

UNITED STATES COURT OF FEDERAL CLAIMS

THERESA CEDILLO AND MICHAEL)
CEDILLO, AS PARENTS AND)
NATURAL GUARDIANS OF)
MICHELLE CEDILLO,)

Petitioners,)

v.)

Docket No.: 98-916V

SECRETARY OF HEALTH AND)
HUMAN SERVICES,)

Respondent.)

Pages: 1024 through 1213

Place: Washington, D.C.

Date: June 15, 2007

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IN THE UNITED STATES COURT OF FEDERAL CLAIMS

THERESA CEDILLO AND MICHAEL)
 CEDILLO, AS PARENTS AND)
 NATURAL GUARDIANS OF)
 MICHELLE CEDILLO,)
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 Petitioners,)
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 v.)
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 SECRETARY OF HEALTH AND)
 HUMAN SERVICES,)
)
 Respondent.)

Docket No.: 98-916V

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

Friday,
 June 15, 2007

The parties met, pursuant to notice of the
 Court, at 9:02 a.m.

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

APPEARANCES:

For the Petitioners:

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C O N T E N T S

| <u>WITNESSES:</u> | <u>DIRECT</u> | <u>CROSS</u> | <u>REDIRECT</u> | <u>RECROSS</u> | <u>VOIR DIRE</u> |
|-----------------------------|---------------|--------------|-----------------|----------------|------------------|
| <u>For the Petitioners:</u> | | | | | |
| Marcel Kinsbourne | 1027 | 1122 | -- | -- | -- |

P R O C E E D I N G S

1

2

(9:02 a.m.)

3

4

SPECIAL MASTER HASTINGS: To those listening at home, we are ready to go up here on the bench.

5

(Pause.)

6

7

SPECIAL MASTER HASTINGS: Are we going to start with Dr. Kinsbourne's testimony, I assume?

8

MS. CHIN-CAPLAN: Yes, sir.

9

10

SPECIAL MASTER HASTINGS: Doctor, please take the witness stand.

11

Are you ready to go, Ms. Chin-Caplan?

12

MS. CHIN-CAPLAN: I am, Special Master.

13

14

SPECIAL MASTER HASTINGS: All right. Dr. Kinsbourne, could you raise your right hand, please?

15

Whereupon,

16

MARCEL KINSBOURNE

17

18

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

19

20

SPECIAL MASTER HASTINGS: All right. Go ahead, Ms. Chin-Caplan.

21

MS. CHIN-CAPLAN: Thank you, Special Master.

22

DIRECT EXAMINATION

23

BY MS. CHIN-CAPLAN:

24

25

Q Could you kindly state your name for the record, please?

1 A Marcel Kinsbourne.

2 Q Dr. Kinsbourne, would you give the Court
3 your current business address?

4 A 158 Cambridge Street, Winchester,
5 Massachusetts.

6 Q Doctor, will you give the Court a brief
7 description of your educational background from
8 college?

9 A Yes, ma'am. I was educated at Oxford
10 University in England, and I went to Oxford University
11 Medical School and Guy's Hospital in London for
12 training.

13 After I got my British equivalent of the
14 M.D. degree, I went into postgraduate training and
15 specialties.

16 SPECIAL MASTER HASTINGS: Doctor, can you
17 maybe move both microphones a little closer to you?
18 The small one as well. As close as they'll get.

19 Speak up as well as you can so the people
20 listening in by phone conference can hear as well.

21 THE WITNESS: Yes, sir.

22 SPECIAL MASTER HASTINGS: I'm sorry to
23 interrupt. Go ahead.

24 THE WITNESS: I undertook training in
25 medical specialties centered in pediatrics and

1 neurology. The period of training in England at the
2 time was longer than it is in this country. It was 11
3 years.

4 At the end of that period of 11 years of
5 training I obtained a university lectureship at Oxford
6 University in experimental psychology, which has been
7 the basic science on which I've relied in the course
8 of my medical career.

9 From there I moved to Duke University
10 Medical Center where I was appointed Associate
11 Professor of Pediatrics and Neurology and chief of the
12 Division of Pediatric Neurology. I was there for
13 seven years and then moved to the University of
14 Toronto and the Hospital for Sick Children where I was
15 Professor of Pediatrics (Neurology).

16 After six years in Canada, I made the
17 decision to no longer work in the conventional faculty
18 fashion in terms of service, teaching and research,
19 but to concentrate on my research program, which by
20 that time had become significant as is reflected by
21 the publications in my curriculum vitae.

22 So instead I got an appointment at a
23 research institute, the Eunice Kennedy Shriver Center
24 in Waltham, Massachusetts, which is dedicated to
25 studying developmental disabilities and mental

1 retardation, and I was chief of the Division of
2 Behavioral Neurology in that center.

3 At that time it was my responsibility to
4 secure federal funding for our research department,
5 and I did that for 10 years with multiple grants from
6 NIH institutes and other agencies and stayed there
7 until 1990.

8 Now, in the course of being in charge of
9 that research institution, I probably did more
10 clinical work than ever before because we were
11 studying hundreds and hundreds, maybe thousands or
12 more, of children with attention deficit hyperactivity
13 disorder and similar conditions. We were studying
14 them exponentially in controlled conditions, but I
15 took a history and examined every single one of them,
16 and I managed many of them for periods of time pro
17 bono.

18 After about 1991 my personal involvement
19 with patients dropped considerably by my own decision
20 so that I only have been taking patients for
21 particular reasons. I've been following some families
22 actually for 20 or 30 years and I see others on
23 request, but I don't have a regular practice on an
24 automatic weekly basis anymore.

25 However, I have maintained close contact

1 with the medical literature. I have written chapters
2 for textbooks, notably the most prominent textbook in
3 child neurology, Dr. Menkes' textbook, where I have
4 contributed a chapter on disorders of mental
5 development in every one of its seven editions, the
6 last one being 2006.

7 Until about a year or two ago I gave roughly
8 monthly grand rounds at the Boston Veterans
9 Administration Medical Center at Jamaica Plain in
10 Boston. They have meetings from 9:00 to 11:00 on
11 Thursday mornings and patient demonstrations. I would
12 take my turn with the other colleagues in
13 demonstrating how to examine people with brain
14 injuries, particularly ones affecting higher mental
15 functions such as memory and language and so on.

16 I've also maintained contact in a more
17 specialized way with events in a disorder which I was
18 the first to describe and has several names, of which
19 one of them is Kinsbourne Syndrome. Because I was the
20 first to present that disorder, people often contact
21 me and continue to do so for advice and information,
22 and I've keep in touch with the science around that,
23 which really it's an immune mediated neurological
24 disorder.

25 In parallel to this work I have been engaged

1 in a variety of inquiries which are less directly
2 biomedical, and my bibliography reflects what those
3 are. In 1995, I took the full-time position of
4 Professor of Psychology in the New School University
5 in New York where it's my responsibility to teach the
6 graduate students basically how the brain works,
7 particularly with respect to disorders of high mental
8 function, of emotion, intellect, memory and so on.

9 I've now been there for 12 years, so I live
10 in the Boston area. I commute to New York to do my
11 university work. I then go back home and do the
12 paperwork associated with that and also do my
13 scholarly work and my articles and preparing for
14 presentations at conferences and so on, and then on
15 weekends I review medical/legal files.

16 My involvement medical/legally is very
17 largely with the program at the U.S. Court of Claims.
18 I do consult in some civil litigation. My major
19 topic there would be defense work in cases of alleged
20 subclinical lead poisoning, and then I have a scatter
21 of other types of cases.

22 BY MS. CHIN-CAPLAN:

23 Q Now, Doctor, you indicated that you publish
24 articles?

25 A Yes.

1 Q To date, how many articles have you
2 published?

3 A It's a little over 400.

4 Q And what was the most recent article that
5 was published? Do you recall?

6 A Well, the most recent one actually was a new
7 departure for me. I'm a member of an international
8 group of scientists, some very prominent, who are
9 interested in sort of bridging the culture gap, the
10 gap between strict science and other intellectual work
11 in humanities.

12 Each year a question is sent to about a
13 hundred of us, and last year the question was what are
14 you optimistic about, and the idea is to let your hair
15 down and speculate, so I listed in my CV an entry
16 which is to be found on the internet in which I am
17 optimistic about the possibility that in the future we
18 will extend our lifespan by needing less sleep. You
19 can understand why I particularly would be interested
20 in that, so if you wish to be amused you could look it
21 up.

22 Recently I've had a publication, an
23 empirical publication, on the specialization of the
24 right and left hemisphere for positive and negative
25 emotion. I have a paper submitted on the effect of

1 interferon-alpha on visual function in people who are
2 receiving it as treatment for hepatitis C.

3 There are a variety. Most years I have a
4 number of articles, and they are listed in my
5 curriculum vitae.

6 Q Doctor, you indicated that you're an author
7 in the textbook by Dr. Menkes on child neurology?

8 A Yes. I've written chapters for other
9 textbooks as well.

10 Q And have you contributed to each edition?

11 A Each edition.

12 Q What is the current edition?

13 A Seven.

14 Q Now, you mentioned Kinsbourne Syndrome,
15 Doctor.

16 A Yes.

17 Q Is that true? What is Kinsbourne Syndrome?

18 A It's a neurological disorder of infants
19 actually age nine months to two years in which
20 unexpectedly and often abruptly they get into a
21 myoclonic state. They have a relentless twitching of
22 many of the muscles of the body in unpredictable
23 sequence while they maintain full consciousness and
24 apparently clarity of mind.

25 I published my article on this in 1962. The

1 point was, first of all, to differentiate it from
2 acute cerebellar ataxia, which is another infant
3 syndrome of motor instability, but actually I
4 recognize as being different from this myoclonic
5 state, which is nowadays called Opsoclonus Myoclonus
6 Syndrome, OMS, or Dancing Eyes Syndrome or Kinsbourne
7 Syndrome.

8 I inferred that it was immune mediated,
9 which in fact turned out to be the case, and we
10 treated the children with ACTH with dramatically
11 positive effects. In fact, one could dispose
12 completely of the motor abnormality with ACTH.

13 However, if you lowered the dose of ACTH
14 beyond a certain level, which you would like to do
15 because it's not totally safe, then the disorder would
16 recur, and then after a few years it, as it were,
17 burns out, and subsequent to that it does leave in
18 most cases some learning disabilities and emotional
19 difficulties. There are permanent consequences.

20 Just recently I was a co-author on an
21 article on one of the children that I published in
22 1962 who was now evidently in his forties, and Dr.
23 Pranzatelli and other colleagues and I wrote about his
24 current state, which is still substantially impaired.
25 There was a 43 year follow-up.

1 Q Was that 43 year old follow-up published?

2 A Oh, yes. It's listed in my CV.

3 Q And you were a co-author on that?

4 A Dr. Pranzatelli was the lead author, and I
5 was one of the co-authors.

6 Q Now, Doctor, you indicated that at that time
7 it was immune-mediated disorder. Is that still the
8 current thinking?

9 A Absolutely. Yes. Of course, much more has
10 been discovered about it now.

11 Q So for almost 50 years now this syndrome
12 that you first discovered was considered to be immune-
13 mediated by you and continues to be immune-mediated
14 currently?

15 A That's correct.

16 Q Doctor, you indicated that you treated these
17 patients with ACTH.

18 A Yes.

19 Q Has that treatment changed significantly?

20 A No. It's still used. It's now supplemented
21 with other measures as well, but it still is perhaps
22 the most immediately effective of the treatments,
23 although one tries not to keep it going for too long
24 if one can substitute other agents. So several agents
25 are used, but ACTH certainly is among them.

1 Q So for the past 50 years the treatment that
2 you instituted is still considered valid treatment in
3 the treatment for Kinsbourne Syndrome?

4 A Correct.

5 Q Doctor, you're a pediatric neurologist?

6 A Yes.

7 Q Was there a board certification available
8 for pediatric neurology when you began practicing it?

9 A No. When I came to the U.S. in 1967, I came
10 with British certifications which covered my
11 activities in neurology and pediatrics, and actually
12 Duke University didn't particularly need me to get
13 further certifications, but I did need to have one of
14 the boards so that I could function as a consultant
15 from the point of view of third party payments.

16 So I took the board of pediatrics, which was
17 the first to come up, in New Orleans in early 1968 --
18 I remember the night before I saw *Bonnie and Clyde*;
19 that sort of marked the occasion for me -- and
20 obtained that certification.

21 I haven't subsequently been asked by any
22 employer to get further certification, so in fact I
23 have not attempted the currently existing neurology/
24 pediatric neurology boards that are prevalent in this
25 country.

1 Q But you were practicing pediatric neurology
2 before it was recognized as a subspecialty here in the
3 United States?

4 A Yes. I mean, very early. There were some
5 pioneers like Dr. Randolph Byers in Boston, but it's
6 amazing how that specialty has escalated.

7 Q Have you been associated with professional
8 and scientific organizations?

9 A Yes.

10 Q Have you been associated with them on a
11 national level?

12 A I'm sorry?

13 Q Professional associations and scientific
14 organizations. Have you been associated with them on
15 a national level?

16 A Well, I'm a member of numerous societies,
17 and I'm a fellow of quite a few of them. I've been
18 president of two of them. I don't know if that's what
19 you mean.

20 Q Well, you indicated you were the president
21 of two associations. Can you name those associations?

22 A Yes. One is the International
23 Neuropsychological Society, and the other is the
24 Society for Philosophy and Psychology, and I'm
25 experted into both of those.

1 Q And have you received any special
2 recognition awards from these societies?

3 A Well, actually a year ago last February
4 there was an international meeting actually in Boston
5 of the International Neuropsychology Society where
6 they organized a symposium in my honor in honor of
7 lifetime achievement, and I have this piece of paper
8 to reflect that fact.

9 Q On an international level, Doctor, did you
10 receive a special recognition award?

11 A International Neuropsychological Society.
12 Yes.

13 Q And your lifetime achievement award was from
14 that organization? Is that what you're saying?

15 A I'm sorry. I'm losing you now.

16 Q Did you indicate that you received a
17 lifetime achievement award from them?

18 A Yes, I did.

19 Q Now, Doctor, have you held any appointments
20 in the National Institutes of Health?

21 A Well, years ago I was policy advisor to one
22 of the branches of the Neurological Institute, the
23 Institute for Communication Disorders, and that's
24 reflected in my curriculum vitae.

25 Q Have you been called to testify before any

1 governmental agencies?

2 A I've testified twice before the Committee
3 for Government Reform.

4 Q Doctor, at some point in time were you asked
5 to review the medical records of Michelle Cedillo?

6 A Yes, ma'am.

7 Q Could you kindly tell the Court the
8 information that you obtained from your review of the
9 medical records?

10 A The information that I gleaned from the
11 medical records?

12 Q Yes.

13 A Yes. In brief overview, Michelle Cedillo
14 was thought to have developed normally for the first
15 year of her life. Her pediatric consultants, whom she
16 saw regularly, found no abnormality in her and made no
17 referrals to any special testing or intervention until
18 she received the MMR vaccination at age 15 months.

19 Now, what happened was that seven days after
20 the vaccination she abruptly had high fevers up to 105
21 lasting for four days. Then the fever abated
22 somewhat, but then recurred I think actually on
23 January 5, 1996, so over a period of about 20 days she
24 had a fluctuating intense febrile response really more
25 than one would normally expect to see after MMR

1 vaccine, but in the timeframe within which one would
2 expect to see fever after MMR vaccine because it was
3 the timeframe of the viremia, the presence of the
4 virus in the blood.

5 Mrs. Cedillo described both in her affidavit
6 and to me in conversation -- I've spoken twice to her
7 incidentally, once I think in 2000 or 2001 when I
8 first reviewed the case and once two days ago when I
9 went to see Michelle at the hotel where they are
10 staying.

11 Her mother described that during the fever
12 Michelle was irritable. She cried not always
13 inconsolably, but sometimes inconsolably, and when the
14 fever abated she did not utter any words at all. She
15 had had a number of words before then. She just fell
16 silent and stayed that way, incidentally, for a long,
17 long time.

18 Also, she no longer showed any interest in
19 being held. One can see from videos, which perhaps
20 will be discussed later, of her first year of life
21 that she was constantly being held and by no means
22 arched away or showed anything other than satisfaction
23 in being held, and that was no longer the case. She
24 didn't want to be held. She wasn't interested in
25 interacting, even looking at her parents.

1 She didn't seem to respond, particularly not
2 to any sound, particularly not when her name was
3 called, which she had done before, and her patterns of
4 play changed in that they became repetitive.

5 Basically she was lining things up or attaching things
6 in a string, and she would do that over and over.

7 Finally, of the items that I recall from
8 those conversations, she had a fixation on Sesame
9 Street videos, which she wanted to watch over and over
10 and over and was excited to be watching at any given
11 time.

12 Q Doctor, when you reviewed the medical
13 records did you discover that a gut biopsy had been
14 done on Michelle?

15 A Yes, but before I say that I should say that
16 within two weeks of the vaccination the records
17 reflect that Michelle began to have diarrhea, and from
18 then on she has had various complications, which
19 according to the records had to do with inflammation
20 of the lining of the gut at various levels. She has
21 been treated and is being treated for that.

22 Q Doctor, you indicated that she started to
23 have diarrhea. Has that diarrhea persisted to this
24 present day?

25 A Yes.

1 Q And her mother sought treatment for that
2 diarrhea?

3 A She did.

4 Q Is that reflected in the medical records?

5 A Yes, it is.

6 Q And does Michelle currently continue under
7 the care of a pediatric gastroenterologist?

8 A Yes, she does.

9 Q And in your review of the medical records
10 does Michelle receive treatment for her
11 gastrointestinal disorder?

12 A Yes.

13 Q Do you recall what that treatment is?

14 A Well, she had Remicade I remember, and I'm
15 sure other agents as well.

16 Q Okay. And from testimony that you've heard
17 now this week are you aware that Michelle is currently
18 receiving Humira?

19 A Yes, I am indeed. Yes.

20 Q And that is from her current pediatric
21 gastroenterologist?

22 A Yes.

23 Q So, Doctor, when you reviewed these records
24 at some point in time did you learn that Michelle had
25 undergone several endoscopic procedures?

1 A That's correct.

2 Q And when you reviewed the records did they
3 indicate that a gut biopsy had been obtained and sent
4 off to Unigenetics?

5 A Yes.

6 Q And do you recall what the result of that
7 gut biopsy was?

8 A It was positive for measles virus.

9 Q Now, Doctor, you indicated that Michelle
10 started to not respond to her name and she didn't want
11 to be held and she was lining up her toys. Did she
12 eventually receive a diagnosis for her behavior?

13 A Yes. She was diagnosed as being autistic.

14 Q Doctor, what is autism?

15 A I'm inclined to say that's a good question,
16 but the current descriptions as it were are children
17 who have abnormality in three major domains. They
18 have language difficulties, sometimes extending to
19 being completely nonverbal, and when verbal the
20 language is deviant in a variety of characteristic
21 ways.

22 The child has social difficulties, which
23 seem at least in part to have to do with lack of usual
24 motivation to be social with other people. Part of
25 what we're born with is an intrinsic motivation to

1 interact. Babies find it very satisfactory to get
2 attention from caretakers and respond in positive
3 ways.

4 It seems that the human brain is
5 preprogrammed to look for this, but not in the case of
6 many autistic children, and of course that disinterest
7 in interacting with others precludes them from
8 acquiring skills in interacting with others so they
9 then are in part unmotivated to interact, and when
10 they do interact or have to for various reasons are
11 not good at it.

12 These are what are called negative domains
13 with deficits, deficiencies, lack of the usual
14 accomplishments. The third domain is positive in the
15 sense that characteristic movements, movement patterns
16 that autistic children are apt to make not always, not
17 all the time, but another characteristic when they do,
18 which are called stereotypic movements.

19 It's very characteristic that whatever these
20 children do they keep on doing. They are repetitive
21 in their interests, repetitive in their play, and when
22 they make these movements these are flapping movements
23 or whirling movements which they make.

24 My own appreciation of it is that they make
25 them particularly when they are overaroused and

1 overanxious or overexcited and that actually these
2 movements aren't so much an abnormality as they are
3 the primitive tactic to calm yourself down. I've
4 written about that. There's a published article which
5 discusses this further.

6 In observation one finds these three
7 characteristics. Of course there are more. The
8 children get very upset if anything is changed, even
9 what others would regard as trivial details. When
10 they observe a scene they don't look at the people in
11 the scene and particularly they don't tend to look at
12 the faces, but they look off away from the faces of
13 people.

14 When they regard objects, they tend to fix
15 on little details on the surface of the subject, of
16 small components of the object, rather than as one
17 typically would, the object as a whole.

18 The children are often peculiarly sensitive
19 to certain sounds like say a vacuum cleaner sound.
20 That might tremendously upset such a child and yet be
21 totally obvious of other sounds like being called by
22 name.

23 Those children who are highly functioning
24 enough to have interests and hobbies tend to choose
25 hobbies that are notable for their lack of practical

1 utility, like dinosaurs. They love to play Legos.
2 They like closed sets of items or a particular set of
3 knowledge like sharks where you can actually master
4 all of it, and none of it is going to get you.

5 I've yet to see an autistic child who has an
6 interest that could be of practical utility other than
7 computer skills, which in a way is a great gift to the
8 highest level children or adults of this kind. So
9 they have unusual interests, and at any one time they
10 have one interest. They might change it to another
11 after maybe a period of years.

12 There are more things to describe. It's an
13 incredibly fascinating and rich topic, but let me
14 suffice that for the moment as a description.

15 Q Have you published articles on autism?

16 A I have published some articles on aspects of
17 autism, yes.

18 Q Doctor, in your evaluation and study of
19 autism, have there been any indications of what the
20 cause of autism is?

21 A Well, yes and no. There are none and there
22 are too many. It is as complex a situation as anyone
23 might find in medical science.

24 In my current chapter with my colleague,
25 Frank Wood, on developmental disorders I have an

1 extensive section on autistic spectrum disorder. It's
2 filed with my report.

3 What is affected there, which is going from
4 the medical literature, is that many, many single gene
5 syndromes, rare syndromes which have autism, has a
6 feature in some cases almost invariably, in many cases
7 occasionally, but there's a great list of different
8 syndromes, all of which are associated to some extent
9 with an autistic outcome, and yet the syndromes
10 themselves are different from each other so there
11 clearly isn't one underlying cause for all this.

12 Actually, in child neurology that is a
13 common situation that there are many outcomes in child
14 neurology -- and cerebral palsy is another one, for
15 example; epilepsy is another one -- where you have
16 what's called a functional convergence to a particular
17 outcome, and yet there are many possible causes that
18 injure the brain in such a fashion that that outcome
19 results.

