

SUPERIOR COURT OF WASHINGTON FOR KING COUNTY

SANDRA LOTT, Individually and as)
 Personal Representative of the Estate)
 Of TIMOTHY LOTT,)
)
 Plaintiff,)
) No.
 vs.) 05-2-06955-4 SEA
)
 BONDEX INTERNATIONAL,)
)
 Defendant.)

FRYE HEARING

(Transcribed from Audio Recording)

November 6, 2006

Seattle, Washington

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1 BE IT REMEMBERED that on Monday,
2 November 6, 2006, before The Honorable Sharon Armstrong, the
3 following proceedings were had, to wit:

4
5 <<<<<< >>>>>>

6
7 THE COURT: Good morning. Please raise
8 your right hand.

9
10 CARL ANDREW BRODKIN, MD, MPH, having been first duly
11 sworn, testified as
12 follows:

13
14 THE COURT: Please be seated and pull up
15 a microphone.

16
17 DIRECT EXAMINATION

18 BY MR. FROST:

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

20 A Carl Andrew Brodtkin.

21 Q And, Dr. Brodtkin, as you've heard, we're going to stipulate
22 to the admission of your curriculum vitae. But very
23 briefly, one of your professional interests is the
24 relationship between asbestos and disease?

25 A Yes. I've been a physician in occupational medicine who has

1 treated and evaluated a number of patients over the
2 years with occupational asbestos exposure and related
3 disease.

4 Q And have you had an opportunity to review Bondex's
5 motion to exclude certain testimony from Dr. Maddox in
6 this case?

7 A I have been provided and discussed issues regarding
8 cumulative exposure to asbestos and whether that issue
9 should be considered in excluding testimony, yes.

10 Q Okay. And do you have an opinion, sir, about the
11 relationship between cumulative exposure to asbestos
12 and the development of mesothelioma?

13 A Yes, I do. This is an issue I have given some thought
14 over the years, yes.

15 Q Okay. What is that opinion, sir?

16 A I believe the body of scientific literature has well
17 established that mesothelioma is a dose response
18 disease. With increasing dose, there is increasing
19 risk for development of mesothelioma in individuals.

20 The dose at which this occurs, I believe the
21 literature has shown, is substantially above
22 background levels, ambient levels, which are quite low
23 in the environment and really represent very
24 identifiable over exposures to asbestos that result in
25 cumulative exposure.

1 Certainly the literature has shown that this
2 linear relationship can occur at low doses of asbestos
3 exposure, but increases as -- the risk increases as
4 the dose increases.

5 Q So if I can summarize, your opinion is that every
6 identifiable occupational or environmental exposure to
7 asbestos, does that contribute to the risk of
8 development of mesothelioma?

9 A Well, I would be very clear in saying not every
10 exposure to asbestos fibers contributes to risk for
11 developing mesothelioma. The body has protected
12 mechanisms of clearing particulates, including
13 asbestos fibers, and certainly minimal exposures, many
14 ambient exposures, would not result in a significant
15 biological dose.

16 However, identifiable exposures where high
17 asbestos fiber levels exist in the air and are
18 available for inhalation would certainly result in
19 increased risk for mesothelioma, and those should be
20 identifiable exposures

21 Q Have you had an opportunity to review the deposition
22 testimony of Mr. Timothy Lott?

23 A Yes, I have.

24 Q And in reviewing that testimony, did you see any
25 identifiable asbestos exposure?

1 A I did. In my review of Mr. Lott's testimony, he
2 reviewed his occupational history between 1973 and
3 1976, and while working at the Jewel Lake Villa
4 Apartments regularly participated in application,
5 sanding and sweeping of joint compounds related to
6 patching of drywalls and repair of apartments in
7 preparation for residents.

8 Q So and is it your testimony today, sir, that that
9 exposure to asbestos contributed to the development of
10 Mr. Lott's mesothelioma?

11 A Well, these are identifiable exposures that would
12 contribute. Joint compound is a substance that has
13 been well studied. Fiber levels associated with
14 sanding, mixing, sweeping have been well characterized
15 in studies from the 1970's and result in significant
16 fiber cc levels and certainly would be identifiable
17 levels that would contribute to mesothelioma risk.

