done PCR?

A I've ordered it several times. But do you mean did I work as a technologist doing PCR?

A Yes.

Q No. I'm not a technologist. I'm a physician.

Q You referred to Dr. Walker.

A Yes.

Q Is Dr. Walker published? What reports by Dr. Walker are you referring to?

A I'm referring to a poster presentation that was done I think about a year ago. And the findings of which were mentioned by Dr., I think her name was Hepburn, in the Cedillo case.

Q Has Dr. Walker published the results that were on that posterboard?

A Has he published it yet? I don't know that he's already published it. I think he's, I believe he's doing some other aspects of his study. I don't think that, I haven't seen the published results yet, so I would assume, just an assumption, that it's not yet published.

Q Is the study complete? Do you know that?

A I don't know that it's completed, either.

Q So you're basing your assertions of Dr.

Walker's report on a posterboard of a study that has not yet been published, that you don't know the results of, because it's not yet complete.

A I have reviewed Dr. Hepburn's study, and I'm very comfortable with her -- first of all, I believe she's an expert, based on her credentials. And based on her assessment, I've actually read through -- again, I'm not a molecular biologist, but I've read through carefully the arguments that she made, not only for Dr. Uhlman's study, but I looked at what some of the other labs did. And I feel very comfortable at least with the information that I have present, that Uhlman's study could be upheld scientifically.

Q Have you ever done any scientific studies on thimerosal yourself?

A I'm a clinician, I'm not a researcher.

Q So you've never published any articles on thimerosal?

A No.

Q Have you ever published any articles on MMR?

A No.

Q Have you ever published a peer-reviewed article on autism?

A No.

Q No? Have you ever published --

MR. MATANOSKI: I'm sorry, did we get an audible answer to that last question?

THE WITNESS: Yes. No, I have not published a peer-reviewed article on autism.

BY MS. RENZI:

Q Have you published a peer-reviewed article on any subject relating to autism or developmental disorders?

A I've written a couple of books, but I have not, as a clinician, I have not chosen the path at this time, or at least early in my career, to get into the field of research with publication. I did mention earlier that I was invited to join the medical staff in Cincinnati to do pediatric stroke, which would have been a, you know, path that would have led to a lot of publications by this time. But I am a clinician, so I have not had the time to publish and peer review in that sense.

Q What are the books that you have published?

A All of them? Or the ones pertaining to autism?

Q The ones pertaining to autism.

A Okay. The first one was Solving the Enigma of Autism, and the second one is Optimal Treatment for Children with Autism and Other Neuropsychiatric

Illnesses. The last one I mentioned is the second book that was published in 2005, I think, and the other one was published earlier.

Q And this is, I have Solving the Enigma of Autism, this is one of your books, is that correct?

A Yes, that's the first book.

Q Did you pay to have that book published?

A Are you asking if, are you inferring was this a self-published book?

Q Yes.

A Yes, it was a self-published. Both of them, all five of my books have been self-published.

Q Were they peer-reviewed by any other pediatric neurologists?

A I have shared that particular book with other neurologists to kind of get their thoughts. But when you say peer-reviewed, in terms of, you know, did they accept what I wrote? I mean, I got comments from other professionals, yes, after it was published.

Q How many patients have you ever treated with SSPE?

A I have not treated any patients with SSPE. SSPE I would say is extremely rare at this time, so I have not treated anyone with SSPE that I could recall.

Q Have you ever treated someone with measles

inclusion body encephalitis?

A No.

Q What kind of virus is measles virus?

A I think it's a paramyxovirus.

Q A paramyxovirus?

A I believe so.

Q And what is a paramyxovirus?

A It's a specific type of virus that is very virulent, and can cause a lot of immunosuppressive symptoms. That's what I can tell you on that type of virus.

MS. RENZI: I have no further questions.

THE COURT: Mr. Webb?

MR. WEBB: I don't have any further questions.

THE COURT: I do. And I have to say, we're probably at the limit. Maybe nobody else had as much coffee, tea, and Diet Coke as I have, so I'll try and move through this very quickly.

(Laughter.)

THE COURT: Dr. Corbier, you are a clinician. In your current patients about the ages of two, three, and four years old now who have recently been diagnosed with autism, do any of these clients fit the profile that you've described for Yates?

THE WITNESS: There are some -- but well, since I moved to North Carolina, a lot of the patients that I see are quite a bit older. And a lot of the patients that I've seen recently have had problems, not so much with the regressive types. I have to say that I have seen less patients with autism here than I did in Montgomery, so the ratio is less.

THE COURT: So you're seeing fewer autism patients.

THE WITNESS: In general, yes. Right now, yes.

THE COURT: The ones that you're seeing who are older, have they followed you generally from your practice in Alabama?

THE WITNESS: I've had several patients that have come from Alabama to North Carolina to see me. And I've had some individuals from out of state. I think Yates is an example. Even when I was in Alabama, they drove from Tennessee to see me, and I'm still following them. The last visit was in July. So does that answer your question?

THE COURT: It does. Have my questions triggered any further questions by counsel?

MS. RENZI: No.

MR. WEBB: I don't have any.

THE COURT: Okay. I thank you all. I think we are scheduled to resume tomorrow with the testimony of Dr. Rust, am I correct?

MS. RENZI: Yes.

THE COURT: And we'll look to resume again at 9:00 a.m. We are in recess until 9:00 a.m. tomorrow.

(Whereupon, at 1:28 p.m., the hearing in the above-entitled matter was recessed, to reconvene at 9:00 a.m. the following day, Wednesday, October 17, 2007.)

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REPORTER'S CERTIFICATE

DOCKET NO.: 03-654V

CASE TITLE: Hazlehurst v. Secretary, HHS

HEARING DATE: October 16, 2007

LOCATION: Charlotte, North Carolina

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: October 16, 2007

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