20 I think most people working in the field of
21 autism recognize that autism has many, many causes.
22 Now, these recognized causes of autism, so-called
23 medically at least to some extent to explain -- not
24 really explain, but at least associated with clear
25 medical abnormalities other than the autism itself --

1 constitute no more than 20 percent, maybe 10 percent,
2 of all of the children diagnosed autistic.

3 The great majority are not single gene
4 cases. There is no doubt that genetic predispositions
5 to becoming autistic are very powerful, and that's
6 been accurately described in literature and I've
7 referenced it in my report.

8 However, many people believe that these
9 predispositions don't in themselves suffice to areas
10 where it condemns the child to becoming autistic, but
11 that they interact with certain environmental factors,
12 and when those environmental factors interact with the
13 predisposition, then the autistic syndrome would
14 result.

15 So what I'm referring to here is what is
16 called gene environment infraction, which is a very
17 interesting growing field at this very time, a field
18 of investigation. Final answers are not available,
19 but, for instance, just a few months ago the Institute
20 of Medicine had a special symposium dedicated to gene
21 environment infraction and environmental factors in
22 autism.

23 Dr. Tom Insel, the head of the National
24 Institute of Mental Health, actually referred to that
25 in the keynote address he gave at an annual autism

1 meeting that happened not long ago It's called IMFAR,
2 I-M-F-A-R. It's an acronym.

3 In the articles I submitted there's an
4 article by Dr. Martha Herbert, which reflects very
5 eloquently this growing point in autism research.

6 So in terms of the cause of autism, what do
7 we definitely know? Rather little outside the special
8 syndromes. What do we to some extent understand,
9 suspect, further pursue and maybe think likely? Quite
10 a lot.

11 Q Doctor, to be perfectly clear you spoke of
12 single gene defects. Has the literature supported
13 that autism is caused by a single gene defect?

14 A Not at all, but you see the word autism is
15 misleading because it's a compendium term for many,
16 many different conditions which haven't had a feature
17 in common, the behavior.

18 Certainly in some of the syndromic cases as
19 well usually children also have what's called facial
20 dysmorphology. They have observable and measurable
21 minor malformation of the arrangement of the features,
22 for example.

23 The actual genetic abnormality has been
24 identified, but in the vast majority of autistics, 80
25 to 90 percent, nobody has found a single gene, and in

1 fact it's generally believed that the inheritance is
2 polygenetic, that multiple genes are involved, not
3 always the same assembly in every case either.

4 So there is a powerful genetic element, but
5 it is to confirm a susceptibility and not a
6 predestination to autism.

7 Q Doctor, when it's thought that there's a
8 genetic susceptibility to developing autism, is that
9 an indication that every child will develop autism?

10 A No. On the contrary, there's little doubt
11 that some children happen not to encounter the
12 provocative or triggering situation, whatever it is,
13 and the wide range of possibilities and not become
14 autistic.

15 Even in identical twins who share the same
16 genome, not always are both twins autistic. When they
17 are, not always are they autistic to the same level of
18 severity.

19 Q Doctor, you mentioned gene environmental
20 interaction.

21 A Yes, ma'am.

22 Q Does the literature indicate that the gene
23 environmental interaction is a potential cause of
24 autism?

25 A There are many references to exactly that

1 fact.

2 Q So, Doctor, to be perfectly clear, the
3 thinking in the literature now is that the individual
4 has a genetic susceptibility to developing autism? Is
5 that true?

6 A Certainly.

7 Q And it's something in the environment that
8 triggers the onset of autism? Is that it?

9 A Yes, although I'll restate it. In many
10 cases, and nobody said necessarily in every case, it
11 requires what some have called a second hit, a second
12 event, to realize the potential risk in an actual
13 development of the disorder.

14 One has to also remember that the term
15 environment is used in a very broad sense, including
16 the prenatal environment and the postnatal
17 environment, and there are various possibilities being
18 followed up in both of these temporal domains.

19 Q Doctor, when we speak of environmental
20 causes, are you speaking of things like infection?

21 A Yes.

22 Q Metabolic disorders?

23 A Well, metabolic disorders would usually be
24 inherited. They would tend to be genetic, but no one
25 has really shown inherited disorder metabolism in the

1 vast majority of autistic children.

2 With respect to environmental causes, a
3 broad range has been and is being looked at, including
4 infections, which would be virus infections and
5 various toxicities.

6 There are rare but very informative cases
7 where a child or even an adolescent or adult has had
8 an encephalitis, in some cases a herpes encephalitis,
9 in other cases I think a cytomegalovirus encephalitis,
10 and becomes autistic at much older than the usual age.

11 Of course, there is a condition called
12 childhood disintegrative disorder, CDD, where the
13 child may become autistic after having been normal for
14 five or six years, so although autism is heavily
15 biased towards the early parts of life, it is not
16 exclusively of origin then.

17 Q Would vaccine be considered a potential
18 environmental trigger?

19 A In my opinion, yes.

20 Q Doctor, you mentioned that there were
21 different types of autism. Could you describe those
22 different types of autism?

23 A There's a number of different ways of
24 characterizing them. The standard one is to talk of
25 autistic individuals as being on a spectrum with

1 infantile autism, the classical syndrome as first
2 described by Kanner, K-A-N-N-E-R, being at one end of
3 the spectrum and then the less severely affected case,
4 the Asperger cases, being at the other end of the
5 spectrum, and in between and in parallel there may be
6 other rare syndromes like Rett Syndrome and CDD, which
7 I've mentioned.

8 There's another subtyping, which I think
9 until recently affected remarkably little attention
10 for the interest that's in it, and that is a
11 distinction that has to do with the manner of onset of
12 the disorder. The majority of autistic children seem
13 to in a sense emerge or go into or at an early point
14 become perhaps more flagrantly obviously autistic, but
15 in looking back one would think of them as earlier or
16 even congenital cases.

17 But there is a minority which, as I
18 mentioned earlier, has been estimated at 20 percent
19 maybe of children who regress, who actually lose
20 skills which they had before. Now, for a child
21 neurologist if a child loses skills that he or she had
22 before, that is a very important anomalous matter and
23 requires serious attention.

24 There are a number of possible reasons --
25 certain forms of epilepsy which are hidden and not

1 totally obvious but cause lapses of consciousness
2 which make the child seem deteriorated. There are
3 certain progressive degenerative disorders which need
4 to be picked out.

5 There is this regression in previously
6 nonautistic children, and I call it autistic
7 regression, which occurs typically, though not
8 exclusively, in the second year of life. Now, this
9 regressive aspect in autism actually has been known
10 for a long time.

11 Looking back on the first chapter I wrote
12 for the Menkes textbook, as I mentioned, maybe in
13 1974, somewhere in the 1970s, I briefly mention it and
14 actually I think give the same incidence figure, and
15 yet it hasn't been studied like so many other aspects
16 of autism.

17 In fact, autism literature is totally
18 enormous, and yet I am not aware of any single study
19 which studied a child with regressive autism during
20 the regression. I don't know how that is, but it's
21 not available.

22 Now, to me that's very important because for
23 a child neurologist if a child regresses something is
24 happening to the brain now. In other words, there's
25 an encephalopathy going on and one would want to study

1 it, but I can't find anything about it.

2 The studies that have been done, and there
3 are very few, are retrospective. Usually what is done
4 is autistic children are studied as a group and then
5 it's mentioned 30 percent of these were regressive or
6 whatever.

7 There have been a number of autopsies
8 published of individuals with autism who died for
9 various unrelated reasons, and these studies have been
10 important information about the organization and
11 deviances in the brains of autistic individuals, but
12 not one is characterized as being a child with
13 regression so that leaves one up in the air. Maybe
14 some were but it wasn't noted, or maybe none were.

15 So I'm saying at the end of all this with
16 the regressive aspect of autism, particularly as it's
17 understudied and needs enormous attention, for the
18 child neurologist something is going on. There is a
19 cause. What is it.

20 Q Is autism relatively rare?

21 A Well, it used to be so considered. When I
22 was young it was rare. Everybody knows about the
23 spectacular increase of autism diagnoses, so we now
24 have a condition that happens maybe one in every 200
25 or 300 children.

1 In fact, I mean, I see it. I hear of
2 children living down the street, around the corner and
3 in the next little part of my area of Boston who are
4 autistic. I used not to hear this sort of thing.

5 There's been a lot of debate about the cause
6 or causes of this spectacular increase, which by the
7 predicting of some people it's three times as many and
8 others 10 times as many. It certainly is a rate of
9 increase not paralleling any other condition that I'm
10 aware of, although some other conditions are also
11 increasing, but not to this extent at all.

12 Now, clearly although prima facie, the
13 condition is getting more common. I can't therefore
14 assume that all of it is actually the condition
15 becoming more common because maybe classifications
16 have changed. Maybe we're better at diagnosing. We
17 may be better at hunting down the cases for
18 ascertainment. Maybe we used to call them by other
19 names. I mean, there are a variety of very legitimate
20 cushions to take care of in coming to a final
21 conclusion about the degree of increase in the
22 diagnosis.

23 Now, my appreciation of the situation as it
24 is is that there are a number of factors that would
25 tend to amplify the apparent increase of the autistic

1 disorders, but it's not been shown at all to my
2 satisfaction that they account for all of it.

3 I think our responsibility is the opposite,
4 that when such a serious disease seems to be coming
5 more frequently, we have to assume as pediatricians,
6 as doctors, that it is becoming more frequent unless
7 it can be shown conclusively that actually it's not,
8 which that has not happened, so I think that although
9 the final answer is not yet at hand it is legitimate
10 to suppose that to some extent this diagnosis is
11 increasing and we need the reason for that.

12 Q Doctor, in your opinion the autism rate does
13 appear to be increasing?

14 A Well, that's not just an opinion. I mean,
15 that's a fact everybody accepts.

16 Q Doctor, you mentioned studies. Have there
17 been epidemiological studies that have sought out the
18 treat of autism?

19 A The what of autism? I'm sorry.

20 Q Sought out the cause of autism.

21 A The cause? Well, yes, indirectly.
22 Epidemiology actually doesn't give you causes. That's
23 not what it's there for, and you can't infer a cause
24 of anything from just epidemiology.

25 What it does is to look for associations,

1 and then other scientific methods can further explore
2 those associations to determine whether they're causal
3 or coincidental or maybe they have some third cause
4 for the relationship. That's not the epidemiology
5 anymore.

6 There have been numerous epidemiological
7 studies with a variety of outcomes, but not ones
8 necessarily relevant to the matter at hand.

9 Q When you say not necessarily relevant to the
10 matter at hand, have you found any study that has
11 evaluated whether the combination of thimerosal and
12 MMR could cause or was an association with autism?

13 A No. That hasn't been studied. At least it
14 hasn't been published to my knowledge.

15 Q So there's nothing in the literature?

16 A No, and there's actually very little that
17 studies regressive autism, as I mentioned before, as
18 opposed to, as it were called, any old autism, so it's
19 hard to know really whether the results of the overall
20 study reply to the group of children we're interested
21 in here.

22 Certainly there's been no study that has
23 looked at a subgroup consisting of autistic children
24 who regressed who have inflammation of the gut, which
25 is pertinent to Michelle Cedillo, so for the single

1 case, the particular child on whose behalf we're
2 assembled, I couldn't find anything in the literature
3 to really help me.

4 Q So, Doctor, to be perfectly clear there's no
5 study out there that looks at the combination of both
6 thimerosal and mercury and its potential association
7 with autism?

8 A The combination of thimerosal and MMR?

9 Q Yes.

10 A No. If there is, I haven't seen it.

11 Q And there is no study that looks at
12 regressive children at all?

13 A Just recently, as one of the advantages of
14 the current enormous interest in autism, there's been
15 a wonderful increase in research efforts, and there
16 have been one or two articles that have looked at
17 regressive autistic individuals as compared to the
18 ones who are nonregressive on a basis of a variety of
19 variables, and there are some similarities and some
20 differences.

21 Q And that's the one study that you found that
22 evaluated regressive autism?

23 A Well, there's one study that was quite
24 interesting that looked at the prevalence of minor
25 congenital anomalies in autistic children as compared

1 to typical children and in early onset autistic
2 children versus regressive autistic children.

3 Minor congenital anomalies are what they say
4 they are, little changes in the features or maybe a
5 so-called Simian crease, an abnormal crease across the
6 palm of the hand or the eyes being wideset or
7 slanting.

8 There's a set of markers which are generally
9 taken as indicating something went wrong -- not
10 necessarily serious, but something went wrong --
11 during pregnancy because that's when the features of
12 the face particularly are first developed.

13 In that particular study children with
14 autism had more of these than typical children
15 because, you know, even people in the general
16 population have some of these quite frequently, and to
17 have one or two is considered to be normal or within
18 normal range.

19 In this study, as I remember it, regressive
20 children didn't have those any more than the typical
21 children, which, to the extent one can interpret it,
22 would suggest that that would not support a prenatal
23 origin for them.

24 That's just a single observation, a single
25 study, and I wouldn't want to overemphasize its

1 ultimate importance, but I note it with interest.

2 Q Doctor, in your review of the literature did
3 you find any publication which looked at the
4 association of thimerosal and MMR in combination in
5 children who developed autism with GI problems?

6 A No.

7 Q Now, Doctor, you indicated that you reviewed
8 Michelle's early developmental records.

9 A Yes.

10 Q Can you tell the Court from your review of
11 the record whether Michelle was meeting her
12 milestones?

13 A She was on the slow side definitely. Well,
14 I know she was sitting independently by nine months
15 because we saw it on the video, and she was sitting
16 firmly and well. She was maybe not sitting until
17 shortly before then, and on the average a child will
18 sit at about six months or soon after. There is a
19 wide range of individual variation.

20 She is said to have crawled at nine months,
21 which is in one of the records -- I forget which --
22 and that would be typical. Incidentally, some
23 children don't crawl at all, and it's not noted as
24 being abnormal. She did it.

25 She's said to have walked by 16 months,

1 which is relatively late, though I have to admit that
2 my oldest of my seven children walked at 16 months and
3 was hilariously amused by his accomplishment. I can
4 still see him laughing as he toddled across.

5 I can't overstate the amount of individual
6 variability. Nonetheless, 16 months is slow. An
7 interesting remark in one of the consultant records is
8 that when she did begin to walk she walked very well.

9 As a matter of temperament, some children
10 are reticent, but others are more risk seeking. Some
11 children will start walking and fall on their faces
12 and start again and start again. They're the
13 adventure in walking. Other children really wait
14 until they're quite sure, as it were, that they can do
15 it properly, so maybe Michelle is one of those, but
16 bottom line she was slow.

17 Q Did she demonstrate any autistic behaviors
18 in her early childhood prior to the MMR?

19 A None are noted. There's nothing in the
20 medical record to indicate any of that, and I did
21 review the videotapes and I saw none of it.

22 Q Doctor, if she did manifest some symptoms of
23 autism in a child that age, what would you find?

24 A Could you ask that again?

25 Q Yes. I said if she did manifest symptoms of

1 autism in a child that age, what would you be looking
2 for?

3 A That's been studied systematically and
4 particularly by Dr. Gerald Dawson, in fact, a doctor
5 from Bonn's. Extensive literature. Two of the
6 articles of that group are included.

7 It's actually surprisingly hard to sort it
8 out when one does it in a controlled way, but my
9 impression is that the most telling sign is the child
10 doesn't respond to his or her name when called.

11 Then others are described such as the child
12 doesn't like to be held and arches away, doesn't
13 particularly invite interaction with herself by
14 others.

15 There's some description of reacting
16 unusually to stimuli, but what I just described is
17 something called tactile defensiveness is the term
18 that's used particularly by occupational therapists.

19 Q Now, Doctor, when you reviewed the video on
20 Michelle did she respond to her name prior to the MMR?

21 A Yes, she did.

22 Q And when you reviewed the video on Michelle
23 prior to the MMR did she arch away from contact with
24 people?

25 A She was smiling, looking at people, engaging

1 with them. She was a pretty cute baby, and it was
2 obvious from her family that they were enjoying her,
3 that they expected her to enjoy interaction with them.

4 Yes. I saw nothing the matter.

5 Q And when you reviewed the video did you
6 notice the interactions between her family members?

7 A Absolutely. It was very positive.

8 Q And when you reviewed the video was there
9 any indication that she was reacting abnormally to
10 interactions with other people?

11 A No.

12 Q So, Doctor, in your opinion prior to the
13 administration of the MMR was Michelle demonstrating
14 any tactile defensiveness?

15 A None at all.

16 Q Doctor, did you review the video after
17 Michelle had received her MMR?

18 A Yes, I did.

19 Q When you reviewed it after she had received
20 the MMR what did you notice?

21 A Well, many things really, but most striking
22 her happy expression was gone. Her happy expression
23 was gone. She looked abstracted or dejected. She
24 seemed preoccupied. I didn't see interaction.

25 I mean, the family was trying to photograph

1 her in certain circumstances, typically interacting
2 with family members, and that didn't seem to succeed
3 really after age 16 months. She was sort of busy
4 doing something on the side, and she'd be keeping on
5 doing whatever it was in this repetitive fashion.

6 There was one video which was very, very
7 sad, although perhaps it was a bit later -- I don't
8 remember the time of it -- where a therapist comes to
9 the home to work with her. She totally panicked. You
10 see her rushing through the room making these
11 agitated, repetitive movements, sort of tumbling away.

12 I mean, she seemed like she was in a total
13 panic because a stranger came in who presumably, being
14 a professional, didn't behave in any inappropriate
15 manner. It was just a different personality
16 altogether.

17 Q Did you review any part of a video where
18 Michelle's name was being called?

19 A Oh, yes. At that point, I mean, she just
20 ignored it. Yes, I did.

21 Q And when you reviewed that portion of the
22 video did Michelle utter any sounds at all?

23 A I didn't hear her do it, and I understood
24 from history that she was silent.

25 Q In any discussions with Mrs. Cedillo did

1 Mrs. Cedillo indicate whether or not Michelle still
2 had speech after these high fevers?

3 A She doesn't have much of any, no.

4 Q Now, Doctor, you've reviewed the medical
5 records. Is that true?

6 A Yes.

7 Q And in your review of the medical records
8 did you find that there was a positive measles gut
9 biopsy?

10 A Yes, I did.

11 Q Doctor, what is measles?

12 A Measles is a virus. It's an infective virus
13 which causes the familiar condition of measles. We
14 call that a wild virus, which is well known to people
15 as one of the childhood infectious disorders which
16 fortunately, because of vaccination, isn't seen as
17 much anymore.

18 Now, from the point of view of neurology --
19 well, first of all, the measles virus itself has the
20 following characteristics which are element to
21 Michelle's case and many others of course too that it
22 is lymphotropic. It can initially as it were
23 accumulate in lymph nodes, and that's where it exerts
24 its effect on the immune system because it is well
25 known to be immunosuppressive.

1 It is also enterotropic, meaning that it
2 tends to head for the lining of the gut, and it is
3 neurotropic that has an affinity for nervous tissue.
4 In that respect there are some diseases of the brain
5 which can complicate the measles.

6 The most important of these and what used to
7 be the most frequent is measles encephalitis, which is
8 a disorder of the brain which comes on within a week
9 or two of an attack of measles and is thought to be
10 autoimmune and can be devastating or deadly. In fact,
11 my understanding is that maybe the major rationale for
12 measles vaccination is to stop this happening, and it
13 has enormously decreased in frequency.

14 Now, there are much rarer other conditions
15 which take a much longer time before they begin to
16 occur. One is called MIBE, and that's a condition in
17 which it's called IB because of inclusion bodies,
18 which really means that you can see various particles
19 by special methods in cells in the brain.

20 MIBE would occur a number of months after a
21 vaccination, a number of months after the wild measles
22 infection. However, I did include in my bibliography
23 and submitted to the Court an article by Bitnun,
24 B-I-T-N-U-N, in which a case of MIBE occurred eight
25 and a half months after measles vaccination, and at

1 autopsy they were able to recover the virus and
2 sequence it and show that it was vaccine strain.

3 So in this rare example the measles vaccine
4 caused that, so it hung around for about eight months.
5 It was able to persist. Normally the measles virus
6 would be cleared within four to six weeks, but
7 obviously not in this case.

8 And then well known, but also very rare, is
9 SSPE, subacute-sclerosing panencephalitis, and that
10 has a latency of anything from eight years to 30
11 years, but then when it recurs it's been well
12 described after a slow, long progress.

13 Q Now, Doctor, is there a period of time after
14 measles when one would expect to see complications
15 associated with measles?

16 A Well, that depends on the complications.
17 There is a period of time when the virus is in the
18 blood and before the system has cleared it where acute
19 complications would presumably occur.

20 Ideally one wouldn't expect to see any after
21 say six weeks because ideally the immune system would
22 have dealt with the problem. If one sees
23 complications arising from measles virus later then
24 the measles must have persisted to that point, which
25 means that the immune system failed to eject it or

1 destroy it, which does indicate some lack of
2 competence on the part of that particular individual's
3 immune system.

4 Q Are you saying that for those people who
5 have developed persistent measles infection, such as
6 in MIBE and SSPE, that the reason that measles persist
7 is because of some sort of immune dysfunction?

8 A Well, the immune system manages to get rid
9 of the measles virus in countless millions of people
10 because vaccination is very prevalent, and this is an
11 aberration that the virus remains.

12 Q Now, Doctor, going back to Michelle's
13 clinical history here, it's been noted that the fever
14 began roughly seven days after the immunization?

15 A Yes.

16 Q Did that coincide with any known properties
17 of the measles vaccine?

18 A Fever doesn't follow every MMR vaccination,
19 but when it does that's about when it might occur, the
20 second week after the vaccination.

21 Q And what would be the reason for that?

22 A Because in that timeframe the vaccine virus
23 is multiplying in the blood. It's called viremia.

24 Q And is that the point in time when you
25 expect to see the viremia to occur?

1 A Yes, and sometimes you even see a little
2 measles rash at that time because it's an attenuated
3 measles virus, but still a live virus and still has
4 the same properties, although much attenuated.

5 Q So Michelle's fever occurred at the right
6 timeframe in which you would expect to see a fever
7 after a measles immunization?

8 A Correct.

9 Q Doctor, you indicated earlier that measles
10 has a lymphotropic effect. What did you mean by that?

11 A The measles virus tends to migrate to lymph
12 glands, and actually it has an affinity for certain
13 cells within lymph glands called dendritic cells.
14 They've been mentioned before by some previous
15 testimony. Dendritic cells take part in developing
16 immunity to the measles and immunity to subsequent
17 exposure to the same virus.

18 In the biopsies of the gut lining in cases
19 where this inflammation occurred I understand that
20 more significant findings were in those little
21 collections of lymphoid tissue close to the mucosa of
22 the gut.