18 Q Sir, your opinion that identifiable exposures to
19 asbestos contribute to the risk of development of
20 mesothelioma, can you tell us what that is based on,
21 sir?

22 A The scientific methodology for determining a cause and
23 effect relationship, and in Mr. Lott's case this deals
24 with chrysotile exposure causing mesothelioma, has
25 been characterized and summarized in a group of

1 criteria known as the Bradford Hill criteria. This
2 was put forward by Dr. Hill in the mid 1960's, and I
3 think is the most recognized and commonly used
4 criteria used by the medical community to assess
5 causation and whether an exposure such as chrysotile
6 would be associated with mesothelioma.

7 Q And the Bradford Hill methodology, is that one that is
8 generally accepted among the scientific community for
9 the determination of cause and effect of
10 asbestos-related disease?

11 A I would say that it's widely and uniformly accepted.
12 It's something that I was taught when I was getting my
13 master's in public health degree and is something that
14 I have used frequently over the years, and I think
15 other physicians in my field and other fields
16 evaluating exposures would routinely use, yes.

17 Q I wonder if you could just take us very quickly
18 through the Bradford Hill criteria?

19 A Yes. There are nine criteria, and in discussing it,
20 this would certainly relate to chrysotile exposure and
21 cumulative exposure and mesothelioma. That's the
22 issue at hand.

23 The Bradford Hill criteria stipulate nine
24 criteria. The first one is what is known as strength
25 of association, and this relates to epidemiologic

1 studies, studies of disease in human populations, in
2 this case mesothelioma in human populations, and
3 ranging from low exposures, for instance, in a study
4 by Welch in a recent publication in the International
5 Journal in 2005, to high exposures that Yano described
6 in American Journal of Industrial Medicine in 2001.
7 We consistently see in this literature a very high
8 magnitude of risk associated with chrysotile exposure
9 for both peritoneal mesothelioma and for pleural
10 mesothelioma.

11 And as well as the epidemiologic studies, lung
12 burden studies, such as Roger's study in the early
13 1990's, show that as chrysotile fibers increase in the
14 lung, the risk for mesothelioma increases dramatically
15 16 fold.

16 So whether one looks at epidemiology or lung
17 burden studies, one consistently sees a strong
18 strength of association. And that's very important
19 medically because strength of association is how you
20 assess attributable risk, whether a disease can be
21 attributed more than 50 percent to an exposure.

22 And often these relative risks are as high as five
23 or greater, which would certainly correlate with an
24 attributable risk greater than 50 percent.

25 The second criteria --

1 Q Hold on just one second. The two methodologies that
2 you mentioned in support of the first criteria were
3 epidemiological studies and also lung fiber burden
4 analysis?

5 A Yes.

6 Q Are both of those methodologies widely used and
7 generally accepted in the medical and scientific
8 communities?

9 A Yes.

10 Q Their uses aren't limited to just asbestos, is it?

11 A No, these would be generally accepted methodologies.

12 Q How about the second criteria?

13 A The second criteria would be temporal association.
14 The exposure to the punitive agent, chrysotile, should
15 precede the disease mesothelioma by an appropriate
16 period of time, which we term the latency period.

17 And in mesothelioma, this has been well studied.
18 Groups that I've been involved in have looked at this
19 and published it in a journal of the National Cancer
20 Institute. And, basically, one sees a latency of
21 certainly over 10 years and often decades between
22 exposure and development of mesothelioma.

23 So I think the literature supports the case of an
24 individual exposed in the mid 1970's developing
25 mesothelioma in 2003 would fit fell within that

1 latency period

2 Q The third criteria?

3 A The third criteria is one that we call biologic
4 gradient or dose response. If there truly is
5 causation, one typically sees a dose response, the
6 greater the dose, the greater the response. And I
7 think that has been well characterized for all
8 asbestos fibers in mesothelioma, and it has certainly
9 been characterized for chrysotile exposure.

10 Studies that have looked at this have really
11 defined levels of exposure, cumulative levels of
12 exposure, where individuals become at risk and
13 actually develop mesothelioma. And I think some
14 important studies would include the study by Iwatsubo
15 in American Journal of Epidemiology in 1997 where at a
16 dose of .5 to .99 fiber cc years, a low exposure, but
17 a very identifiable exposure, there's about a four
18 fold increased risk in mesothelioma.