23 Q Now, Doctor, the lymph glands are part of
24 the immune system?.

25 A Yes.

1 Q Is that true?

2 A Yes.

3 Q We've heard testimony that measles is an
4 immunosuppressant. Is that true?

5 A Correct. That's well known.

6 Q Does the literature indicate at what point
7 in time that immunosuppression begins?

8 A I am not certain. I would defer to an
9 immunologist for the exact time. It certainly begins
10 very soon after the infection, but I can't give you
11 exactly.

12 Q Were you present for the testimony of Dr.
13 Kennedy?

14 A Yes.

15 Q Do you recall Dr. Kennedy indicating that
16 the period of immunosuppression began at approximately
17 one week after exposure?

18 A Well, you reminded me of it.

19 Q Doctor, Michelle's fever. It occurred
20 within the period of maximum viremia. Is that true?

21 A Yes.

22 Q And it also occurred at a time when the
23 immune system was starting to be affected. Is that
24 true?

25 A That would be correct.

1 Q And you indicated that it extends out to
2 roughly 14 days. Is that true?

3 A Yes.

4 Q And from seven to 14 days did Michelle have
5 recurrent fever?

6 A Yes, she did, and high fever. Unusually
7 high. The fever that you get after MMR tends not to
8 be that high.

9 Q Now, Doctor, you also indicated that measles
10 is a neurotropic virus.

11 A Yes.

12 Q What do you mean by that?

13 A It has an affinity for nervous tissue.
14 That's not to say it always gets there, but it tends
15 to settle in the nervous system if it has the
16 opportunity to do so.

17 Q And you indicated also that it was
18 enterotropic. Is that correct?

19 A Yes.

20 Q What does that mean?

21 A That it tends to settle in the lining of the
22 gut.

23 Q Doctor, when you look at Michelle's case
24 does she have an enterotropic problem?

25 A Well, she has an enteritic problem, which is

1 consistent with an enterotropic agent.

2 Q And has an enterotropic agent been recovered
3 from her gut tissue?

4 A Yes.

5 Q And what was that enterotropic agent?

6 A Measles virus.

7 Q Doctor, does Michelle have a neurological
8 disorder?

9 A Yes, she does.

10 Q And what is that neurological disorder?

11 A Autistic disorder.

12 Q Doctor, when you reviewed the literature and
13 you were looking at the literature for viral
14 persistence did you find any literature which
15 indicated how viruses could persist in the brain?

16 A You're saying the brain?

17 Q The brain or the nervous system.

18 A Right. Yes, there was some.

19 Q And what in the literature did you find?

20 A Well, the dominant way for virus to persist
21 is within cells, so they could be within the cells in
22 the brain, and just for clarity let me go with the
23 distinction that there are two main categories of
24 cells in the brain, the neurons and the glia.

25 The neurons, of course, do the basic work of

1 the brain and to control organs. The glia are more
2 like connective tissue cells, including cells of the
3 immune system as it manifests in the brain.

4 SPECIAL MASTER HASTINGS: Doctor, the second
5 type? Can you repeat that?

6 THE WITNESS: The glia, G-L-I-A.

7 SPECIAL MASTER HASTINGS: Okay. Thank you.

8 THE WITNESS: So you have, for example, the
9 astroglia, which is because they're shaped like stars
10 allegedly, and they are very, very prevalent in the
11 brain. They're in between the neurons, scattered in
12 among the neurons.

13 Then there are two important subsets. One
14 are called the oligodendroglia. They are the cells
15 that manufacture the fatty sheaths along the axons,
16 the so-called myelin, which makes it possible for
17 neurons to communicate with each other at long
18 distance.

19 The other are the microglia, which are the
20 immune cells. These are the cells that become
21 activated if there is an immune challenge. They are
22 part of the so-called innate immune system, which was
23 described by previous testimony.

24 SPECIAL MASTER VOWELL: Doctor, did you say
25 microglia or macroglia?

1 THE WITNESS: Micro, M-I. Yes, that's
2 right.

3 MS. CHIN-CAPLAN: Doctor, I'm going to refer
4 you to your report on page 11. Your report.

5 If I could just have a moment, Special
6 Master?

7 (Pause.)

8 BY MS. CHIN-CAPLAN:

9 Q Page 11 of your report, Doctor.

10 A Yes.

11 Q Are you there?

12 A I am on what alleges to be page 11, yes.

13 Q Doctor, in your report on page 11 you
14 indicated that you reviewed an article by Dr.
15 Oldstone. Is that true?

16 I'm referring the Court to Petitioners'
17 Exhibit --

18 SPECIAL MASTER HASTINGS: Sixty-one.

19 MS. CHIN-CAPLAN: -- 61-DD. DD.

20 SPECIAL MASTER HASTINGS: DD? Okay. I see.
21 You want to go to the exhibit? To the tab? Okay.

22 MS. CHIN-CAPLAN: Yes.

23 SPECIAL MASTER HASTINGS: Great.

24 BY MS. CHIN-CAPLAN:

25 Q Doctor, did you review an article by Dr.

1 Oldstone?

2 A Yes. I have a reference to it in front of
3 me under the subheading of Viral Persistence and
4 Disease.

5 MS. CHIN-CAPLAN: May I just approach the
6 witness, Special Master?

7 SPECIAL MASTER HASTINGS: Go ahead.

8 MS. CHIN-CAPLAN: I'm afraid I'm going to
9 have to ask questions over his shoulder.

10 SPECIAL MASTER HASTINGS: Okay.

11 BY MS. CHIN-CAPLAN:

12 Q Doctor, what is the title of this article?

13 A *Viral Persistence, Parameters, Mechanisms*
14 *and Future Predictions.*

15 Q And who is it authored by?

16 A Michael B.A. Oldstone.

17 Q Are you familiar with Dr. Oldstone's
18 reputation within the community?

19 A Yes. It's a very high reputation.

20 Q Doctor, under Introduction would you kindly
21 read to the Court that first paragraph by Dr.
22 Oldstone?

23 A Yes, ma'am. "One of the remarkable advances
24 in modern virology is the realization that persistent
25 viral infections exist and are common. Hence,

1 understanding the principles by which persistence is
2 initiated and maintained, as well as the pathology
3 consequences of continued virus replication in a host
4 over its life in terms of causing disease, provides
5 research areas of high significance, as well as
6 opportunities for challenging investigation."

7 Q Doctor, does Dr. Oldstone later on in this
8 article indicate how viruses can persist?

9 A Yes.

10 Q Could you tell the Court what Dr. Oldstone
11 indicates?

12 A Well, what he says is the host immune
13 response --

14 SPECIAL MASTER HASTINGS: Are you going to
15 read from something?

16 THE WITNESS: Well, it's actually a
17 continuation from the next paragraph from where I
18 read.

19 SPECIAL MASTER HASTINGS: The very next
20 paragraph?

21 THE WITNESS: Yes.

22 SPECIAL MASTER HASTINGS: Okay. Fine.

23 THE WITNESS: I'm sorry. I should have
24 said. Let me just read the whole of it.

25 "The key foundations upon which the

1 understanding of persistent infection rests are first
2 that the host immune response fails to form or fails
3 to purge virus from the infected host. Thus, viral
4 persistence is synonymous with evasion of the host's
5 immunological surveillance system. Recent advances
6 have shed light on the cellular and molecular players
7 involved. Secondly, viruses can acquire unique
8 components as to factors of replications."

9 BY MS. CHIN-CAPLAN:

10 Q And does that sentence continue on by
11 saying: "Viruses can regulate expression of both
12 their own genes and host genes to achieve residence in
13 a nonlytic state within the cells they infect."

14 A Correct. The same nonlytic state means that
15 the virus is inside the cells, but the virus does not
16 destroy the cells. In other words, the virus lives
17 within a cell that remains intact, although its
18 function is likely to be impaired.

19 Q Doctor, you were in here for the testimony
20 of Dr. Kennedy?

21 A Yes.

22 Q Do you recall Dr. Kennedy saying that a
23 virus could persist in a cell?

24 A Yes.

25 Q Do you recall his testimony that if the cell

1 is destroyed that is the way the virus can no longer
2 persist in the cell?

3 A Right. In that way the virus is really like
4 a suicide bomber. It destroys the cell and itself at
5 the same time.

6 Q Doctor, what was the third conclusion, the
7 third point that Dr. Oldstone wanted to make in his
8 article?

9 A Yes. This is now on the same page, and it
10 begins at line 5 on the right. "Third, the type of
11 disease that persisting viruses cause are often novel
12 and unexpected."

13 Q Thank you, Doctor. Doctor, in this article
14 too did Dr. Oldstone mention the measles virus at all?

15 A I'm sure he did. It was mostly using the
16 LCMV virus for illustration, but he certainly had
17 worked extensively with the measles virus. Here's a
18 mention here.

19 SPECIAL MASTER HASTINGS: You need to speak
20 up a little, Doctor. We're not quite getting it.

21 THE WITNESS: I'm sorry.

22 SPECIAL MASTER HASTINGS: You need to speak
23 up. Your last two sentences we couldn't hear.

24 THE WITNESS: I'm sorry. I tend to mutter
25 to myself.

1 The mention of the measles virus, for
2 example, in the caption to Figure 2 on page 115, but I
3 would have to re-read the article to see specifically
4 what he said.

5 MS. CHIN-CAPLAN: Thank you.

6 BY MS. CHIN-CAPLAN:

7 Q So, Doctor, after reviewing that
8 introduction is there a take away message from this
9 introduction?

10 A Yes. What I glean from it is that more
11 disease and more diseases are probably caused by
12 viruses persisting inside cells, including cells of
13 the nervous system, than we currently appreciate and
14 that a mechanism that invokes viruses persisting in
15 cells is a biologically plausible, medically
16 reasonable mechanism to invoke when trying to explain
17 disease.

18 Q Doctor, were you here for the testimony of
19 Dr. Byers?

20 A Yes.

21 Q And did you hear Dr. Byers speak of the
22 effect of systemic inflammation on the blood-brain
23 barrier?

24 A Yes.

25 Q Did you hear her testimony about -- strike

1 that.

2 Could you kindly tell the Court what Dr.
3 Byers indicated was the effect of systemic
4 inflammation on the blood-brain barrier?

5 A The inflammatory process generates or is
6 fueled by and disperses into the circulation
7 proinflammatory cytokines, and proinflammatory
8 cytokines circulating from the locus of inflammation
9 when the circulation reaches the blood vessels that
10 irrigate the brain, which is where the blood-brain
11 barrier is located, these cytokines are capable of
12 breaching the blood-brain barrier such that the brain
13 is no longer protected from larger protein particles
14 which could be infected.

15 Q Doctor, once the infection breaches the
16 blood-brain barrier do you recall or do you know what
17 happens within the central nervous system?

18 A Well, once the infection has caused the
19 blood-brain barrier the infectious agent might be
20 found in the cerebrospinal fluid. If it is found in
21 the cerebrospinal fluid it's a virtual certainty that
22 it's also in the brain itself.

23 Q And to your knowledge is there a specific
24 area of the brain that the infection would affect?

25 A It depends really on the infectious agent.

1 For example, the herpes virus has an important
2 affinity for the medial temporal lobes. People who
3 survive a severe herpes virus encephalitis attack will
4 tend to often have severe memory problems because
5 that's one of the things that area does.

6 Other virus infections are less specific.
7 For example, in SSPE I don't believe there is a
8 specific area that is affected more than others, but
9 rather the infection spreads from neuron to neuron and
10 so it differs.

11 Q Doctor, I'm going to refer you to
12 Petitioners' Trial Exhibit No. 9, page 38. This is
13 one of the slides that was presented to Dr. Byers or
14 that Dr. Byers presented to the Court rather.

15 Could you read the title of this slide to
16 the Court?

17 A The title is Effect of Inflammation on the
18 Microglia of the Brain. It is derived from an article
19 by Qin, Q-I-N, et al. in 2007.

20 SPECIAL MASTER HASTINGS: This is page 38 of
21 Dr. Byers' Trial Exhibit 9?

22 MS. CHIN-CAPLAN: Correct, Special Master.

23 BY MS. CHIN-CAPLAN:

24 Q Doctor, what are microglia?

25 A The microglia are glial cells, as I

1 explained, not new ones, and they are part of the
2 innate immune system as it is represented in the
3 brain.

4 Q And what do they do as part of the innate
5 immune system of the brain?

6 A Well, when they're activated by a foreign
7 antigen, something entering their space which they
8 aren't acquainted with as it were, they will do what
9 it says in this abstract, which is they would increase
10 the expression of brain proinflammatory factors.

11 In other words, they will cause cytokines to
12 be released which cause inflammation. Several of
13 these are named in the abstract.

14 Q Doctor, does that mean that they're the
15 immune system of the brain?

16 A Yes.

17 Q Doctor, when somebody has a localized
18 infection can it become systemic?

19 A Yes, indeed.

20 Q And when I say systemic, could you tell the
21 Court what that means?

22 A Well, the infection can be localized or be
23 in an abscess which has a fibrous wall around it so
24 the infection can't spread beyond it. On the other
25 hand, nothing can get in it to stop it happening.

1 Antibodies can't get in.

2 However, if the walls rupture or the wall
3 doesn't form then the infection can spread. It can
4 either spread locally to neighboring territory within
5 the affected organ or it can spread by the blood,
6 circulation, and when it's in the blood it's called
7 septicemia when it's bacteria anyway. Then it can
8 spread to any organ in the body, of course, because
9 all organs of the body are irrigated by blood vessels.

10 Q You indicated that it can spread to all
11 organs of the body. Does that include the central
12 nervous system?

13 A It certainly does.

14 Q Does the brain have a certain amount of
15 protection from infection?

16 A Well, the blood-brain barrier, when it's
17 intact, protects it to quite an extent.

18 Q When there's a systemic infection, what
19 effect does that have on the blood-brain barrier?

20 A Well, as described, it can cause breaches in
21 the blood-brain barrier at which point the protection
22 is no longer present.

23 Q So any infectious agent could come in and
24 infect the brain at that point?

25 A In principle, yes.

1 Q Doctor, if there was an infectious measles
2 virus in the body would it be able to breach the
3 blood-brain barrier?

4 A It could be able to pass through a breached
5 blood-brain barrier.

6 Q And once within the brain what would happen
7 then?

8 A Well, it would settle in those cells for
9 which it has affinity, and it actually might very well
10 settle in the glial cells, the astroglia and the
11 microglia, though it can also settle in neurons, but
12 it would enter cells.

13 Q Doctor, after your review of the records and
14 the relevant literature, do you have an opinion
15 whether the measles RNA which was found in Michelle's
16 gut tissue was a substantial contributing factor in
17 the onset of her autism?

18 A I have the opinion that the measles vaccine
19 virus, traces of which were discovered in the form of
20 measles RNA, was a substantial factor in the causation
21 of Michelle Cedillo's autism.

22 Q And could you tell the Court what the basis
23 of your opinion is?

24 A Yes. The fact of the presence of the
25 measles vaccine virus, and I say measles vaccine virus

1 because she was vaccinated and because she has no
2 record of having had wild measles and because in my
3 experience in the literature there are similar cases
4 where the virus material has been sequenced and shown
5 to be a vaccine virus.

6 The measles vaccine virus in her case was
7 not rejected by her immune system, what was able to
8 persist, and was therefore a potentially neuropathic
9 agent harbored in her body, and when one keeps that
10 fact in mind in relation to the fact that she did have
11 an otherwise unexplained encephalopathy in the form of
12 the regression that we have already discussed then in
13 my opinion that is the number one suspect for the
14 cause of the regression or the encephalopathy which
15 implemented the regression leaving her in the state in
16 which she remains.

17 Q Doctor, as part of your opinion have you
18 considered the potential mechanism by how this could
19 have occurred?

20 A When you say "this" are you talking about
21 the brain damage?

22 Q Correct.

23 A Yes. Yes, I have.

24 Q Could you tell the Court the opinion you
25 have formed?

1 A Well, I think I should actually divert into
2 a brief account of what the present state is of
3 knowledge of how the brain looks in a case of autism.

4 Q Okay.

5 A Because there are a number of studies
6 pertaining to that.

7 From the point of view of the structure of
8 the brain, there are a number of findings. One
9 finding is that in many cases the cells in the
10 hippocampus, which is part of the medial temporal lobe
11 of the cortex, and the cerebellum and also sometimes
12 in the medulla are disorganized. They're not lined up
13 in the appropriate way.

14 The big cells in the cerebellum called the
15 purkinje cells, P-U-R-K-I-N-J-E, are deficient,
16 impaired, but there's also information that the
17 organization of the gray matter of the cortex itself
18 is impaired.

19 The picture is that the gray matter, which
20 is the cortex, is sort of on the outside of the
21 cerebrum, and it has the neurons in it. The inside is
22 the white matter, which is just a communication
23 system.

24 Now, the cells in the gray matter are
25 arranged in columns at right angles to the surface so

1 it's a columnar arrangement. It's a very orderly
2 arrangement of columns all over the cortex. A
3 disorganization of these columns has been described in
4 autopsy material of the brain.

5 Another finding which has been achieved by
6 MRI, by neuroimaging, is that in the younger cases --
7 and these things can change over the years -- the area
8 of the brain which is white matter which lights up in
9 that fashion in the scan is greater, more voluminous
10 than it should be, particularly in the area behind the
11 frontal lobe. This is work particularly by Dr. Martha
12 Herbert, which is represented in my bibliography.

13 Now, the nature of that enlargement of the
14 white matter is unclear. It could be that there are
15 more fibers, but that's not thought to be likely. It
16 could be the fibers are swollen, or it could be that
17 the spaces between the fibers have increased and maybe
18 because of inflammation and edema fluid and it's not
19 been resolved.

20 Now, in addition to the study of the static
21 organization of the brain in autistic individuals,
22 there's also been some work on the function of cells
23 in the brain of autistics as it deviates from what is
24 expected in normal cases. And the chief publication
25 is one by Vargas, V-A-R-G-A-S, et al. from Johns

1 Hopkins. It's a very important article. This
2 incidentally is the group to which Dr. Zimmerman, whom
3 we'll hear from later in the proceedings, he's a
4 member of that group.

5 Now Vargas, et al. presented two types of
6 information, one type on autopsy material of
7 individuals with autism who had died and one on
8 cerebral spinal fluid taken by lumbar puncture from
9 autistic individuals who were of course alive.

10 Now, in their examination of the tissues of
11 the brain at autopsy they noted areas of inflammation.
12 This is a very important observation in my view
13 because it is an indication that autism, at least in
14 many cases, isn't what it had been considered to be.

15 It had been considered to be a static
16 encephalopathy as it were. Something went wrong maybe
17 in the first trimester of pregnancy and then the
18 individual is left with an abnormality of development
19 which then remains for the rest of their life.

20 This indicates rather that the individual
21 who is autistic has in his or her brain an ongoing
22 disease. There's an active process smoldering over
23 the years, and I think the notion that autism is more
24 than a disorder, it's a disease, is a very important
25 one. There are numbers of sources of indications that

1 it is an appropriate concept for many autistic
2 individuals.

3 Now, the information was of the type that
4 characterizes a response of the innate immune system,
5 and what was found in particular was the involvement
6 of the astroglia and the microglia. The microglia
7 were discovered activated, which means that they were
8 actively responding to the presence of some agent that
9 they considered foreign.

10 Now, you can have microglia activation in a
11 variety of circumstances, and the interpretation is
12 not always the same. The main distinction is that the
13 microglia may be activated in response to an invading
14 agent, or an invading agent might have destroyed
15 neurons and the microglia may respond to the breakdown
16 product of the neurons, which is also foreign
17 material, so you can't immediately determine which is
18 the case.

19 This case is in point. You get microglia
20 activation in Parkinsonism and in Alzheimer disease,
21 and the interpretations are debated. Certainly the
22 response of the microglia in these autistic brains is
23 compatible with the notion that there is a foreign
24 agent to which they are responding.

25 The fact that this response was the case at

1 autopsy suggested it had been going on a long time,
2 which means that this response was not effective in
3 removing the invading agent. In other words, it must
4 still be sitting in the cells so this is what Dr.
5 Oldstone called noncytolytic.

6 The virus is in the cells. It stimulates an
7 immune response against it. That immune response is
8 not effective, but that immune response can
9 nonetheless injure itself in the neighborhood, sort of
10 bystander cells. There is other evidence of the
11 gliosis of astrocytes, meaning scarring of the
12 astrocytes, which suggests that the astrocytes were
13 being inactivated or destroyed by an immune attack.

14 Why is this important? Well, one reason why
15 it might be important, and there's certainly I'm sure
16 other interpretations, has to do with one of the
17 important functions of astrocytes. To explain this
18 again, that's a segue for a moment into some more
19 general comments.

20 In the human brain there are so-called
21 neurotransmitters. These are chemical messengers that
22 assist in communication between neurons and other
23 neurons, and they're of two kinds. There are the
24 excitatory ones where the receiving neuron is
25 activated by the message that it gets, and there are

1 the inhibitory ones where the receiving neuron is
2 inhibited by the message that it gets.

3 SPECIAL MASTER HASTINGS: What was the
4 second one? The first one was excitatory?

5 THE WITNESS: Correct, sir.

6 SPECIAL MASTER HASTINGS: And the second one
7 was?

8 THE WITNESS: Inhibitory.

9 SPECIAL MASTER HASTINGS: Inhibitory?

10 THE WITNESS: Correct. The inhibitory, as
11 the name suggests, if a cell receives an inhibitory
12 message it will tend to diminish its firing rate.

13 It's essential in the brain that there be a
14 balance maintained between excitation and inhibition.
15 If there's too much inhibition the business of the
16 brain can't proceed. If there's too much excitation
17 then it also can't proceed and seizures may occur, for
18 example.

19 I should mention, because I haven't already,
20 that within the last few years Michelle has initiated
21 a serious, severe seizure disorder which does happen
22 in severe cases of autism and perhaps more frequently
23 in the regressive type than in the standard type.

24 Now, the way that the housekeeping of this
25 inhibitory/excitatory balance is maintained does

1 involve the astrocytes.

2 SPECIAL MASTER HASTINGS: It involves the
3 what?

4 THE WITNESS: The astrocytes.

5 SPECIAL MASTER HASTINGS: The astrocytes?

6 THE WITNESS: Right. Those glial cells that
7 I was mentioning as being affected by the inflammation
8 in the Vargas study.

9 SPECIAL MASTER HASTINGS: Okay. But these
10 are different from the astroglia?

11 THE WITNESS: No. They are the same. I'm
12 sorry.

13 SPECIAL MASTER HASTINGS: They are the same?
14 It's another name for the same thing?

15 THE WITNESS: It is another name for the
16 same cell.

17 SPECIAL MASTER HASTINGS: Did you say
18 astrocells or astrocytes?

19 THE WITNESS: I said astrocytes, which
20 really means a star-shaped cell. It's just
21 descriptive. Astroglia indicates that they are part
22 of the glial system, but those two terms are
23 synonymous. I should stick to one of them.

24 Now, the most prevalent excitatory
25 neurotransmitter is glutamate, and it is of course

1 essential to the effective functioning of the
2 activities of the brain.

3 However, if there is an excessive glutamate
4 it will stimulate too much. Some of the areas will
5 simply fire too much, and that may blunt the
6 specificity of their response. If it's really too
7 much the glutamate itself can kill neurons.

8 There they are being what is called
9 excitotoxic. In other words, they activate the
10 neurons to the extent it kills them, and that's an
11 important area of study in child neurology in many
12 fields, not just in this one.

13 Now, one function of the astrocytes is to
14 mop up excess glutamate, so at a synapse, which is the
15 bridge between or the space between the end of one
16 neuron and the beginning of another, glutamate is
17 released into that little space. Some of it can
18 scatter sideways, and astrocytes will mop it up.

19 In other words, astrocytes have a functional
20 role in precluding overactivation, and if astrocytes
21 are destroyed or lacking then overactivation may well
22 occur. So now the next thing I need to address to
23 show the relevance of this is what is the relevance of
24 overactivation to autistic disorder.

25 I actually wrote an article more than 20

1 years ago -- it's my recollection it's in what I
2 submitted -- suggesting that many of the symptoms of
3 autism can be explained by an overactivation of
4 arousal systems in the autistic person.

5 SPECIAL MASTER HASTINGS: What kind of
6 system?

7 THE WITNESS: Arousal.

8 SPECIAL MASTER HASTINGS: Arousal. Okay.

9 THE WITNESS: But what it feels like to be
10 overaroused is what it feels like to be anxious, what
11 it feels like to be panicky, in suspense. It's that
12 kind of a feeling.

13 In my opinion, much of what autistic
14 individuals typically do is not a basic expression of
15 an abnormality, but they're attempts to deal with
16 their own overanxiety and arousal.

17 I've argued I think correctly that these
18 repetitive movements have a calming effect, which is
19 why the children do them and use them when they are
20 overexcited, such as when a stranger comes into the
21 house, for instance. This overarousal is the reason
22 for what I take to be the overriding characteristic of
23 the autistic state, which is an internal locus of
24 attention.

25 What to me best describes overall what it's

1 like to be autistic is a preoccupation with one's own
2 mind and one's own state and therefore a
3 disassociation from what's happening outside so that
4 autistic individuals are less occupied with, are less
5 concerned with, take less care about what's going on,
6 and only when circumstances force them do they turn
7 their attention outward, but preferentially they're
8 concerned with whatever is going on in their minds and
9 their feelings.

10 What I suggested was that that is because
11 they were constantly dealing with those feelings,
12 which really was overriding all other interests in
13 many cases. I might say that similar considerations
14 may apply to some cases of schizophrenia, which
15 certainly differ from autism in many ways, but have
16 some commonalities as well.

17 Recently an important article was published
18 by Rubenstein and Merzenich. Dr. Merzenich is a very
19 highly respected, well-known neurophysiological
20 investigator who has accomplished a lot of work in
21 neuroplasticity.

22 They argue along the lines that I've
23 described, but with a lot of neuroscience support for
24 the concepts, and they in fact argue that the autistic
25 state is a state of overexcitation, of this inhibited

1 excitation, and in one of the sections they discuss a
2 possible role of glutamate in setting up that state.

3 I am relying to some extent on that article
4 in the opinions that I've presented to the Court, so
5 this outlines my theory of the mechanism of causation.
6 To be totally clear, what I am providing here is what
7 I take to be a medically reasonable mechanism of
8 injury.

9 I'm not purporting that it is definitely
10 known that this is the case. I'm not testifying to a
11 scientific level of certainty, but to a level of
12 probability.

13 BY MS. CHIN-CAPLAN:

14 Q Doctor, to summarize it's your opinion that
15 the measles RNA which was recovered from Michelle's
16 gut tissue more probably than not caused her autistic
17 symptoms?

18 A Yes. RNA by itself doesn't cause anything.
19 The measles virus, of which the RNA was discovered,
20 is the agent that caused Michelle's symptoms.

21 Q Doctor, to briefly summarize, the manner in
22 which the measles virus caused Michelle's symptoms was
23 by what manner? Strike that.

24 Can you just summarize how this occurred?

25 A In synopsis, the measles vaccine virus in

1 this possibly quite unusual case was able to access
2 the brain, was able to invade neurons or I say brain
3 cells, astroglia, was able to stain those cells that
4 it had invaded without killing them, but did evoke a
5 vigorous response of the innate immune system against
6 them, and the inflammation that resulted from that
7 response disorganized critical circuits in her brain,
8 interrupted communication between various areas in
9 such a manner as to limit the type of mental
10 operations that she was able to perform.

11 Actually to add a gloss to that, to expand
12 that further, it's been observed by others and myself
13 that autistic individuals have more problems with
14 complex than with simple mental operations not just
15 because the complex are more difficult.
16 Disproportionately more problems.

17 One way of construing that is to say that
18 when one deals with a complex issue one has to use
19 many parts of the brain, and they have to
20 intercommunicate appropriately and be coordinated to
21 solve the puzzle, solve the problem, achieve the goal.
22 When one is doing something simple, and this is shown
23 in your imaging, only a small part of the brain is
24 involved. When you see there is some activation, this
25 has been clearly shown.

1 This communication between different parts
2 of the brain would not be expected to handicap so much
3 the simple operation, which is in one place, as the
4 complex one where communication is of the essence in
5 accomplishing the task.

6 This is the best I can do in describing the
7 type of injury which, in my opinion, is most likely to
8 impose on its victim the type of limitations that an
9 autistic child has.

10 Q Doctor, there's been mention of the U.K. MMR
11 litigation several times during this hearing. Is that
12 true?

13 A It is true.

14 Q Were you a consultant to this U.K.
15 litigation?

16 A Yes.

17 Q Could you describe the circumstances under
18 which you became a consultant?

19 A Yes. I received a phone call from a
20 solicitor.

21 MS. CHIN-CAPLAN: Special Master, it's
22 11:00. Should we just take a break before we start
23 this section?

24 SPECIAL MASTER HASTINGS: Yes. I was going
25 to ask you. You have a substantial amount more

1 direct?

2 MS. CHIN-CAPLAN: Yes.

3 SPECIAL MASTER HASTINGS: Okay. Let's take
4 a 15 minute break at this point. Thank you.

5 (Whereupon, a short recess was taken.)

6 SPECIAL MASTER HASTINGS: All right. We're
7 going to go back on the record here.

8 Ms. Chin-Caplan will continue her
9 examination of Dr. Kinsbourne. Go ahead, Ms. Chin-
10 Caplan.

11 MS. CHIN-CAPLAN: Thank you, Special Master.

12 BY MS. CHIN-CAPLAN:

13 Q I believe when we broke, Dr. Kinsbourne, you
14 were asked how you got involved in the U.K.
15 litigation.

16 A Yes. I received a phone call from the
17 solicitor.

18 SPECIAL MASTER CAMPBELL-SMITH: You've got
19 to speak up.

20 THE WITNESS: I'm sorry. I'm getting as
21 close as I can to this thing.

22 I received a phone call -- no?

23 SPECIAL MASTER VOWELL: That's the court
24 reporter's mic.

25 SPECIAL MASTER HASTINGS: We need both.

1 SPECIAL MASTER VOWELL: Both are important.

2 SPECIAL MASTER HASTINGS: Pull them both
3 over.

4 THE WITNESS: Yes, I know she matters too.
5 I'm sorry.

6 I received a telephone call from a solicitor
7 from London who told me about a very extensive class
8 action that was underway with respect to the possible
9 influence of the MMR vaccine on the development of
10 autism in a large number of children and also with
11 respect to other complications of the MMR vaccine such
12 as encephalitis, deafness, epilepsy.

13 He invited me to come as a consultant to
14 evaluate the evidence from the point of view of
15 pediatric neurology. It sounded interesting and
16 turned out to be remarkably interesting and certainly
17 the most extensive project that I've ever been in and
18 ever will be in.

19 So I went over there and met a number of
20 experts already engaged in this work, including some
21 very distinguished people, and for about four years
22 worked intensively on this project in a number of ways
23 which I'm able to describe if asked.

24 BY MS. CHIN-CAPLAN:

25 Q Doctor, did you fly over to London?

1 A On numerous occasions I flew to London. We
2 had meetings of the experts lasting two or three days.
3 Very intensive, very interesting and involving many,
4 many different disciplines and levels of analysis in
5 the biomedical fields.

6 These meetings were obviously preceded by
7 preparation. Every time one came back from a meeting
8 one had more work to do in follow-up. Communication
9 was continuous. My phone rang every day. My e-mail
10 came every day. My wife was disturbed with me every
11 day. It was a fascinating, disrupting thing that
12 lasted four years.

13 What did it involve? The type of activities
14 were, first of all, obviously review of files. There
15 were something like 1,000 claimants. I didn't review
16 all those files, but I reviewed well over 100, maybe
17 200.

18 Many of them were sent to me, to my home in
19 Massachusetts. Others I reviewed when I was in one of
20 my multiple visits to either London or Norwich, which
21 was the other place in which the meetings occurred.

22 Q Doctor, just to stop, you indicated you
23 reviewed hundreds of records?

24 A Yes. Yes.

25 Q Hundreds, you say?

1 A Yes.

2 Q Doctor, how many files did that involve? If
3 you could compare it to the files in back of the
4 Justice Department attorneys and compare that for the
5 Court?

6 A Well, actually I would compare those to the
7 apparent millions of research articles I got rather
8 than to the -- greatly in excess of this. My house
9 was occupied by this material.

10 Q You say greatly in excess?

11 A Greatly in excess.

12 Q Are you talking three times as much?

13 A Oh, easily. Easily.

14 Q Four times as much?

15 A Well, let me be more specific. At home I
16 had about 100 files or case records, you know, typical
17 case records like we have in this country.

18 I had I think it was 46 binders like this --
19 now, it may have been 44, but I think it was 46 --
20 with between 3,000 and 4,000 articles.

21 Q So, Doctor, you had you believe 46 binders?

22 A Yes.

23 Q I count the binders at the back of the
24 Justice Department attorneys, and I see 15.

25 A Oh, that's nothing compared.

1 Q So you had at least three times as many
2 files, patient files. Is that it?

3 A Yes. No, no. I'm talking now about the
4 scientific articles.

5 Q The scientific articles constituted 47
6 volumes?

7 A Yes. Forty-six, I believe.

8 Q Forty-six volumes?

9 A Now, they didn't all come at once. I mean,
10 this is over a course of four years, but yes.

11 Q How many patient files did you review?

12 A Well, I would say at home I reviewed about
13 100, and then I reviewed more -- I didn't count them
14 -- when I was over in the U.K.

15 Q And when you reviewed the patient files were
16 they as extensive as the files in back of the Justice
17 Department attorneys?

18 A Not all of them, but many of them were.

19 Q So there were multiple volumes of medical
20 records for these patients?

21 A Some patients had a single volume. Some had
22 more. It varies, as it always does.

23 Q And you reviewed hundreds of claimants'
24 medical records?

25 A Yes.

1 Q And you reviewed 46 volumes of scientific
2 literature?

3 A Right.

4 Q But you had more involvement than that? Is
5 that true?

6 A I had more?

7 Q You were more involved than just reviewing
8 medical records and reviewing scientific literature?

9 A Well, it's a different matter. With the
10 medical records one really wants more specific things
11 about the children, the diagnosis. One wants to see
12 what the syndrome is. One wants to determine and
13 wants to both learn from them and also ultimately
14 assist in the selection of the test cases, which is
15 what it finally came to.

16 With the literature one has problems to
17 solve. One refers back to the same article multiple
18 times, depending on the questions asked. We did
19 several series of intermediate reports. Not causation
20 reports, but reports on possible mechanisms of the
21 disease, and we would refer to articles.

22 We would share our writings with the
23 defense, and inevitably the defense would fail to
24 understand what we had said. In Britain they call
25 this for the avoidance of doubt. They would ask many,

1 many questions, interrogatories, and we would answer
2 them, and then they would ask questions about the
3 answers we had given to the interrogatories, and we
4 would answer those. That was an ongoing traffic that
5 kept us going.

6 Of course, we would discuss among ourselves
7 issues, and it was particularly my job more than that
8 of most to know not only what was going on at my level
9 of neurology, but what was going on at all the other
10 levels because all the other levels are the
11 infrastructure for my opinion so I need to be aware of
12 them and here and there assist.

13 Q So you had to discuss the other disciplines
14 that were involved in formulating an opinion. Was
15 that it?

16 A Absolutely.

17 Q Doctor, the Respondent showed this to the
18 Court.

19 MS. CHIN-CAPLAN: I'm afraid I don't have an
20 exhibit number, Special Master.

21 SPECIAL MASTER HASTINGS: I believe that was
22 Respondent's Trial Exhibit No. 6. It was marked as
23 such.

24 BY MS. CHIN-CAPLAN:

25 Q Doctor, I'm going to show you Respondent's

1 Exhibit No. 6. I kept on counting and losing count of
2 the number of people that were involved in this case.

3 This was for the Plaintiff. Was that it?

4 A That's correct. Everybody listed consulted
5 at various levels of intensity with respect to the
6 Plaintiff's case.

7 Q How did they end up with so many experts?

8 A Well, there are so many disciplines
9 involved, and there were multiple experts in each.
10 I'm sure the defense had at least as many.

11 Actually, although I'm anticipating, when it
12 comes to writing reports, which it finally came to,
13 there was an exact match between the number of reports
14 of Plaintiff and defense matched by the area of
15 interest, so there would be an equal number of
16 epidemiology experts, an equal number of immunology,
17 equal number of gastroenterology. They would be
18 matched. In the end I think it was something like 12
19 or 14 reports on each side

20 Q Did you say 40 report?

21 A No, no. We had all sorts of memos and
22 intermediate reports, but the formal filing which
23 occurred sometime in 2003 involved -- I may not
24 remember it correctly -- maybe 14 or 16 reports from
25 the defense, matching the same number of reports from

1 the Plaintiff.

2 Q But before those final reports came in the
3 Defendant had approximately an equal number of experts
4 as the Plaintiff did?

5 A I think that they probably had more, but I
6 don't know that.

7 Q So if the Plaintiff had at least 40 or 50,
8 the Defendant had approximately the same amount?

9 A Certainly at least, but we weren't privy to
10 that information. It was only at the time of the
11 reports that we found out, you know, who would be
12 writing them, and then we received the materials.
13 They of course waited until we had issued our reports.

14 Q Doctor, when you wrote your report were you
15 required to consult with the 40 to 50 experts that I
16 see on Respondent's Exhibit No. 6?

17 A Well, not required exactly. My involvement
18 would vary from person to person, but I had an
19 organizational role and interpersonal communication
20 role.

21 If a new expert were joining the group,
22 obviously the solicitor but I also would explain to
23 them, you know, what the issues were and the way of
24 proceeding, so in one way or another I certainly got
25 to talk to most of these people and some of them very

1 much.

2 Q Doctor, when you put together your report
3 did you consult with the other people on this
4 Plaintiff's list of 40 to 50 experts?

5 A Well, mine had to be the last to be
6 formulated. I was able to look at drafts of other
7 people's, you know, and then when I had made my
8 comments and also fully understood I would be finally
9 ready to write my report.

10 Q So you reviewed the reports of all of the
11 previous experts before you could draft yours?

12 A Yes.

13 Q Doctor, did you review the reports of the
14 Defendant experts as well?

15 A No. They came afterwards.

16 Q They came afterwards?

17 A Yes.

18 Q Doctor, I'm going to ask you to take a look
19 at this list. Doctor, who are these people?

20 A They're all different, you know, but one way
21 of answering the question is if I could just read off
22 the disciplines involved?

23 So we have neurology, we have
24 neuropsychology, immunology, micropathology, peptide
25 studies, epidemiology, histopathology, blood-brain

1 barrier, an excellent specialist in that area,
2 genetics, gastroenterology, pathology, virology. I
3 think those are the areas involved.

4 Q Doctor, the participants in this group of
5 experts for the Plaintiff, were they highly recognized
6 within their field?

7 A Oh, there were some very distinguished
8 people. For example, the immunologists included Dr.
9 Suissa and Dr. Shapiro, Dr. Montgomery, all excellent.
10 Actually there was another very excellent doctor,
11 Professor Spitzer, who was very well known. He
12 unfortunately died.

13 In peptide studies, Dr. Castagnoli is a very
14 recognized authority. Professor Menkes was there,
15 pediatric neurology, from UCLA. He's the editor of
16 the highest regarded textbook of child neurology.
17 Professor Marchulonis, a brilliant immunologist, made
18 important discoveries.

19 I don't know if you'd like me to go on, but
20 they're interesting people.

21 Q Dr. Byers was there?

22 A Very interesting people. Dr. Byers.

23 Q And the molecular biology field? Was there
24 an expert there? I notice the name of Dr. Tedder.

25 A That's right. In fact, Dr. Tedder, correct,

1 is considered one of the top virologists in the
2 country.

3 Q And when you say the country --

4 A In the U.K. Yes.

5 Q Doctor, you indicated that you had to speak
6 to most of these people before you could even draft
7 your report?

8 A Yes. Another one, by the way, is Dr.
9 Schoenfeld from Israel, who's very highly regarded in
10 immunology. I enjoyed speaking with him.

11 SPECIAL MASTER HASTINGS: Doctor, I think
12 your voice was dropping as you talked about that
13 witness list. Please speak up.

14 THE WITNESS: Yes. I was unfortunately
15 holding this paper between me and the microphone.

16 SPECIAL MASTER HASTINGS: Not a good idea.

17 BY MS. CHIN-CAPLAN:

18 Q Doctor, you indicated that you had to speak
19 to most of these people before you could draft an
20 opinion?

21 A Well, it's not that I had to, but it's that
22 I wanted to because I needed to because I need to know
23 what they were saying. You know, everybody has their
24 own opinion, and I can't represent what other people
25 say without having some contact.

1 Q So you had to incorporate the opinions of
2 some of these people into your final report?

3 A Well, in modern medicine in complex issues
4 there's no one discipline usually that's sufficient to
5 address a difficult problem.

6 I mean, we all rely upon each other, and
7 there's no good me making a statement which is not
8 realistic in terms of somebody else's discipline
9 because the disciplines don't recognize these boundaries.

10 Q Doctor, you indicated that when the legal
11 process began you had to answer questions. Was that
12 it?

13 A Are you referring to the interrogatories?

14 Q Yes.

15 A Yes. There was a barrage of to and fro that
16 came at regular intervals.

17 Q Doctor, these interrogatories, did they
18 concern the individual people or did they concern
19 science and medicine?

20 A They were about mostly science.

21 Q More science?

22 A Yes.

23 Q Doctor, when you answered your questions did
24 you then send those answers over to the Defendant?

25 A Yes.

1 Q And what happened then?

2 A There were more questions.

3 Q And you would answer those?

4 A To the best of my ability. Not just me. I
5 mean, we all did this, you know, so it went on.

6 Q And that process was the same? The
7 Plaintiff would submit it to the Defendant?

8 A Well, when they submitted some of their
9 reports we tried to also ask questions because it
10 seemed to be the thing to do, but we weren't as
11 energetic about it as the other side was.

12 Q So not only did you have to compile your
13 information; you had to review the other side's
14 information?

15 A Yes.

16 Q And you indicated that there were expert
17 meetings. Was that it?

18 A Oh, numerous. Regular, yes.

19 Q Were they across the ocean in the U.K.?

20 A Yes, they were. Well, actually a few times
21 the barristers came over to the Boston area to meet
22 with me, but mostly we met in the U.K.

23 Q Can you describe those meetings?

24 A Well, they were not unlike really most of
25 the scientific meetings on a smaller scale.

1 We would meet in a hotel and have regular
2 sessions starting say at 9:00 and break for lunch, you
3 know, except we wouldn't finish until very late in the
4 evening because people would be following and pursuing
5 various lines of thought.

6 It was actually very well organized and very
7 rewarding. There were formal presentations. There
8 were little groups, discussion groups. There was
9 spontaneous interaction, of course.

10 The lawyers actually had very little role in
11 that. I mean, they were some lawyers present, but it
12 was really a meeting of the experts.

13 Q And, Doctor, this formed part of your
14 educational training?

15 A Oh, yes. I mean, immensely.

16 Q Doctor, when you were first retained did you
17 provide an immediate opinion as to whether MMR could
18 cause autism?

19 A Not at all.

20 Q How long did it take you to form that
21 opinion?

22 A I would say about three and a half years.

23 Q Did you say three and a half years?

24 A Yes.

25 Q And did something happen that convinced you

1 that there was a relationship?

2 A Well, the piece of evidence which finally
3 made me able to enter an opinion, in the context of
4 all the previous evidence -- I mean, not just
5 individually, but in that context -- was the actual
6 retrieval of measles virus in genomic material from
7 the cerebrospinal fluid of three of the children in
8 the Plaintiff group.

9 Q You say the recovery of measles virus
10 genomic material in the CSF?

11 A Right. The same as we've been discussing in
12 the gut lining, but actually found in the CSF.

13 Q And would that be an indication that it was
14 present in the brain?

15 A Yes, it would.

16 Q Doctor, did this trial ever take place?

17 A No.

18 Q Do you know the reason why it did not take
19 place?

20 A I know the reasons that were given. Well,
21 the material reason was -- I have to explain the
22 system there. The British Government pays for people
23 to sue the British Government.

24 Q That's odd.

25 A And to sue its own public health service and

1 to sue the manufacturer, you know. It's a different
2 philosophy.

3 You see, class actions as such in the
4 British system are not practical because the firms
5 don't have the financial resources to mount major
6 efforts of the kind that we're describing, so there is
7 a commission currently called the Legal Services
8 Commission. It had a slightly different name when we
9 started.

10 Legal Services Commission grants funds to
11 Plaintiffs who otherwise wouldn't be able to proceed
12 for lack of funds on issues of major importance. Now,
13 as I understand the criterion, they will grant funds
14 if they believe that the action has a more than 50
15 percent probability of success. They will withdraw it
16 if they change that belief.

17 Now, the Legal Services Commission did in
18 fact withdraw funding some months after we had
19 exchanged reports and after the CSF finding, and as I
20 recall it it's my recollection -- I read this document
21 a long time ago, but as I recall it -- there were two
22 kinds of reasons given.

23 One reason was that in the evaluation of the
24 state of the action, and this is based on reports
25 rendered by the barristers on both sides periodically

1 as to the state of the action to the Services
2 Commission. They made separate determinations for the
3 enterocolitis and the autism. They determined that
4 the enterocolitis action had a more than 50 percent
5 chance of success, but the autism had a less than 50
6 percent chance of success.

7 They argued as follows: We have now spent
8 15 million pounds. A trial date had already been
9 fixed. If we went to trial it would cost us another
10 nine million. To proceed on the issue of
11 enterocolitis at that level of expense was not
12 justified in their opinion, so that was the reason for
13 suspending funding.

14 They had another reason as well. That
15 reason was to do with the fact that in developing the
16 evidence of the case it wasn't possible simply to rely
17 on the evidence already present in the year 2000. One
18 had to make new findings, which one could call
19 research, though obviously they weren't formal
20 research efforts, but such as the finding of the
21 measles virus material in the CSF.

22 That finding wasn't available when the
23 action began, and they suddenly discovered that they'd
24 been funding research, which apparently they hadn't
25 realized for the five years in which they'd been

1 funding research, and they said that really it should
2 be the Medical Research Council that should fund
3 research.

4 So those were the two reasons given. At any
5 rate, it never came for a decision. The case was not
6 heard. The case was not dismissed. The case ran out
7 of cash.

8 Q So if I understand correctly, the Legal
9 Services Commission didn't think that they should
10 spend nine million more pounds to determine whether
11 the children should receive compensation for their GI
12 problems?

13 A Correct.

14 Q And they also made the determination that
15 they were funding research for the past five years,
16 and they were no longer going to fund research because
17 it was the responsibility of the General Medical
18 Council to fund research?

19 A No. Sorry. Correct except not the General
20 Medical Council. That's a licensing body of the
21 Medical Research Council, MRC, which is the equivalent
22 of our NIH.

23 They said that there should have been
24 regular applications for funding for research
25 projects, and maybe there should, but nothing had

1 changed, you know. The way that we'd been proceeding
2 was the same all along.

3 Q So the funding for the research was
4 discontinued?

5 A Yes. It was discontinued in September of
6 2003.

7 Q And that was the reason the case was
8 dismissed?

9 A Yes, that's right.

10 Q Doctor, one last question. Are you
11 antivaccine?

12 A Oh, no. I think vaccination programs are
13 essential, critically important and to be supported by
14 all means. In fact, all seven of my children have
15 been fully vaccinated, including my three
16 preschoolers, and they all did fine.

17 MS. CHIN-CAPLAN: Thank you, Doctor. I have
18 no further questions.

19 SPECIAL MASTER HASTINGS: All right. Mr.
20 Matanoski, do you have any questions for this witness?

21 MR. MATANOSKI: I do, sir, but I almost
22 think that it might be better to take the lunch break
23 now because I will have probably some extended cross-
24 examination rather than going for a little while and
25 then starting again.

1 SPECIAL MASTER HASTINGS: Do you have any
2 idea what timeframe? I'm not sure why you don't want
3 to do some now and some after lunch. It's a bit early
4 for lunch, I think.

5 MR. MATANOSKI: I'll probably be going on.
6 I know that my last two lasted about two hours.

7 SPECIAL MASTER HASTINGS: Right.

8 MR. MATANOSKI: I'll probably go on about
9 that long.

10 We could start. Conceptually the
11 presentation of cross-examination would make better
12 sense, at least the beginning of the cross-examination
13 would make better sense, if it was heard as one piece
14 rather than broken up.

15 SPECIAL MASTER HASTINGS: All right. Let's
16 take our one-hour lunch break at this point.

17 MR. MATANOSKI: Thank you, sir.

18 SPECIAL MASTER HASTINGS: We'll start again
19 at I have 11:46. We'll start about 12:45.

20 (Whereupon, at 11:46 a.m., the hearing in
21 the above-entitled matter was recessed, to reconvene
22 this same day, June 15, 2007, at 12:45 p.m.)

23 //

24 //

25 //

26

1 quantify what that means in terms of the level of
2 confidence?

3 A Yes. The standard that I am instructed
4 prevails in these proceedings, which is a more likely
5 than not or about 50 percent standard.

6 Q I'm about to go through your chain of
7 causation in this case, and at each step of the way
8 I'd like you to state for me what your level of
9 confidence is in the matter that you had just
10 testified to, whether it is at that standard, about 50
11 percent; whether it's greater than that, let's say
12 you're fairly confident or very confident and it rises
13 above to say 60 percent.

14 Just give us a sense of when it hovers right
15 at that breakpoint of 50 percent and when it's greater
16 than that.

17 A I don't actually attach medical
18 probabilities to steps in an argument. I only
19 attach --

20 Q I'm having trouble hearing you.

21 A I don't attach probability ratings to the
22 steps of an argument because I look at the total
23 clinical picture before I make a diagnosis, so it
24 would be a meaningless exercise to do that.

25 Now, of course, that doesn't mean I won't do

1 it if you ask me to, but I warn you that whatever the
2 probabilities are if you take these steps in isolation
3 it's really unhelpful to how it looks when those steps
4 are taken in combination because in a clinical picture
5 the various features of a case interact with each
6 other, so cutting it up into pieces is medically
7 irrelevant.

8 Having said this, I'll be happy to do
9 whatever you tell me.

10 Q I'm sorry. Cutting it up into pieces? You
11 have a sequence here that you've just gone through on
12 direct --

13 A Yes.

14 Q -- where you explained the whole sequence --

15 A Correct.

16 Q -- that leads you to a conclusion --

17 A Correct.

18 Q -- that MMR vaccine causes autistic spectrum
19 disorder.

20 A Right.

21 Q We're going to go through that chain. At
22 each point I just want to know is this at the
23 breakpoint or is this particular part of your theory
24 fairly well accepted.

25 A And I agree to do that, sir. I have no

1 problem.

2 Q Thank you.

3 A I was just attempting to caution about how
4 that might reflect on my ultimate opinion.

5 Q Okay. So when we go through it you might
6 actually change your ultimate opinion?

7 A No. Anything could happen, of course. I
8 mean, we might have a Perry Mason moment.

9 Q I seriously doubt that, sir. Let's start
10 with one of the theories that has been laid out here,
11 but I didn't hear you speak on it this morning, and
12 that is the role of thimerosal in the causation chain
13 here.

14 A For that role I rely on Dr. Aposhian. I
15 have no independent opinion on it.

16 Q Okay. So you don't know what role it plays?

17 A No. I'm saying I rely on Dr. Aposhian for
18 that proposition. I'm not a toxicologist, and I
19 haven't reviewed this case with respect to forming an
20 opinion on that particular step.

21 Q So you offer no opinion there?

22 A Correct.

23 Q And the next step seems to me is the receipt
24 of measles, mumps and rubella vaccine. Now, at that
25 point when the child receives measles, mumps and

1 rubella vaccine what does the virus do, the measles
2 virus, which is the one we're interested in?

3 A What does the measles virus do?

4 Q Right, after the child has received the
5 vaccine.

6 A Well, initially it's in the tissues.

7 Q How does it get into the tissues?

8 A Through the needle.

9 Q Okay.

10 A And then bit-by-bit it's carried off by the
11 circulation, local circulation, into the systemic
12 circulation.

13 SPECIAL MASTER HASTINGS: Doctor, for some
14 reason I'm not hearing you as well as I did this
15 morning. I'm sure it's not just because it's cross-
16 examination.

17 THE WITNESS: Yes.

18 SPECIAL MASTER HASTINGS: Do the best you
19 can.

20 MR. SHOEMAKER: Could we use this wireless
21 microphone, the one that Sylvia was using this
22 morning? I think it's worth a try.

23 SPECIAL MASTER HASTINGS: Okay. Are you
24 qualified to set it up, Mr. Shoemaker?

25 MR. SHOEMAKER: No. And I'll take full

1 responsibility if I don't do it right. Is there any
2 way we can test this?

3 THE WITNESS: Testing.

4 SPECIAL MASTER HASTINGS: All right.

5 THE WITNESS: Thank you.

6 BY MR. MATANOSKI:

7 Q So you're at the point where the virus has
8 now entered the body. It's gotten into the local
9 system, and then you said it gets into the systemic
10 system.

11 A It is bit-by-bit carried away into the
12 circulation to set up the viremia.

13 Q Okay. So at the point of injection you have
14 it in the local area where it was injected, and then
15 bit-by-bit it's carried into the bloodstream? Is that
16 what's happening?

17 A Yes.

18 Q Okay. How long does it take before that
19 happens?

20 A I'm not a virologist. I don't know that.

21 Q And after it enters the bloodstream what
22 happens next?

23 A It circulates, and primarily it enters the
24 lymph glands and may actually enter those before it
25 enters the bloodstream through the lymphatic system.

1 In the lymph glands the major immune attack on the
2 virus is launched.

3 Q So the body actually starts trying to fight
4 it when it gets into the lymph glands?

5 A Right.

6 Q Okay. Then what happens? How long does it
7 take after the injection before it gets into the lymph
8 glands?

9 A I'm not a virologist. I don't know that.

10 Q After it gets into the lymph glands and the
11 body immune system starts fighting it, what happens
12 next?

13 A Well, on the one hand the virus is
14 circulating in the circulation. That's the viremia.
15 On the other hand it's being neutralized by the immune
16 attack, and the immune attack will be both humoral and
17 cellular so the virus will be attacked both when it's
18 free and when it's in cells. In conjunction, the two
19 systems will in almost every case eliminate the virus.

20 Q Now, when the immune system starts to
21 operate, when it's gotten into the lymph glands and
22 the immune system starts to operate, would we see any
23 symptoms?

24 A Well, we will see, for example, the rash
25 which would be generated by antibody-antigen

1 complexes.

2 In other words, the rash itself is a
3 manifestation of an infraction of the virus with the
4 immune defense against it, and we will see fever
5 through the cytokines released by the effects of the
6 virus.

7 Q And this is when the virus first enters the
8 lymph glands?

9 A I can't tell you the time. There is some
10 immune reaction already locally, which we can tell of
11 course by the swelling that may sometimes occur, the
12 local inflammation, swelling, redness, pain.

13 That would be by the innate immune system
14 that is the early responder, and then in the lymph
15 glands you would also get the adaptive immune system
16 formulating a more specific attack on the particular
17 antigen in this particular virus. Timeframes I can't
18 give you.

19 Q Okay. So you don't know when exactly that
20 would occur.

21 You talked about the immune system
22 responding. If we were to do some testing when a
23 person could see whether that happens, what would we
24 look for to see whether they had launched an immune
25 system response?

1 A Well, we would in theory need to look at
2 both arms of the immune system, of the adaptive immune
3 system, namely the humoral and the cellular. The
4 humoral is what is customarily tested in terms of the
5 antibody formation against the virus, although
6 actually it is the less important part of the
7 response, but easier to measure.

8 The more important defense is the cellular
9 because it's a virus into cells, and it is possible,
10 but expensive, to measure cellular immunity, and it's
11 not a process that I am personally expert in.

12 Q How confident are you up to this point? How
13 confident are you in what you just stated?

14 A I am fairly confident both in what I know
15 and what I don't know.

16 Q Yes, and I know you've been very careful to
17 tell us what you didn't know.

18 From what you've stated that you do know, is
19 that at the 50 percent level of confidence, or is that
20 much higher than that?

21 A I would say it's higher.

22 Q Now, you said that the standard measure that
23 you would take to see whether the body mounted an
24 immune response would be to test the humoral part or
25 the antibody response. Is that right?

1 A Yes.

2 Q And what would you expect to see if the body
3 mounted a successful attack on the measles virus?

4 A You would expect to see the presence at what
5 I take to be satisfactory levels of antimeasles
6 antibody.

7 Q And in terms of that part of your opinion,
8 what level of confidence do you hold to that?

9 A I'm pretty confident of that.

10 Q Now, what does the virus do at this point?

11 A At which point?

12 Q Where it's now elicited a humoral response,
13 an antibody response, and the other arm, the cell-
14 mediated response.

15 Let me just step back. It's initiated the
16 immune response, and the viremia has begun.

17 A Right. You're asking me what it does after
18 the viremia?

19 Q Actually, I'll strike that question. What
20 in Michelle Cedillo's case have you looked at that's
21 pertinent to what I've said so far?

22 A The fever.

23 Q The fever. Was there something else?

24 A There was. There was a faint rash also
25 reported, yes.

1 Q And did she have a test for antibodies as
2 well?

3 A No, she did not.

4 Q Okay. She had that when?

5 A Oh, much later when Dr. Gupta investigated
6 her. I don't know if she had it any other time, but
7 she had it then.

8 Q Okay. And do you recall what that test
9 showed?

10 A No.

11 Q Do you know whether she had a measles-mumps-
12 rubella vaccine after the one that we're talking about
13 in 1995?

14 A No. I mean, she didn't.

15 Q She didn't?

16 A No.

17 Q And you don't believe that she was exposed
18 to wild measles virus. Is that right?

19 A It's not the matter of my belief. I didn't
20 find any evidence in the records that she had been.

21 Q Now we have the viremia. Where does the
22 virus go next after the viremia or during the viremia?

23 MS. CHIN-CAPLAN: Special Master, these are
24 all virology questions. Dr. Kinsbourne is a
25 neurologist.

1 We had Dr. Kennedy here. It seems to me
2 that Mr. Matanoski could have questioned Dr. Kennedy
3 about all this information.

4 SPECIAL MASTER HASTINGS: I don't think Mr.
5 Matanoski is looking for information for its own sake.
6 He's trying to test Dr. Kinsbourne's understanding,
7 and since he's the one that presents the ultimate
8 opinion I'll give him some play here to ask a few
9 questions in this regard.

10 MR. MATANOSKI: Thank you, sir.

11 BY MR. MATANOSKI:

12 Q Do you need me to repeat my question?

13 A Yes, please.

14 Q After the viremia begins, where does the
15 virus go next?

16 A The virus is cleared from the system and
17 disappears.

18 Q And when it doesn't, as is the posture
19 within this case, where does it go next?

20 A When it doesn't it would be likely to settle
21 in macrophages, maybe in dendritic cells and possibly
22 in other places of which I'm not aware.

23 SPECIAL MASTER HASTINGS: Before we go on,
24 Doctor, I'm hoping that that microphone we just
25 clipped to you is helping the people at home.

1 Unfortunately, that microphone is for the phone
2 conferencing.

3 That big microphone, if you could pull that
4 a little closer to you? That would help here in the
5 courtroom.

6 THE WITNESS: Yes, sir.

7 SPECIAL MASTER HASTINGS: Just a little
8 closer to you on the stand. Great.

9 BY MR. MATANOSKI:

10 Q I'm sorry, Doctor. You said that what
11 happened? I lost you.

12 A Well, the virus would settle in macrophages
13 and it would settle in dendritic cells, and there may
14 be reservoirs of it established elsewhere such as the
15 bone marrow, but I don't know that for a fact.

16 Q And how long does it take to do that?

17 A I have no idea.

18 Q Do you know how it does it? How does the
19 virus get into the macrophages? Do you know?

20 A The virus gets into a cell by attaching with
21 that sequence, the genetic sequence that Dr. Kennedy
22 explained.

23 Q And would we see any symptoms externally,
24 clinical symptoms, of this happening?

25 A Not to my knowledge.

1 Q And it still isn't resident in any organ
2 other than blood at this point, or is it resident in
3 some organs?

4 A Well, it's resident in lymphoid tissue and
5 maybe elsewhere, but I don't know that.

6 Q Is there any diagnostic test we could do at
7 that point to find the presence of measles virus?

8 A I would imagine that one could biopsy a
9 lymph node and test for the virus.

10 Q And if it's in the macrophages could you
11 test the blood?

12 A Yes.

13 Q And if it's circulating in the blood you
14 could test the blood. Is that right?

15 A I would think so.

16 Q Now, from there in the course of your theory
17 it seems that it needs to get to the gut. How does it
18 do that?

19 A It would get to the gut by the circulation,
20 by the blood circulation, and then settle in the
21 lymphoid tissue in the gut, in the gut lining.

22 Q And how does it get into the lymphoid tissue
23 in the gut lining? Is it the same process, or is it a
24 different process that we were talking about for --

25 A I don't know whether it's the same or

1 different process.

2 Q I'm sorry?

3 A I don't know whether it's the same or a
4 different process.

5 Q How long would it take from the time period
6 that you got MMR to it being in the gut?

7 A I don't know.

8 Q What symptoms would we expect to see at the
9 time that the measles virus entered the gut?

10 A That is a question for a gastroenterologist.
11 I haven't asked about the concourse of symptoms
12 following viral invasion of the gut lining.

13 Q Do you know, and if you don't that's a
14 perfectly fine answer if you don't know the answer to
15 this. Do you know what kind of diagnostic testing we
16 would do to test whether it would be in the gut?

17 A Well, I'm sure I don't know it as
18 comprehensively as somebody who is in that discipline.

19 Q In which discipline?

20 A Gastroenterology. You would, for instance,
21 determine by scope whether there's inflammation, and
22 if there's inflammation you could take a biopsy and
23 analyze the sample.

24 Q Doctor, from what you know of measles virus,
25 would it preferentially attack the gut or find its way

1 to the gut, or would it find its way to other places
2 as well?

3 A It's described as preferentially being
4 enterotropic, as I mentioned this morning.

5 Q Any other place in the body?

6 A I've already mentioned the lymphatic system.
7 If it gets access to the nervous system it will go
8 there, but it may not get access.

9 Q Now, you told me you didn't know exactly
10 what kind of gut symptoms we should expect.

11 A Well, I do know that one would get diarrhea.
12 What I don't know is the timeframe.

13 Q Is that specific to measles virus, the
14 diarrhea?

15 A No. There are some other causes of
16 diarrhea.

17 Q Is it described that measles virus, the
18 typical response to it is diarrhea?

19 A I don't know whether it's typical. I have
20 seen descriptions of diarrhea following measles
21 infections, yes.

22 Q You mentioned that it also is preferential
23 to lymphatic tissue. Is that right?

24 A Yes.

25 Q What symptoms would we expect to see when it

1 was in the lymphatic tissue?

2 A Well, if a child has measles -- in other
3 words, if it's a measles infection that you're talking
4 about, which I'm not clear about -- then the lymph
5 glands could be swollen.

6 Q And I am talking about measles virus
7 infection.

8 A You are? I see.

9 Q So these preferential areas are the lymph
10 glands and the gut, and if it's hitting these
11 preferential areas you're not sure what symptoms other
12 than diarrhea for the gut, but you'd expect to see
13 swollen lymph glands. Is that right?

14 A And if we're talking about measles you would
15 obviously get the skin involved with a rash, and you'd
16 get spots in the mucosa inside the mouth, and you
17 would get a measly appearance of the child with
18 conjunctivitis and runny nose. At least when I was
19 young that was a familiar situation.

20 Q And when the symptoms went away what would
21 that indicate to you, the symptoms of swollen glands
22 or rash disappearing?

23 A Well, I would suppose that the immune system
24 had disposed of the virus effectively, although it may
25 take quite a while for the lymph glands to go down

1 actually.

2 Q And is this generally well known in terms of
3 the course of that viral entity?

4 A I think the rather simplistic description
5 I've given is well known. I'm sure there's much more
6 known.

7 Q I'm sorry?

8 A I'm sure there's much more known by a
9 virologist than that.

10 Q Now, if the postulate that we have was still
11 resident in the gut, would you expect it to be
12 resident in other areas of the body at the same time?

13 A I don't know.

14 Q Would you please describe how it would get
15 from the gut to the brain? This is the measles virus.

16 A If the measles virus were stored, as it
17 were, in the gut lining and latent there then it would
18 get to the brain through the circulation of the blood.

19 Q If the measles virus was persisting in the
20 gut would the virus continue to circulate through the
21 body in the blood?

22 A Sometimes and sometimes not.

23 Q Okay. How often does it do that? How often
24 does it circulate in the body if it was in the gut?

25 A I don't know. I don't know such a thing.

1 No.

2 Q You don't know what the probabilities are
3 that it would not be in the circulating blood?

4 A We're talking about a normal person? No, I
5 don't know.

6 Q Let's talk about someone whose postulate has
7 an immune deficiency as in this case. How would it
8 occur in that instance?

9 A It is likely --

10 MS. CHIN-CAPLAN: Special Master, these are
11 all virology questions. I haven't heard a
12 neurological question yet.

13 MR. MATANOSKI: We're getting to neurology,
14 but I did hear this morning offered testimony in the
15 area of virology, and Dr. Kinsbourne is free to say he
16 does not know on any question I ask.

17 SPECIAL MASTER HASTINGS: I'll let him
18 continue.

19 MR. MATANOSKI: Thank you.

20 THE WITNESS: I'm sorry. Could you say it
21 again?

22 BY MR. MATANOSKI:

23 Q Doctor, in the person that we're talking
24 about who has an immune dysfunction as in this case,
25 if the virus was in the gut how is it getting to the

1 brain?

2 A Well, by the circulation of the blood.

3 Q In that same person that you're postulating
4 that it's persisting, if it's persisting in the gut
5 would it continue to circulate in the blood
6 continuously?

7 A Not necessarily, no.

8 Q Is the virus going to be only resident in
9 the gut and not having it circulate in the blood at
10 all?

11 A It's a possibility.

12 Q Is it more likely than not that that would
13 be the case if it was resident in the case?

14 MS. CHIN-CAPLAN: Now we have a
15 gastroenterology question. The experts were here.

16 SPECIAL MASTER HASTINGS: Ms. Chin-Caplan,
17 as I understand your case, this is the only medical
18 doctor who puts it all together, who gives the
19 ultimate opinion.

20 I don't think either the immunologist or the
21 gastroenterologist gave the ultimate opinion of
22 causation, so I think it's appropriate. I think these
23 questions are appropriate for the physician who's
24 giving the ultimate opinion on causation for you.

25 To the extent he doesn't know the answer, he

1 obviously feels free to state when he doesn't.

2 BY MR. MATANOSKI:

3 Q Doctor, back to you.

4 A Thank you. Again, if you'll be so kind?

5 Q We'll start back with the gut thing. In the
6 postulate you have, if the measles virus is resident
7 and persisting in the gut how is it getting to the
8 brain?

9 A By the circulation of the blood.

10 Q Can it exist in the gut alone without it
11 entering the bloodstream to get to the brain?

12 A It couldn't get to the brain without getting
13 into the bloodstream.

14 Q If it's persisting in the gut, would it
15 continue to be in the circulating blood?

16 A I would imagine that sometimes it would and
17 sometimes it wouldn't.

18 Q How often would it not be in the circulating
19 blood?

20 A I don't know.

21 Q So this is your guess that it sometimes
22 might not?

23 A It seems biologically reasonable for any
24 substance in lymph glands at times to percolate in
25 amounts -- tiny, small, medium, large or enormous --

1 into the blood and other times not to do that.

2 That would be on general principles, but how
3 often? If anybody knows the answer to that question,
4 which I doubt, I certainly am not the person.

5 Q I don't want to ask a question that's
6 already been answered.

7 I believe you said you didn't know how long
8 between the receipt of measles virus vaccine would it
9 be before the virus entered the gut. Is that correct?

10 A Yes. I don't know that.

11 Q Do you know how long it would take before
12 the virus went from the gut to the brain?

13 A I know that it can take months or years for
14 the virus to reach the brain from its source, but I
15 don't know specifically how that's modified if the gut
16 is the source.

17 Q So you don't know how long it would take if
18 the gut was the source of the virus? You don't know
19 how long it would take before it would enter the
20 brain. Is that right?

21 A I imagine it would be very variable. I
22 don't know that there are time limits on this.

23 Q Now, for the part of the theory that you
24 have from getting to MMR into the gut, what studies do
25 you rely on?

1 A For my knowledge that the measles virus gets
2 into the gut it is a property of the measles virus to
3 be enterotropic.

4 Q No. For your proposition that measles-
5 mumps-rubella vaccine, the measles component of it,
6 would enter the gut, what studies do you rely upon?

7 A No particular studies. I rely upon the
8 opinion of the virologist, of the appropriate
9 specialist. I have not studied that independently.

10 SPECIAL MASTER HASTINGS: Let me stop again.

11 Again, Doctor, I hate to repeat this. I had no
12 trouble hearing you this morning, and I'm having a
13 hard time now.

14 I don't know if it's something I ate, but if
15 you could speak up as best you can?

16 SPECIAL MASTER VOWELL: Perhaps if he pulls
17 his chair forward?

18 I notice it's sort of facing back, Doctor.
19 That might help get you a little bit closer to the
20 mic.

21 THE WITNESS: To the extent my body permits.

22 SPECIAL MASTER HASTINGS: We appreciate it.

23 Thank you.

24 THE WITNESS: Okay.

25 SPECIAL MASTER VOWELL: That's much better.

1 THE WITNESS: I hope so.

2 SPECIAL MASTER HASTINGS: We want to hear
3 what you have to say.

4 THE WITNESS: Yes.

5 SPECIAL MASTER HASTINGS: Mr Matanoski, go
6 ahead.

7 MR. MATANOSKI: Thank you.

8 BY MR. MATANOSKI:

9 Q Up to this point when I had asked you
10 questions about how confident you are you ventured an
11 opinion. Now, how confident are you in the notion
12 that the vaccine, the measles component, would enter
13 the gut?

14 A See, my difficulty is with the formulation
15 of the question. Would enter the gut in every child?
16 In some children? Nothing happens always.

17 Q I'm sorry. Let's deal with this case then.
18 In this case.

19 A How confident?

20 Q How confident are you in this case that it
21 would enter the gut?

22 A Okay. I have a degree of confidence about
23 50 percent in this case.

24 Q Now, when the virus gets to the brain, how
25 does it enter the brain?

1 A It would enter the brain through the lining
2 of the blood vessels otherwise known as the blood-
3 brain barrier.

4 Q And how does it do that?

5 A It would typically be transported by
6 macrophages. In other words, it would be inside the
7 macrophage which then can permeate the blood-brain
8 barrier.

9 Q And once the macrophages enter the brain --
10 let me take a step back. Would you see any clinical
11 symptoms of that happening?

12 A I can't locate any such symptoms.

13 Q In the factual context of this case how long
14 would it take between the receipt of MMR with the
15 measles vaccine component before the macrophages would
16 bring measles virus into the brain?

17 A In the context of this case my opinion is
18 that it took seven days, but as a general principle I
19 can't give you an answer.

20 Q You just said it would happen in seven days.
21 Then you know or have a guess as to how long it
22 should take as a pathologic process or as a
23 physiologic process?

24 A I have no opinion as to whether it should
25 take longer. I don't believe that it should take

1 longer.

2 Q I'm trying to find out why seven days is
3 okay in this --

4 A No. You asked in the context of this case.

5 Q I understand that seven days is good in this
6 case, but that has to be based on some body of
7 knowledge about how long it should take in order to be
8 appropriate in this case. What is that body of
9 knowledge or what is that range of how long it should
10 take if you know?

11 A I have not reviewed virologic literature on
12 that process, so I don't know.

13 Q Okay. Once it's in the brain what happens
14 next? What does the virus do next?

15 A The virus will settle inside cells.

16 Q What kind of cells?

17 A They could be neurons, they could be
18 astroglia.

19 Q Any other kind?

20 A They could be microglia, they could be a
21 combination.

22 Q Does measles virus preferentially select
23 certain cells?

24 A I believe that it is most apt to be in
25 astroglia.

1 Q Because that's preferentially selected by
2 measles virus?

3 A For whatever reason I think that's where
4 it's most commonly found. I mean, I don't know, and
5 there's a reason for saying it. I'm not sure that the
6 measles virus makes the decision or maybe the cell
7 makes the decision. The microglia has a phagocytic
8 function and would actually encompass the virus. On
9 the other hand, the virus would attach itself to
10 another brain cell which had no particular interest in
11 it.

12 Q Okay. So it's your belief then that it's
13 actually the cells themselves that are better
14 attracted to these particular cells? The astroglia
15 are attracted to the measles virus?

16 A I'm not sure I said it like that, but let me
17 say it again. A phagocyte is cell that's specialized
18 to encompass foreign material, and the virus is
19 foreign material and the microglia has a phagocytic
20 function, so one would expect it to take up virus
21 particles. On the other hand, the virus has certainly
22 entered cells that are not phagocytes, and they do
23 that by the mechanism Dr. Kennedy describes, by
24 attaching themselves to the cell membrane and entering
25 the cell.

1 That could happen either with neurons or
2 with astrocytes.

3 Q After it enters the astroglia what happens?

4 A It stays there. Now, depending on the
5 circumstances it either destroys the cells whereupon
6 it also destroys itself, that's calls a cytolytic
7 process, or it remains persistent in the cell where
8 the cell continues to be intact, which is a not
9 cytolytic process. Both occur.

10 Q Both occur simultaneously?

11 A No. Well, I don't know. Maybe they do, but
12 there are circumstances where you have your cells
13 being destroyed and others where you don't. It's not
14 an essential characteristic of all measles virus
15 infections of the brain that there be cytolysis, but
16 it certainly can occur. But there are other
17 circumstances where the measles virus stays inside the
18 cell, and the cell maintains its integrity but not
19 necessarily its function.

20 Q Okay. So there are variable results? It
21 can do some cytolytic damage and it may not do some
22 cytolytic damage?

23 A Correct.

24 Q How do you measure whether it's going to do
25 cytolytic damage or not?

1 A Well, if you have the opportunity at the
2 right time then microscopically you can see the
3 necrosis of cells, whether the cells are in fact
4 process of dying, or subsequently you can see whether
5 the number of the cells is depleted so that you can
6 assume that some were killed.

7 Q Do you know of any time when measles virus
8 entered the brain and it was non-cytolytic? It had no
9 cytolytic activity at all?

10 A I'm not aware of a question like no
11 cytolytic activity all. I don't know where that's the
12 case.

13 Q So you don't know if it can exist without
14 doing some cytolytic damage?

15 A Some is such a minimal. I mean, anything
16 can happen.

17 Q Should I direct this question to a
18 virologist?

19 A Well, it's certainly up to you, but it might
20 be appropriate.

21 Q Would it be more appropriate to direct this
22 question to a virologist?

23 A Yes. I think that at the infinite level of
24 the infection between the virus and the cell
25 regardless of the system of the body this is more in

1 the domain of a virologist.

2 Q What studies are you relying on for your
3 description of what the measles virus will do on a
4 cellular level in the brain?

5 A Do you mean what do I rely upon in the
6 proposition that the measles virus enters the cells?

7 Q You just gave us a description of what the
8 measles virus would do on a cellular level in the
9 brain. I'm asking for what studies you're thinking of
10 or support for that proposition.

11 A I've read about this. I don't think that
12 what I said was cutting edge science, so I don't
13 remember any recent specific article. I mentioned Dr.
14 Oldstone's caution that the virus can persist without
15 cytolysis. Obviously the virus can also cause
16 cytolysis. I mean, I didn't know that was
17 controversial.

18 Q Okay. But you don't know what the relative
19 probabilities are of it being non-cytolytic?

20 A No, I don't.

21 Q Now, after its entered the cells in the
22 brain, what happens next?

23 A The innate immune system of the brain would
24 launch an immune attack on the cell with the virus.
25 In other words, the presence of the virus antigen

1 would activate the microglia, and the microglia would
2 produce proinflammatory cytokines and the
3 proinflammatory cytokines would generate inflammation.

4 Q And this is a proper response by the body to
5 the entrance of a virus. Is that right?

6 A Yes, it is.

7 Q Now, can you get us from what the virus is
8 doing in the brain to how the virus is creating
9 autistic spectrum disorder? Actually, let me take a
10 step back. How long would it take after the virus
11 entered the brain before the innate immune system
12 would begin to kick in?

13 A The innate immune system typically has a
14 short latency, so should react quickly.

15 Q So as the innate immune system begins what's
16 the next step in the process?

17 A Well, inflammation is created.

18 Q I'm sorry?

19 A Inflammation.

20 Q Inflammation is the next step?

21 A After the innate inflammation.

22 Q And the inflammation is for which purpose?

23 A Well, it's the same purpose as when you
24 scratch yourself on the arm. It is for removing the
25 invader or the substance that has the characteristics

1 of an invader.

2 Q It's a proper immune response?

3 A Correct.

4 Q Now, what's the next step in the process
5 that leads us to autistic spectrum disorder?

6 A The process that we've described so far as
7 mentioned involves astrocytes. The astrocytes are
8 inactivated or killed, depleted in number, or weakened
9 in their function and the consequences in the
10 particular mechanism of injury that I offered this
11 morning are that a glutamate excess may result. I
12 could go over those further steps again, but they
13 would be the same as I testified this morning.

14 Q Taking a step back, the innate immune
15 response to measles virus in the brain, how confident
16 are you in that occurring?

17 A I'm confident. About 50 percent.

18 Q Only about 50 percent?

19 A I said about.

20 Q I'm sorry?

21 A About 50 percent.

22 Q You're not much above the 50 percent level
23 that there's going to be an innate immune response?

24 A Well, let me be more specific because this
25 is a really contrived exercise. I know about this

1 from the Vargas work, and the Vargas work I think is
2 of high quality, and the findings are explicit and
3 they are well-regarded. However, they are a single
4 study so I'm not going to be 100 percent confident on
5 anything on a single study, but it gives me a
6 sufficient basis for my opinion.

7 Q So you believe that it's pretty well-
8 established in your view that there be an innate
9 immune response to a virus in the brain. Is that
10 right?

11 A Actually, I'm sorry. I was talking about
12 the autistic children.

13 Q No, no, no. I'm sorry, Doctor. I didn't
14 mean to confuse you. I'm taking you back a step to
15 the innate immune response.

16 A Okay. The question of whether the innate
17 immune system responds in this fashion in the brain of
18 normally functioning children is not one that I have
19 considered, and therefore I can't offer an opinion on
20 it.

21 Q I'm sorry. You can't offer an opinion on?
22 Could you repeat your last answer? I just had trouble
23 hearing you.

24 A Yeah. Should the measles virus enter the
25 brain under normal circumstances, which I believe it

1 is a very uncommon event, then all arms of the immune
2 system represented in the brain will presumably react,
3 innate and adaptive. I have no reason to doubt that.

4 Q You feel fairly confident?

5 A Yes.

6 Q Okay. The next step that you were
7 describing was getting us to how we have autistic
8 spectrum disorder.

9 A Right, and the mechanism of injury which I
10 offered, which I present as medically reasonable but I
11 do not present as scientifically proven, involves the
12 pesticides, which as we have discussed can be
13 inactivated or destroyed by the virus, and involves a
14 known function of the astrocytes, which is not a
15 controversial but an accepted function, with respect
16 to the control of the level of glutamate, which is an
17 excitatory neurotransmitter.

18 I explained this morning in a simple way
19 what the astrocytes normally contribute to the control
20 of glutamate levels and the consequence of that
21 control lacking being that the glutamates would be
22 produced and persist in the brain in excessive
23 amounts.

24 Q Okay. So the former proposition that
25 glutamates would be present is fairly well-

1 established?

2 A That astrocytes are involved in glutamate
3 control is supported by current literature. Yes.

4 Q How confident are you in that happening?

5 A I've read multiple publications making that
6 point. I'm not aware of seeing controversy about it.
7 There is hardly anything in your science about which
8 isn't some controversy so I can't give you guarantees.

9 Q Okay. But you're pretty confident in that
10 one?

11 A Yes.

12 Q This particular part of the theory about
13 astrocytes, their role in controlling glutamates, is
14 that a new theory?

15 A I don't know how new it is. I suspect the
16 finding is recent, but I couldn't tell you exactly.

17 Q So you're not that familiar with the
18 literature on that particular --

19 A I haven't made that issue a particular focus
20 of my study. It is something that I'm aware of in my
21 knowledge of brain function.

22 Q How long does it take from the time the
23 virus enters the brain before this process would
24 begin?

25 A Before it?

1 Q Before this process would begin.

2 A I have no information on that.

3 Q Has it ever been studied?

4 A I don't even know that.

5 Q The next step in the process, could you
6 describe that, please? What happens next?

7 A The shift in the balance between excitatory
8 and inhibitory influences in the brain, which as I
9 mentioned this morning is always a fluctuating factor
10 in brain function, would be skewed to the excitatory
11 extreme. That would affect the communication between
12 neurons in different parts of the brain in a
13 particular manner.

14 In order for the brain to do what it does,
15 which is to form specific patterns of activation which
16 correspond to specific experiences in the outside
17 world, specific memories in the inside world, specific
18 emotions, they correspond to particular considerations
19 of neuronal activation. It's like peaks and balances.
20 Just think of mountainscapes, only the mountains are
21 levels of activation.

22 Then you would have a conceptual model of
23 the pattern of brain activation, which ideally a
24 million years from now we'll be able to specify from
25 that pattern what a person is experiencing.

1 Obviously, we can't do that. Now, the point that I'm
2 addressing isn't a particular pattern representing a
3 particular experience, but the ability of the brain to
4 discriminate between different experiences by making
5 differential patterns.

6 For it to do that in a refined way, it needs
7 two elements. It needs the activation to cause the
8 amplitude and a deep inhibition to model one, to
9 circumscribe them so they don't overflow, because
10 otherwise it's like ink running. The pattern would
11 become less specific and the behavior would become
12 less refined and more crude.

13 So what I'm saying is that the precision
14 with which a person can think is more a function of
15 the ability to inhibit specifically just like the
16 precision of a sculpture is a function of the ability
17 of the sculptor to decide exactly what to remove from
18 the medium. Now, if the inhibitory aspect of forming
19 brain cells is overwhelmed by the activation, one is
20 going to get fewer differential brain cells available,
21 and that makes fewer distinctions. That's one point.

22 In other words, as I was saying this
23 morning, only simpler issues can be addressed would be
24 the correspondent. It would be what would correspond
25 to this particular perspective.

1 Q My question was a little simpler than that.
2 I'm fine with going on, sir, but it was a little
3 simpler than that, but go ahead.

4 A I'm sorry. I do want to respond to what
5 you're asking me. Am I doing all right?

6 Q Go right ahead if you have more to say.

7 A There's always more. The last point to make
8 before I let you reenter this conversation is that it
9 is particularly hard for a brain to have a specific
10 distribution of patterns in different parts of it, and
11 in the cruder brain they would collapse into fewer
12 simpler aggregations. This I think happens in the
13 immature brain, in the damaged brain, in the demented
14 brain.

15 In order to have very specific patterns
16 involving different modes of thinking, for example,
17 sensation, movement, better memory, you have to hold
18 these activations apart so they each contribute to the
19 refined mental state.

20 If you have over activation then you're
21 unable to form these refined distributive patterns and
22 you are thrown back on the more simplistic local
23 selective patterns such as ascending to just one thing
24 or one part of a thing, or obsessing in one's mind
25 with one thought over, and over and over, or engaging

1 in a particular activity again, and again and again as
2 opposed to flexibly moving from one thought to
3 another, one perception to another, one memory to
4 another, one activity to another, as is required by
5 the contingencies of normal living.

6 Okay. That's an answer, but please proceed.

7 Q Thank you, sir. How long does it take
8 before the disregulation and the excitatory process
9 begin?

10 A I don't know.

11 Q What studies do you rely on for this result,
12 the excitatory disregulation?

13 A As I mentioned this morning the concept of a
14 diet in the excitation inhibition ratio or balance is
15 formulated by the article of Rubenstein and Merzenich
16 which I have included in my bibliography.

17 Q Is that your best support for this
18 proposition?

19 A Yes.

20 Q How confident are you that this is what's
21 happening? Is that the 50 percent threshold or is it
22 greater?

23 A It's about 50 percent, but it certainly
24 requires more studying.

25 Q What's the measles virus doing meanwhile in

1 the brain?

2 A As far as I know, it's sitting there.

3 Q Which link in your chain of causation are
4 you least confident in?

5 A For me to answer this accurately, could you
6 give me the particular links that you want me to
7 compare?

8 Q Take your whole chain that we just went
9 through. Which link in it are you least confident in?

10 A I'm taking my time reviewing my chain, okay?

11 Q Take as much time as you want, Doctor.

12 A Thank you. I think it's a question of how
13 to define the body and the link. My opinion renders
14 that more likely than not the measles virus is the
15 cause of the encephalopathic process, which with
16 regards to an autistic behavior is based on arguments
17 such as the measles virus is neurotropic, it's
18 neuropathic, it's been shown to be persistent in this
19 individual person, this person did in fact evidence
20 encephalopathic regression, there's no evidence for
21 other viruses or other causes for that. And that in
22 my opinion already meets the burden of an opinion at
23 the level of reasonable medical probability.

24 Now I have gone beyond that degree of
25 explanation to attempt to define some of the links

1 within links if you like in terms of more precisely
2 how the infective agent affects brain cells and so on.
3 And in doing so, I have tried to do something which
4 neurologists and neurophysiologists often don't know
5 about or talk about this.

6 Neuroscience isn't at the level where one
7 can routinely expect all the links in such a chain to
8 be definite and defined. Absolutely not. So I
9 actually feel that when offering the mechanism I did,
10 I was exceeding my burden in the context of the
11 present proceedings.

12 Q That wasn't my question, though, Doctor. If
13 you don't have at least a theory of how it lays out a
14 sequence, then it's just your say-so. It's what we
15 call in law ipse dixit. But we've laid out a nice
16 theory, a nice sequence, a nice chain here, and all my
17 question asked you is which part of that sequence is
18 the weakest in your mind in your opinion?

19 A No, I understood the question. I'm having
20 real trouble with it because it's so hard to compare
21 them with each other, you know?

22 Q Well, then name your top three weakest.

23 A Let me choose my particular mechanism of
24 injury, let's call it P.glutamate excitation
25 hypothesis, okay? I certainly don't purport that's

1 the only available explanation, and I certainly can't
2 say it's been scientifically proven. I think it's a
3 good enough possibility, or probability, or
4 plausibility to meet our standards, but I can't say
5 that I'm intensely confident about it because there
6 are alternatives, and literature has them and we've
7 got controversial areas.

8 Q So if you had to choose that would be the
9 weakest?

10 A Yes.

11 Q Doctor, is it accurate to say your pediatric
12 practice at this time is very limited?

13 A Yes, sir.

14 Q It's been about 20 years since you've had an
15 active pediatric practice. Is that right?

16 A Well, yes, almost. It's 17 or something
17 like that. Yes.

18 Q In your CV you stated you have accumulated
19 extensive experience with disorders in mental
20 development. You listed autistic spectrum disorders
21 as one of these, but you haven't treated children on a
22 regular basis in 17 years. Is that right?

23 A Yes. That's right.

24 Q So your extensive experience to the extent
25 it had anything to do with autism would be 17 years

1 ago?

2 A My experience in the clinical management of
3 autistic children on any ongoing regular basis
4 certainly expired at that timeframe. My study of the
5 literature, my scholarly involvement, my experience of
6 the type that I described this morning under
7 questioning from Ms. Chin-Caplan, my interactions with
8 colleagues interested in autism, my collaboration in
9 scientific investigations have been very ongoing and
10 continuous.

11 I've had a continuous interest in autism for
12 many years, but that interest is more expressed at the
13 level of biomedical and biobehavioral investigation,
14 which is the level at which we are operating here,
15 than it is in the routine clinical management of
16 children.

17 Q I'm sorry. I'm not sure I understood the
18 distinction there. Your interest in autism for the
19 past 17 years has been elsewhere? I understand you to
20 say it's not in actually treating autistic children.

21 A Okay. Let me see if I can rephrase.
22 Sources of information that a physician acquires about
23 a disorder come basically in two ways. One is his or
24 her clinical experience in it with the actual
25 individuals who are suffering from the problem. The

1 other area is his or her acquaintance with the sum of
2 the scientific literature with respect to the
3 condition in question.

4 Now a person can be seeing hundreds or
5 thousands of autistic children and have no
6 understanding of the type of brain mechanisms that we
7 are discussing at this time. What I was pointing out
8 is that although my interpersonal interaction with
9 autistic children has greatly diminished my
10 interaction with the pertinent biobehavioral and
11 biomedical literature continues to be intense.

12 Q You said there are two different sources but
13 a scientist could get information or experience in
14 both of those sources, correct?

15 A Yes. If you take the concept of evidence-
16 based medicine, which is much discussed and certainly
17 an excellent concept, that basically says you have to
18 base yourself not on your personal experience, that's
19 anecdotal, but on the products of systematic studies.
20 And I think that the trend at this time is very much
21 in that particular direction.

22 Q You don't mean to suggest that someone who
23 treats, and also who researches and spends their time
24 with the medical literature on autism is somehow less
25 qualified to speak on that topic, are you?

1 A You're right, I don't mean to suggest that.

2 Q Since you've acknowledged that exposure in
3 treatment to children would be one source of
4 information informing you about autism would they have
5 greater knowledge than someone who has limited
6 themselves to reading medical literature on it?

7 A It depends on the quality of the individual
8 and their engagement than on any number of exposure of
9 one or other kind. I've had massive exposure to
10 attention deficit disorder as it happens, and I've
11 learned an awful lot from it. You know, the last
12 thing I would do is to criticize that type of
13 engagement.

14 Q You don't belong to any societies or
15 associations that are devoted to the research, care or
16 treatment of autism, do you?

17 A No. Well, actually, I think I'm a member of
18 IMFAR. I joined IMFAR. Yes. The association that
19 meets once a year.

20 Q You think you might be a member of that?

21 A Huh?

22 Q You think you might be a member of that?

23 A Well, I think I might. I attended a
24 meeting, and I believe I signed up. I didn't go last
25 year. But to tell the truth I hadn't thought about

1 that until now. I'll check on it.

2 Q So it doesn't come to the top of your mind?

3 A Not constantly. No.

4 Q No. And not in response to my question. In
5 the past 20 years how many articles have you published
6 on autism?

7 A I should say five or six. I could locate
8 them for you.

9 Q That's in the past 20 years. Sure it's that
10 many?

11 A I'm sorry?

12 Q Are you sure it's that many?

13 A I don't know why I'm not understanding this.
14 I'm sure it's my fault.

15 Q All right. You think it's five or six.

16 A Well, I don't need to think. If you give me
17 my CV I can count them for you.

18 Q How many of those articles were original
19 research by you?

20 A Well, the most recent is and that would be
21 the Fein, F-E-I-N, and colleagues. That came out I
22 think just within about a year ago. It's listed.

23 Q Fein and colleagues?

24 A Yeah.

25 Q This is on autism?

1 A Yeah. Actually, it will be Fein, but not
2 the first author. I'm sorry. If you show me the CV
3 I'll show it to you.

4 Q That's okay, Doctor. It's on file.

5 A It is, and it was within the last year. It
6 was published in *Autism* I believe. Liss actually is
7 the first author, L-I-S-S. Thank you.

8 MR. MATANOSKI: For the record, counsel for
9 the Petitioners just handed Dr. Kinsbourne -- I take
10 it you're handing him his CV?

11 MS. CHIN-CAPLAN: CV.

12 BY MR. MATANOSKI:

13 Q I really don't have any other question in
14 that particular area.

15 A Then I'll put it away, sir.

16 Q Thank you. I'm fine with you reading it,
17 but I just want to move on to the next question.

18 A No. I understand.

19 Q You mentioned that you periodically, I guess
20 every time that Dr. Menkes publishes his textbook, you
21 do a chapter on there on developmental disorders. Is
22 that right?

23 A Yes. I do what's now Chapter 18, which is
24 the one entitled *Disorders of Mental Development*.

25 Q In the 2006 version you mentioned you worked

1 with Frank Wood on that?

2 A Yes.

3 Q You discussed in your direct that there's an
4 extensive section on autism in that?

5 A Yes.

6 Q Did you or he write the section on autism?

7 A I wrote that.

8 Q And in that you developed a chart that had
9 the concomitance of autism?

10 A Concomitance. Yes.

11 Q I'm sorry.

12 A Yeah. No. I understand. There is this
13 long, rather dreary table of names.

14 MR. MATANOSKI: Yes. Actually, I think we
15 could show you what it looks like in your book
16 chapter.

17 SPECIAL MASTER HASTINGS: Is this --

18 MR. MATANOSKI: This is I believe submitted
19 as a Petitioners' exhibit.

20 SPECIAL MASTER HASTINGS: I believe so, too.
21 Anybody have the cite to it? Which tab?

22 MR. ROONEY: PP.

23 SPECIAL MASTER HASTINGS: PP. Thank you.

24 BY MR. MATANOSKI:

25 Q On that you listed a number of causes or the

1 concomitance. I'll get it before we're done, Doctor.

2 Under that you listed viral as one of them. You

3 listed three different viral concomitance.

4 A Yes.

5 Q There they are, rubella, herpes and

6 cytomegalovirus.

7 A Okay.

8 Q Now, in your report you pretty much

9 reproduce this chart.

10 A Yes.

11 Q There was one change. You added one. You

12 added measles.

13 A Right.

14 Q Anything happen in the last year to cause

15 you to add measles to that chart?

16 A Nothing happened, but my mind being so much

17 on this seemingly endless litigation I thought to

18 myself, hey, you didn't put in measles.

19 Q I see.

20 A I mean, it's extremely rare, but so are the

21 others.

22 Q On that same publication, 2006, you put out

23 a chart of mental development. We don't need to show

24 you that.

25 A Okay.

1 Q Is that an accurate representation in that
2 publication of what you expect to be the developmental
3 progress of an infant?

4 A You mean a listing of milestones?

5 Q Yes.

6 A Yes. I would hope that what I put in is an
7 accurate representation.

8 Q Now, in this case you've testified to
9 reviewing all the videos that the family --

10 A I did look through the videos. Yes.

11 Q Was this the first time you ever reviewed
12 videos for the purpose of identifying signs of
13 autistic behavior?

14 A Yes.

15 Q Doctor, I'm going to ask you a series of
16 hypothetical questions. They're going to be based on
17 the facts of this case. What I'm going to do is I'm
18 going to take one fact out and ask you if your opinion
19 is the same.

20 A Okay.

21 Q If the answer is yes, no, or you don't know
22 whether that would make any difference, whatever your
23 answer is just feel free to shout it out.

24 A Okay.

25 Q Same facts of this case, but there's a

1 showing that thimerosal either has no role at all or
2 there's no thimerosal. Let's just take the thimerosal
3 out of the equation.

4 A It wouldn't change my opinion. However, I
5 would like a moment if I may to explain that to the
6 Court in the particular manner.

7 Q Sure.

8 A The Special Master designated three
9 questions for us at the beginning of the proceedings.
10 Number one was about the relationship between
11 thimerosal and immune function. The second was the
12 relationship between the measles or MMR vaccination
13 and autism. The third one was about collaboration as
14 it were between thimerosal and the MMR in the
15 causation of autism.

16 I see myself as addressing number two, the
17 relationship between the MMR and autism. From that
18 vantage point the issue of thimerosal does not impact
19 on my opinion.

20 SPECIAL MASTER HASTINGS: Just for the
21 record I'll note that it was not the Special Masters,
22 it was the attorneys for the Petitioners, the
23 Petitioners' Steering Committee, who chose to divide
24 up the causation issues into those three categories,
25 but I think you've accurately described --

1 THE WITNESS: Really? I have a inflated
2 memory I'm afraid.

3 SPECIAL MASTER HASTINGS: Okay.

4 BY MR. MATANOSKI:

5 Q Actually, just to be clear did you think
6 that they're broken up into immune and the MMR and
7 autism and another function?

8 A Well, to repeat what I thought and I
9 misattributed, but in my mind I looked at three
10 questions and I thought, I'm addressing the middle
11 one. As I saw them, and now I'm totally uncertain
12 about the source of all this, there was a separate
13 question about does thimerosal depress or disregulate
14 the immune system? That was the question.

15 That's outside my domain and not something
16 that would impact on my opinion as given today. Then
17 there was a question about the relationship between
18 the MMR vaccine and Michelle's autism, and indeed that
19 is what I testified about this morning. Then there
20 was a question about the interaction between
21 thimerosal and MMR.

22 With respect to the immune system, because
23 we're discussing thimerosal with terms of the immune
24 system not in terms directly of the brain, which is a
25 different topic not being addressed, that wasn't

1 exactly my area either. This is a very long-winded
2 way of saying that the issue of thimerosal, although
3 very interesting, is not critical to my opinion today.

4 Q With respect to your opinion is it necessary
5 for -- never mind, Doctor. I'm going to rephrase the
6 question. Use the same fact pattern, but this time
7 there's no fever.

8 A That there's no fever. That would not
9 change my opinion. Perhaps I could add a comment that
10 point. It seems to me from an overview of the
11 situation that Michelle has an unusual propensity to
12 react to the inflammation to provocative stimuli. The
13 interesting thing about the fever is that it was so
14 high and so long. In other words, the child was
15 reacting with more inflammation to a given
16 provocation, namely the vaccination, than most anybody
17 else.

18 I was wondering, is this a tendency that she
19 has that she also exhibits elsewhere? It seems she
20 does. She reacted with inflammation in the gut, and
21 not just in one part but in multiple parts, she
22 reacted with inflammation in the eye in the iris, the
23 rear and maybe also the optic nerve, and she reacted
24 with inflammation in the two ankle joints.

25 So I do think that the fever is of interest

1 in terms of being part of a general impression of a
2 rather excessive tendency to react with inflammation
3 which is of interest to me because I am invoking
4 information in yet another organ, the brain. So in
5 that general sense of a pattern it's significant to
6 me, but if you were to ask me everything else except
7 the fever, no, that wouldn't change anything.

8 Q So in this fact pattern with no fever you
9 have the MMR and seven days later no fever but the
10 onset of some symptoms that are later believed to be
11 signs of autism your opinion would stay the same?

12 A My opinion would be the same. Well, again,
13 if I may have a bit of license to say a few more
14 things on that topic?

15 Q Sure.

16 A If it's okay to make this remark, I hope.
17 The onset was unusually abrupt in this child. Being
18 that abrupt I'm not surprised that there was some
19 systemic reaction such as the fever.

20 Q But wait. Just to be clear you said it's
21 okay to not have the fever, you're opinion stays the
22 same. So we're taking the fever out. The onset of
23 uveitis and other inflammation isn't so much later,
24 but the onset of autistic symptoms is within seven
25 days. Is your opinion the same?

1 A Well, my opinion is the same. Yes.

2 Q Now, if we move the onset period out to two
3 weeks after is your opinion the same? The onset of
4 the autism.

5 A My opinion will be the same, but I think
6 that hypothetical is a little improbable. This is
7 what I'm trying to explain, that I'm really receptive
8 to hypotheticals, and I'm not trying to obstruct you.

9 Q I understand that. I understand that.
10 Please go ahead. So the timing is important to you in
11 some fashion?

12 A Well, it is of interest in the following
13 respect, that I have seen many, many descriptions of
14 regressions and some are more subtle than others, some
15 are more gross than others, but they tend to take more
16 time. This came on really fast, which is why in my
17 first report I actually suggested a table
18 encephalopathy it came on so fast.

19 Given that it came on so fast I'm more
20 comfortable with a systemic disturbance like a fever
21 to accompany such a rapid process. I would much less
22 need the fever if it came on very, very slowly over
23 three months, you know, at some subsequent time. Now,
24 if you remove the onset two weeks as we do
25 hypothetically then the fever really loses meaning

1 compared to the meaning it did have for me in the
2 actual state of affairs.

3 Q So in this factual scenario where can I move
4 the timing for the first onset of the autism before
5 you lose confidence that it's related to the MMR? And
6 let's move it each way. Let's start by saying how far
7 away from the MMR, how far out.

8 A You said each way?

9 Q Yes. I mean we're going to move it closer
10 to the MMR.

11 A You mean as compared to seven days? Is that
12 what you're saying?

13 Q Yes. It will be easier than me trying to
14 guess where the limits are. I want to figure it out
15 by just you telling me where does it go from the point
16 of MMR to the onset of autism that you start going
17 below that 50 percent threshold of confidence in your
18 opinion?

19 A Okay. Well, the systematic literature on
20 this is not available. In fact, it is as I earlier
21 remarked amazing how little actual descriptive ways of
22 regression, although it's actually mentioned. My
23 information on this is basically derived from my
24 experience in the U.K. when I reviewed many, many
25 children with regressive autism.

1 That was part of my job, and it was
2 mentioned this morning. The distribution of onset
3 relative to MMR was quite variable. We expect that in
4 natural disease. In some cases they did come on
5 within say a week or so as in this case, and perhaps
6 the majority came on after two months or perhaps three
7 months and then it sort of tailed off.

8 I didn't see a cut-off point. So what I
9 would say is that beyond about three months my
10 confidence might decrease, but not necessarily to a
11 below 50 percent level. That depends on other
12 features of the case as well.

13 Q Okay. We're talking about this case. I was
14 trying to make it easy by just using this fact
15 pattern.

16 A But by moving it you make it a different
17 case.

18 Q Yes. I just want you to say in this factual
19 scenario where do you start losing confidence when you
20 take it out to the end point? How far away from the
21 MMR?

22 A See, I have such trouble losing confidence
23 because we've got a positive measles virus. The virus
24 is there, it shouldn't be, it's neuropathic and we
25 have an unexplained encephalopathy. So I'm trying to

1 lose confidence as the interval gets larger, and I
2 might in other cases, but in this case I have trouble
3 doing that.

4 Q Okay. That's fair, Doctor. So it's the
5 measles virus genomic material that was recovered
6 that's really key?

7 A It is.

8 Q What if in this fact pattern there was no
9 finding of inflammatory bowel disease, no inflammation
10 of the gut, but all the other factors are the same?

11 A I would lose confidence in the
12 gastroenterologist because why would he or she take a
13 chunk out of a gut when there is no inflammation?

14 SPECIAL MASTER HASTINGS: You'd lose
15 confidence in the treating gastroenterologist?

16 THE WITNESS: Yes, I would. Not in our
17 expert. No, no.

18 SPECIAL MASTER HASTINGS: Okay. The one who
19 decided to take the biopsy?

20 THE WITNESS: Yes, because I wouldn't
21 understand if there was no inflammation on what it is
22 he or she was biopsying and for what purpose. So I'm
23 being a bit contrite I'm afraid.

24 BY MR. MATANOSKI:

25 Q I understand sort of. What it really keeps

1 coming back to is that positive measles virus genomic
2 material in the gut?

3 A Yes.

4 Q Okay. Then let's take that one away. Same
5 fact pattern but there's no recovery of positive
6 measles virus genomic materials.

7 A No opinion from me.

8 Q You would not find causation in that
9 instance?

10 A No.

11 Q Would you find that in any case of MMR and
12 eventual ASD whether there is autistic enterocolitis
13 or not if there wasn't, is this the sine qua non, the
14 recovery of the positive measles virus?

15 A As you correctly perceive I was talking
16 about Michelle Cedillo. This case, this hearing, this
17 situation, the first case in an important process. I
18 would not give an opinion on a case that didn't have
19 positive biopsy in this situation, nor would I give an
20 opinion if there was no reason to even think of
21 measles. I wouldn't then say it's of measles. Let us
22 suppose that the Court has made its determinations and
23 we were now looking at individual cases.

24 It would be extravagant on my part to
25 require that every one of them has had a biopsy given

1 how specialized it is and how hard it is to get them,
2 and I would be open to other evidences compatible with
3 the measles virus, but I can't tell you now because I
4 haven't considered that really.

5 Q If we take the measles virus away and you
6 still have the enterocolitis and the other factors
7 this case is really in terms of your theory a strong
8 case, right?

9 A Yes, I do believe so.

10 Q And if you took it away from this case and
11 you wouldn't be willing to, and in the other cases
12 they may not have the fever, they may not have the
13 autistic enterocolitis, is it going to be more likely
14 to you?

15 A Well, no. As I tried to explain I really
16 thought that the first case heard by this Court should
17 be one that potentially would impress the Special
18 Masters, and I would not have thought that this would
19 be a right case to present, nor representative, really
20 informative about the science that is being determined
21 if it didn't have that finding because that finding as
22 mentioned as counsel brought out in direct examination
23 this morning it was really when -- in England I
24 required virus in the gut and in the cerebrospinal
25 fluid.

1 I gave opinions in four cases. Maybe that's
2 the best way of explaining it. They all had positive
3 virus in the gut, three had positive virus in the
4 cerebrospinal fluid, okay? I gave opinions on the
5 three that had positive in gut and spinal fluid, and I
6 gave one more opinion. There was a child who was
7 typical except there was nothing in the cerebrospinal
8 fluid found.

9 I convinced myself, argued to myself that
10 since that child was in all other respects so similar
11 to the first three that it reached my criterion for
12 causation. Now, in the sense all these different
13 cases you're asking me a similar question I believe,
14 which is once we have got a reliable picture of what a
15 full house would be, an ideal case, then we can decide
16 that the world is never like that over and over again,
17 which elements can we relax?

18 Now, the question is a difficult one for me
19 when this key element of evidence that there is
20 measles virus in the system when it shouldn't be
21 cannot be addressed. So let us suppose, for example,
22 that immunologist's informance that immunological
23 findings, which would be proxy for that finding if one
24 could make inference out of the antimeasles antibody
25 or some reaction of the cellular immune system to

1 measles which could stand in instead, I would accept
2 that.

3 Then as consensus increased and we keep
4 seeing that same pattern which has been already
5 validated by the investigation then we would do what
6 all doctors do all the time, accept a diagnosis with
7 less evidence then when it was first established.

8 Q Doctor, we've already had testimony from you
9 on direct that in this fact pattern, and I take it
10 this fact pattern is the strong fact pattern, there's
11 only one thing missing that you'd want more and that's
12 recovery of measles virus genomic material from the
13 CFS, correct?

14 A Right.

15 Q So that would make this case stronger?

16 A That's correct.

17 Q Now, in this case you just testified under
18 oath that if I were to take away the measles virus
19 genomic material recovery, that test result from the
20 gut biopsy tissue, you would no longer hold the
21 opinion that this was a case of MMR causing ASD?

22 A I would no longer have held that opinion for
23 purposes of this hearing.

24 Q Okay. Now, other fact patterns that come up
25 that don't have what you're presuming is an immune

1 disregulation, indeed, evidence of inflammation not
2 only in the gut but arthritis, possibly arthritis at
3 least, uveitis, the onset of autism within seven days
4 after, at least that's the understanding that you have
5 in this case, correct?

6 A Yeah.

7 Q Other cases that do not have those features
8 that are important to your theory of causation are in
9 your view supportive of your theory of causation would
10 be weaker, wouldn't they, Doctor?

11 A You're absolutely right. Absolutely.

12 Q So in those cases wouldn't your opinion
13 logically be that you couldn't render an opinion that
14 MMR caused autism?

15 A Well, that certainly sounds logical, but
16 life is really difficult. But let me give you one
17 example.

18 Q It was really just a simple question.

19 A No. It was a good question.

20 Q I don't have many of them. Don't ponder it,
21 Doctor.

22 A We both do our best. Here's my attempt at a
23 good response.

24 Q I think you already had one. As far as I'm
25 concerned your response no, you couldn't offer an

1 opinion in those cases was a good response.

2 Q I understand that it's satisfactory, but I
3 would like to add one point if the Court --

4 SPECIAL MASTER HASTINGS: Please do, Doctor.

5 THE WITNESS: There might be, and in fact
6 I'm sure there are cases which do indeed lack of some
7 of those ingredients like Mr. Matanoski correctly
8 listed but have another ingredient which isn't at
9 issue here, a child that had MMR twice and had an
10 autistic regression twice, in other words, a sort of
11 double hit or challenge recharge.

12 If we have a case like that then I would be
13 much easier convinced even without a number of the
14 other ingredients that we've been discussing. So what
15 I'm trying to do is not to preclude children I haven't
16 even heard about by injudicious testimony at this
17 point. I may not have managed it, but I'm trying my
18 best to do that.

19 BY MR. MATANOSKI:

20 Q Certainly, Doctor, if another case came by
21 and your opinion changed you'd be free to come in here
22 and say why your opinion changed. Now, with the
23 example you just gave, two MMR, and a regression and
24 then a further regression, is that what you're --

25 A Yeah. There are two patterns. I was aware

1 of such cases in Britain, and I do believe they exist
2 even in the current cohort here, but I haven't
3 actually reviewed any. One of two things happens.
4 One of the things that can happen is that there's an
5 MMR, there's the onset within approximate time period
6 of an autistic regression and the child reaches some
7 plateau of dysfunction and then gets better because
8 some children do get better.

9 Some were better, or a lot better, or
10 essentially better and then the second MMR is given
11 and we have another regression. That's one pattern.
12 The other pattern is that the child has an MMR, has
13 the regression, stays at the plateau for a period of
14 months at the second MMR and gets worse still. I have
15 come across examples of both of these on a clinical
16 basis.

17 I mean, not ones that I studied
18 exhaustively, but examples of these, and these are the
19 two situations. Both challenge-rechallenge, which I
20 would find very persuasive even in the absence of some
21 other evidence that you listed.

22 Q From a virological standpoint how is that
23 possible, Doctor? Your theory is the virus is
24 persisting and causing damage. Why would a second MMR
25 make any difference?

1 A It would increase the viral challenge.

2 Q There's more virus now? Is that what it is?

3 A Yes. But, you see, in the challenge-
4 rechallenge, the mechanism is by definition not an
5 issue. You know, as the Institute of Medicine has
6 found it's a situation in which the alternative
7 interpretation of coincidence is so remote that
8 certainly at the standards of reasonable medical
9 probability I think that the criterion is easily
10 reached.

11 Now, what the mechanism is is of great
12 interest but not critical to my opinion in such a
13 case.

14 Q So you may, though you aren't going to be
15 held to this is the way I understand this, if you had
16 a situation where there are two MMRs and you had
17 regression after the first and consistent with your
18 theory that there's persisting virus and then you gave
19 a second MMR and there's, what, more regression? I'm
20 not sure I understand you.

21 A Well, if I could repeat?

22 Q I want to understand what you're saying is a
23 situation where you might still find that.

24 A Absolutely, and by the way, the virus, I
25 mean, it is permitted to disappear. I'm not claiming

1 that once it's persisting it's going to stay forever.
2 I don't know that. I don't know whether some of the
3 virus was eliminated before the second challenge
4 happened. This is all way beyond the level at which
5 we're discussing detail.

6 I just wanted to make clear the point that
7 there are two patterns of challenge-rechallenge. One
8 pattern is that you give the first MMR, there is a
9 regression, there is a measure of recovery, and then
10 after a second MMR that is lost and the child is again
11 more deeply implicated. The second pattern is that,
12 indeed, there is a plateau of autistic deficit after
13 regression which then after the second MMR is made
14 worse yet.

15 I've seen reports, I've seen case files in
16 which either of those two things appear to have
17 happened.

18 Q So this is based on your litigation work?

19 A Well, it's based on my precious clinical
20 experience which I gathered over four arduous years
21 while involved in litigation. Yes.

22 Q So it was involved in your litigation work
23 not based on your --

24 A Yeah. It really tingled my interest.

25 Q Okay. Not based on your research into

1 autism?

2 A Yes. I understand. I am not aware of any
3 article which in fact documents challenge-rechallenge
4 in this context.

5 Q This is your theory?

6 A No. It's my observation.

7 Q It's an observation.

8 A It's my clinical experience in a sense if
9 you can regard reviewing charts as clinical
10 experience. I'm aware of such children, but I'm not
11 aware of a scientific publication which has assembled,
12 done and presented the case.

13 Q So it's based on clinical experience if one
14 considers working on litigation clinical experience?

15 A No. If one considers reviewing medical
16 records. If I review medical records the quality of
17 my review is not impaired by the fact that lawyers are
18 interested in my doing it. I'm reviewing a chart.
19 I'm drawing my conclusions.

20 Q And those charts are given to you for a
21 reason, correct?

22 A Definitely.

23 Q And that's to make a case. Is that right?

24 A Yes.

25 Q Now, you were testifying earlier about the

1 opsoclonus myoclonus condition that you first
2 described 45 years ago?

3 A 1962. Yeah. Correct.

4 Q That's a neuroimmunologic condition?

5 A Yes.

6 Q Clinically what's the first thing that you
7 do when you suspect that's the condition?

8 A One of the first things you do is to test
9 for neuroblastoma.

10 Q How do you do that?

11 A Well, we used to do it be looking at the
12 substance called VMA in the urine, but it was really
13 to inconstant. Nowadays, you'll do an MRI and see
14 whether you can detect a tumor.

15 Q After you do the MRI nowadays what would be
16 the next step?

17 A It depends a bit on what the MRI shows.

18 Q If the MRI doesn't show anything what would
19 be the next step?

20 A Well, actually, you could start your ACTH
21 even before you --

22 Q Would you do a spinal tap?

23 A For the opsoclonus myoclonus? You probably
24 would as part of the investigation, but it doesn't
25 usually give you information that changes anything in

1 practice.

2 A If you suspect it you'd probably do that?

3 A Yeah. You would also do an EEG. I mean, so
4 I didn't know that you were asking about work up.

5 Q Okay. I'm sorry.

6 A You have a child with myoclonic immunology
7 hardly anything is, you know, it's not like shooting
8 pigeons. You very rarely have one diagnosis and
9 that's it, so you always have to rule out and take
10 precautions. In case of a child with myoclonus you
11 want to be sure there's no epilepsy going on, and you
12 do an EEG and you would do a spinal tap and maybe look
13 for some evidence of inherited disorders of
14 metabolism.

15 You know, there is a work up that you would
16 do.

17 Q You mentioned in your report that since 1989
18 you testified in hundreds of vaccine cases?

19 A Yes.

20 Q You were mentioning towards the end of your
21 testimony this morning your work in the litigation in
22 the United Kingdom?

23 A Yes, sir.

24 Q What hourly rate were you paid?

25 A In terms of the exchange rate, which

1 fluctuates of course, it was about \$300 an hour on a
2 par with what I was charging in this program.

3 Q I'm sorry. It's on a par with?

4 A With what my charge is in this program.

5 \$300 an hour has been over a long period my rate. Was
6 at the time my rate is what I'm saying.

7 Q You said you were working on that litigation
8 for about four years. Is that right?

9 A Yes.

10 Q Your work day or work week nowadays is
11 pretty much taken up with some lectures occasionally,
12 but your litigation work you described mostly as being
13 on the weekends?

14 A Yes.

15 Q Now, at that period of time for those four
16 years was your work week similar to now or was it
17 different?

18 A My work week was similar. I didn't have
19 three little girls, who are quite time-consuming, but
20 yes, it was similar.

21 Q Because it's been described that you were
22 spending a lot of time on this litigation. Isn't that
23 accurate?

24 A Yes.

25 Q You were?

1 A Yeah.

2 Q You were spending hours and hours on the
3 litigation?

4 A Absolutely.

5 Q Wasn't it more like a full-time job?

6 A Well, if full-time means 40 hours a day I
7 work much more than that.

8 Q Forty hours a week, you mean?

9 A Sorry. It's getting late in the day. I
10 work a lot more than 40 hours a week. It was a lot to
11 do. Yes. I agree.

12 Q And during that time you were also
13 testifying in vaccine cases here, too, right?

14 A Yes.

15 Q You were spending a lot of time as a
16 litigation witness during that whole period, correct?

17 A Yes.

18 Q And you were talking about discussions that
19 you had with Ms. Chin-Caplan. You were talking about
20 discussions that you had during that litigation.
21 During that time you said that you saw that there were
22 reports exchanged between the different parties, and
23 you got to see not only your fellow experts on the
24 Claimant's side but you also got to see the defense
25 reports?

1 A No.

2 Q You didn't?

3 A We didn't see the defense reports. In fact,
4 we didn't even know who the defense experts were until
5 their definitive reports came in. What we had were
6 interrogatories initiated by the defense.

7 Q So you didn't see the Defendant's reports at
8 that point?

9 A I saw no preliminary reports of the
10 Defendant or that were intermediate reports. I only
11 saw their final opinion reports that were in that.

12 Q Did you meet with the other experts at the
13 same time? Did you have discussions with Claimant's
14 experts?

15 A I met with them numerous times.

16 Q Did those include experts who were talking
17 about the PCR results that were being used in the
18 case?

19 A Yes.

20 Q And those were experts from Unigenetics?

21 A I certainly met Dr. O'Leary and Dr. --

22 Q Did they explain problems that they were
23 having at the Unigenetics lab?

24 A I don't recall that. What problems are you
25 referring to?

1 Q Did they explain that they were having
2 problems getting positive results from negative
3 samples?

4 A This is new to me.

5 Q They didn't discuss that with you?

6 A If there were such problems they would have
7 discussed them with people like Dr. Kennedy and his
8 group of colleagues, and I might have been peripheral
9 to such a discussion because it's not in my field.

10 Q If you were relying, though, on the PCR
11 results --

12 A If something major had happened I would have
13 known. Voice is gone.

14 Q Absolutely. It's been a long time.

15 A Yeah.

16 Q Thank God.

17 A I can't locate that information I'm afraid.
18 I don't remember that, but I'm not testifying it
19 didn't happen, but I think Dr. Kennedy and other
20 colleagues would be better informed.

21 Q There were some peripheral discussions about
22 the Unigenetics lab?

23 A Not particularly.

24 Q So you don't have any knowledge of that?

25 A I have knowledge of the results, but I have

1 no knowledge of any technical issues that might have
2 arisen. They're way outside my expertise.

3 Q If it were shown to you that those results
4 were of questionable reliability would your opinion
5 change?

6 A Well, of course.

7 Q You said that the Legal Services in Great
8 Britain seeks funding of litigation because they felt
9 that they didn't have enough proof to show that
10 measles, mumps, rubella virus is causing autism?

11 A It wasn't really an issue of anybody
12 presenting them proof because they are not the Judge.
13 However, it is a part of their role to monitor the
14 progress of an extended litigation of that kind of
15 scale that I described to you, and the lead barristers
16 on both sides render detailed reports annually on the
17 progress of the litigation.

18 For I guess four or five years the Legal
19 Services Commission had been satisfied that further
20 funding was justified. In other words, that the
21 promise of the litigation was at a sufficient level.
22 Then abruptly the last year given evidence of progress
23 that was actually better, particularly in terms of
24 discovering the cerebrospinal fluid findings, they
25 came to the opinion that the chances of success were

1 now below 50 percent.

2 So I'm relating what happened factually.

3 I'm not privy to their state of mind.

4 Q The cerebrospinal fluid findings that you
5 were talking about, were those from Unigenetics?

6 A Yes.

7 Q And those findings were never published?

8 A Those particular ones were not. There were
9 three cases. No, they weren't published.

10 Q And Unigenetics is the same lab with Dr.
11 O'Leary, Dr. Uhlmann and Dr. Martin. Is that right?

12 A Correct.

13 Q This was in late 2003 as you recall?

14 A I think maybe in early 2003. I have some
15 notion it might have been about April, but I won't
16 swear to it.

17 Q Now you mentioned the distinguished
18 individuals on that list of individuals who had
19 received money from Legal Services for their
20 participation on behalf of the claimants in the U.K.
21 MMR litigation.

22 A The ones who were consulting and were paid a
23 consulting fee, yes.

24 Q You mentioned that there are some very
25 distinguished people on that list.

1 A Yes.

2 Q You didn't mention Andrew Wakefield as one
3 of the distinguished people.

4 A I was talking about consultants attracted to
5 the problem from several countries, from many
6 disciplines. Andrew Wakefield was there from the
7 beginning. I do believe he is distinguished if that's
8 the question you're asking me. I wasn't leaving him
9 out as a pointed gesture.

10 Q In your expert report, you rely on Dr.
11 Wakefield's work, including his 1999 report, as
12 evidence of a condition that too described an autistic
13 enterocolitis condition.

14 A Yes, I refer to his work, and I work with
15 his group on that topic.

16 Q Were you aware that undisclosed at the time
17 of the publication of that work Dr. Wakefield had been
18 contacted by Legal Services and lawyers for Legal
19 Services to be involved in the U.K. MMR litigation?

20 A I mean, I have become aware of that. Yes.

21 Q Okay. You've become aware of that?

22 A Right. I mean, I wasn't around at the time,
23 but yes.

24 Q And were you aware that 1998 *Lancet* article
25 Dr. Wakefield didn't disclose that he had actually

1 received money from lawyers bringing litigation
2 against MMR alleging that MMR caused autism?

3 A Right. I was aware of two more things. May
4 I mention them?

5 Q Certainly.

6 A One is that some weeks, I think six weeks,
7 after publication Dr. Wakefield wrote a letter in
8 response to another letter in which he mentioned that
9 involvement. So the information was available to
10 *Lancet* in that general time period and it didn't cause
11 the outrage which it caused many years later.

12 The second thing that I'm aware of because I
13 happened to look through the volume *Lancet* 1998, I
14 couldn't find anybody who had disclosed conflicts of
15 interest, and I have trouble envisioning an enormous
16 number of -- this is a fat volume -- investigators all
17 sending in experiments worth publishing in *Lancet*,
18 which is an outstanding journal, with no conflict of
19 interest.

20 So it seems to me, although I'm sure others
21 could speak to this better than myself, that it wasn't
22 at the time standard practice to communicate that kind
23 of information when you submitted articles because why
24 is there nothing there anyplace? Unless I overlooked
25 something.

1 Q Are you aware of Dr. Wakefield ever making
2 that apparent to the general public in Great Britain
3 or the world that he had this potential conflict of
4 interest at any time before it became apparent by the
5 publication of the newspaper article reviewing that?

6 A Well, I just testified that he actually
7 informed the journal itself of this matter within
8 weeks of the publication of the article. So it's not
9 as if he seemed to be hiding it.

10 Q He appeared at numerous news conferences.
11 Did he ever disclose it at that point?

12 A I certainly wasn't present for these news
13 conferences, but I'm trying to imagine a scientist
14 going to a news conference or giving a public lecture
15 and saying by the way, I've told you what my work is,
16 but don't forget I was in conflict of interest. I
17 don't think it's customary practice to do that. I
18 think if there is a conflict or you believe there is
19 one you make it known and that's it.

20 By the way, what happens is not that the
21 journal turns you down, it is that they record the
22 conflict of interest so the reader can decide what
23 weight to give to the findings in view of that. But
24 once you've done that you don't run around mentioning
25 it every time you give a lecture on the matter, and I

1 wouldn't expect him to do that.

2 Q I want to come back to that in a moment. I
3 just wanted to actually ask you something back to your
4 report in another matter. You mentioned Dr.
5 Bradstreet's CSF finding as important to your thinking
6 in this case.

7 A Yes.

8 Q Doctor, do you know Dr. Bradstreet?

9 A I've met him. I don't know him.

10 Q Do you know -- and this is the part of your
11 report that's coming up now. Now, he's not a
12 neurologist, is he?

13 A No, no. Not at all.

14 Q He's not a virologist, is he?

15 A I don't think that he has a particular
16 specialty. I'm not totally sure of that, but I'm not
17 aware of him being a specialist in the domains that
18 we're discussing.

19 Q Do you know how he recovered measles vaccine
20 virus material from cerebrospinal fluid?

21 A Well, he didn't recover it. I mean, he sent
22 it to Ireland.

23 Q So it was sent to the same lab?

24 A Yes.

25 Q Unigenetics?

1 A Correct.

2 Q Do you know if the journal that he published
3 these results in is indexed?

4 A Which journal was it in?

5 Q You cited it, Doctor.

6 A Yeah. Okay. I think it was probably the
7 *Journal of American Physicians and Surgeons* or some
8 such title.

9 Q That's what it was.

10 A Okay. I don't know much about the *Journal*.

11 Q So you don't know whether it was indexed?

12 A Whether it's?

13 Q It's indexed?

14 A I have no idea.

15 Q Do you know what the term indexed means?

16 A Sorry. Could you say it again?

17 Q Do you know what the term indexed means in
18 relation to a journal?

19 A In the indexed medicals? Maybe I should
20 know something. I don't.

21 Q Do you publish important work in nonindexed
22 journals?

23 A Apparently not. When I've sent an article
24 for publication I've never first tried to determine
25 whether the journal was indexed mostly because I don't

1 know what that means.

2 Q That's fine, Doctor. So Dr. Bradstreet is
3 not a neurologist, he's not a virologist. He's
4 reporting on a neurologic and virologic finding. Is
5 that right?

6 A Correct.

7 Q Do you know anything else about him? I
8 mean, you know of him?

9 A I know of him. I know he has a practice in
10 Florida someplace, and he sees a lot of autistic
11 children. That's pretty much what I know.

12 Q And he sells them medication?

13 A He does what with them?

14 Q He has a mail order business to sell them
15 medication?

16 A I'm not aware of that. I don't know.

17 Q Do you know that he calls himself the good
18 news doctor? Were you aware of that?

19 A No. It's pretty funny. No, I didn't know
20 that.

21 Q And he has a website to that effect?

22 A I'm interested to hear it.

23 Q I'm turning back to the *Lancet* article in
24 1998.

25 A Yes, sir.

1 Q You said that you know that Dr. Wakefield
2 wrote a letter to the journal disclosing the conflict
3 of interest?

4 A I don't think he was disclosing it if he was
5 referring to it. I don't think he was aware that he
6 was in conflict, but my information is that he
7 referred to the conflicts of the work in that letter.

8 Q Now, a potential conflict of interest can
9 influence whether a journal will go forward and
10 publish an article, can't it?

11 A Certainly, but in fact what the journals do
12 is simply record it and let the reader decide. If
13 they didn't do that you would get hardly any
14 pharmacological information published because the
15 realities of the surge on drugs including vaccines is
16 that it's expensive and the NIH can't be relied upon
17 to fund it fully and many very, very reputable
18 investigators are heavily supported by industry.

19 It would be absurd not to publish their work
20 because of that.

21 Q I understand that, Doctor. It's a factor,
22 though, that journals may consider in deciding whether
23 or not to publish an article, correct?

24 A If it was exceptionally egregious in its
25 appearance, but I don't want to say it would have to

1 be that the person was -- I don't want to make it up.

2 It would have to be an exceptional case.

3 Q Didn't the *Lancet* say that this was a very
4 critical factor for them that was not disclosed to
5 them at the time of publication? Didn't they say that
6 in 2004?

7 A In 2004, they said it. In 1998, they
8 didn't.

9 Q In 1998 they didn't when Dr. Wakefield
10 didn't disclose it to them.

11 A Right, but they didn't after that letter
12 either. I don't know what they're thinking of, but it
13 seems to me that *Lancet's* reaction on the scientific
14 point of view is totally out of proportion to the
15 infraction that they are complaining about.

16 Q In your view, it's out of proportion?

17 A Well, I'm testifying. In my view, yes. It
18 really requires further explanation in my opinion.

19 Q The *Lancet* actually published that it was
20 material to them and it had not been disclosed. They
21 put that in their publication, did they not?

22 A You mean if I understand you correctly, sir,
23 they published the fact that it hadn't been disclosed.
24 Is that what you're saying?

25 Q Yes.

1 A Yes, I'm aware of that.

2 Q And that was material to them?

3 A Well, enough where they had some reason for
4 publishing it.

5 Q And of the 12 authors of that article 10
6 could be contacted, 10 and Dr. Wakefield, one could
7 not be contacted, but of those who could be contacted
8 all but Dr. Wakefield pulled their support for that
9 report. Isn't that right?

10 A No, it's wrong. That's completely wrong.

11 Q If you look on the screen there's retraction
12 of interpretation, and it's signed by 10 of the 12
13 authors of that article.

14 A Right. Well, that's different from what I
15 heard you say before. Let me explain that because
16 it's an important point. What you have in the 1998
17 article, which incidentally is only one of many but
18 somehow always seems to be the topic of discussion,
19 are findings. It's a report. It reports findings.

20 Not a single finding in that report has been
21 retracted. Now, it says an interpretation was
22 retracted, the interpretation that the MMR caused
23 these children's problems, but there is no such
24 interpretation in the original article. All that they
25 did was to mention that it's possible that the MMR did

1 this.

2 And what these 10 poor people retracted was
3 a possibility. Now, it's probably the first time in
4 the history of medical science that somebody has
5 retracted a possibility because the alternative to a
6 possibility is an impossibility, and doctors don't
7 generally say that things are impossible. So I don't
8 know what went on in the mind of these 10 people that
9 six years after the event they suddenly woke up and
10 thought, we need to retract this.

11 Q Six years after the event, shortly after it
12 was disclosed to the public this conflict of interest
13 that Dr. Wakefield had. Is that right?

14 A Well, they must have had a reason.

15 Q And those were his co-authors?

16 A They were his co-authors. Yes.

17 Q Were you aware, Doctor, that at the time
18 that Dr. Wakefield published that 1998 report he had
19 on file a patent for a monovalent measles vaccine, and
20 that he stood to gain financially from any dispute
21 over the use of MMR?

22 MR. MATANOSKI: Yes, sir?

23 SPECIAL MASTER HASTINGS: Before we go on to
24 that one, Mr. Matanoski, let's just make our record
25 here. Just a minute ago you showed a document that

1 was headlined Retraction of an Interpretation.

2 MR. MATANOSKI: Yes, sir.

3 SPECIAL MASTER HASTINGS: Is that already in
4 the record?

5 MR. MATANOSKI: I believe it is, sir.

6 SPECIAL MASTER HASTINGS: I believe it is.
7 For the record, does anybody know offhand where that
8 is? If you don't, that's okay, we can find it. Okay.
9 So we don't need to mark that as a trial exhibit. I
10 just wanted to clarify that. Okay. Now you're going
11 on to a different document. Go ahead, sir.

12 THE WITNESS: I remember the question.
13 Sorry.

14 BY MR. MATANOSKI:

15 Q Were you aware that he stood to gain
16 financially from any criticism that would be generated
17 of the MMR vaccine? If that vaccine were to fall into
18 disuse he stood to gain financially.

19 A Okay. Two responses. One is if that were
20 really true as opposed to being a myth then that would
21 be reprehensible. Secondly, at this point I wonder
22 why we're discussing Dr. Wakefield.

23 Q You cited him, Doctor. Your part of this
24 whole case is MMR autism. That's what he was writing
25 on, that's his theory.

1 A It's true he and numerous co-authors were
2 presenting data which was not challenged and not
3 disconfirmed by the *Lancet*. What I'm saying is I have
4 no reason to believe that Dr. Wakefield, whatever his
5 financial involvement that you represent to me
6 occurred, that his findings were inaccurate. Now, as
7 to his interpretations I don't have to accept them. I
8 make my own interpretations.

9 SPECIAL MASTER HASTINGS: Before we go on --

10 MR. MATANOSKI: I don't believe you have the
11 actual patent application.

12 SPECIAL MASTER HASTINGS: Okay. The patent
13 application was the document you just mentioned.

14 MR. MATANOSKI: We'll make that a trial
15 exhibit, sir. I'm not sure that's in the file.

16 SPECIAL MASTER HASTINGS: Okay. That would
17 be No. 7.

18 MR. MATANOSKI: Okay.

19 BY MR. MATANOSKI:

20 Q And, Doctor, isn't it true that of the
21 payments made for the U.K. litigation the top three
22 recipients of payment involved in that litigation were
23 Unigenetics, Dr. Wakefield and yourself?

24 A Correct.

25 SPECIAL MASTER HASTINGS: Now, again, that

1 was Trial Exhibit 6. I'd like to mention, why I do
2 this, folks, is that when I go back and read the
3 transcript, when we each do, if it's mentioned in the
4 record in the transcript what document we're looking
5 at it makes an awful lot easier to find at that time.

6 So for that purpose I will also say the
7 document about the retraction of the interpretation
8 was mentioned several places in the record including
9 Exhibit P, Tab 114. Go ahead, Mr. Matanoski.

10 MR. MATANOSKI: I'm finished at this time.

11 SPECIAL MASTER HASTINGS: Okay. You're done
12 at this point. All right.

13 Ms. Chin-Caplan, any redirect?

14 Wait, wait. Before we do that do Special
15 Masters?

16 (No response.)

17 SPECIAL MASTER HASTINGS: I think now would
18 be a great time for a 15 minute break. We will take a
19 15 minute break and then be back.

20 (Whereupon, a short recess was taken.)

21 SPECIAL MASTER HASTINGS: All right. We're
22 back in session here. Dr. Kinsbourne is back at the
23 witness table.

24 Ms. Chin-Caplan, did you have any redirect?

25 MS. CHIN-CAPLAN: No, Special Master.

1 SPECIAL MASTER HASTINGS: All right. Any
2 questions from the Special Masters?

3 (No response.)

4 SPECIAL MASTER HASTINGS: All right. Then,
5 Ms. Chin-Caplan, I assume that concludes the
6 Petitioners' case in chief.

7 MS. CHIN-CAPLAN: Yes, it does, Special
8 Master.

9 SPECIAL MASTER HASTINGS: All right. Then
10 that's all the testimony we're going to take this
11 week. Are there any matters, counsel, that we should
12 talk about on the record before we adjourn for the
13 day?

14 Mr. Matanoski, anything?

15 MR. MATANOSKI: No, sir.

16 SPECIAL MASTER HASTINGS: Ms. Chin-Caplan,
17 anything?

18 MS. CHIN-CAPLAN: No, sir.

19 SPECIAL MASTER HASTINGS: All right. Well,
20 then, to all the folks here in the courtroom and those
21 listening in we are done for the day and for the week.
22 We will start again on Monday morning with the
23 beginning of the Respondent's case. We have Dr.
24 Fombonne and Dr. Cook slated to testify on Monday, and
25 we thank you all for your patience and your attendance

1 this week. We are adjourned until Monday, 9:00 a.m.

2 (Whereupon, at 3:27 p.m., the hearing in the
3 above-entitled matter was recessed, to reconvene
4 Monday, June 18, 2007, at 9:00 a.m.)

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REPORTER'S CERTIFICATE

DOCKET NO.: 98-916V
CASE TITLE: Theresa Cedillo v. HHS
HEARING DATE: June 15, 2007
LOCATION: Washington, D.C.

I hereby certify that the proceedings and evidence are contained fully and accurately on the tapes and notes reported by me at the hearing in the above case before the Office of Special Masters.

Date: June 15, 2007

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