19 And then other studies in the German Registry by
20 Rodelsperger in American Journal of Industrial
21 Medicine in 2001 very consistently show that at .15
22 fiber cc years, you see about an eight fold increased
23 risk in mesothelioma. And certainly the risk
24 increases as the dose increases, but these are the
25 doses where one starts to see increasing risk and I

1 think firmly establishes a dose response.

2 And briefly the other studies that I look at are
3 studies that look at time, how long workers have
4 worked with asbestos-containing materials. And in a
5 number of the important studies of asbestos cement
6 workers new New Orleans that Hughes and Weill
7 published and other studies, they find that workers
8 that have worked less than one year have developed
9 both pleural and peritoneal mesotheliomas at high
10 risk.

11 So while certainly workers with high durations of
12 exposure of many years are at greater risk, the
13 workers with less than a year of exposure certainly do
14 develop this condition in chrysotile environments.

15 Q The next Bradford Hill criteria?

16 A The next criteria would be one of consistency. In the
17 epidemiologic literature, one should see a consistency
18 of the magnitude of risk. And this is really well
19 demonstrated in chrysotile exposure. If you look at
20 Quebec miners and millers, if you look at textile
21 workers in the Carolinas, if you look at asbestos
22 cement workers in New Orleans or if you look
23 internationally to South Africa or France or Germany,
24 studies consistently show a dose response of
25 increasing exposure, including chrysotile and

1 development of mesothelioma.

2 Now, I think this has best been described by
3 Leslie Stayner in the American Journal of Public
4 Health in 1996 who did a nice review of this and
5 concluded that chrysotile is indeed a potent
6 carcinogen that causes mesothelioma.

7 And although one may see differences in potency
8 among the fibers, and certainly you do, all fiber
9 types are carcinogenic.

10 Q And I think we're on No. 5?

11 A I've lost count, but I can keep going.

12 Q Okay.

13 A In addition to consistency, one wants to see
14 biological evidence or biologic plausibility that an
15 exposure such as chrysotile can cause mesothelioma,
16 and that really relates to the mechanism of disease.
17 And while medical science is always refining and
18 learning about the mechanism of disease, for an
19 exposure like asbestos, this has really been very well
20 described and was reviewed in the New England Journal
21 of Medicine, one of the leading medical journals, last
22 year by Robinson.

23 And the mechanisms include both tumor initiation
24 through damage of the genetic material in the lining
25 of the pleura or the peritoneum by asbestos fibers, by

1 a variety of mechanisms, changes in the pattern of
2 division of the mitotic division of cells that
3 encourage development of cancers, and inflammatory
4 responses, direct physical injury to the lining of the
5 lung or peritoneum by asbestos fibers.

6 And these all provide a cogent mechanism for
7 development of mesothelioma that I think has been well
8 established.

9 Q How would you describe the science that you just
10 talked about? Is that molecular biology?

11 A That is what I would describe generally as basic
12 science and cellular biology. Quajaron (sic.) who has
13 studied this significantly and published in
14 Environmental Health Perspectives in the late 1990's
15 basically looked at cellular models and looked at
16 mesothelioma at the cellular level and molecular
17 level.

18 Q And the methods used by cellular biologists, are they
19 generally accepted in the field of science in
20 medicine?

21 A Yes, they are an important complement to epidemiologic
22 literature which looks at human experience and
23 basically looks at it, if you will, from another
24 perspective.

25 Q The next Bradford Hill criteria?

1 A In addition to mechanisms and biologic plausibility,
2 one wants to see specificity. A specific exposure
3 should cause a specific effect. In this case, we have
4 asbestos fibers and chrysotile fibers, and we have
5 mesothelioma. I think there has been rarely a disease
6 described where such a strong specificity exists for
7 an exposure such as asbestos as preeminently causing a
8 specific disease. So obviously mesothelioma would
9 well meet the specificity criteria.

10 Q And the next criteria?

11 A The next criteria would really be experimental models.
12 Does the punitive exposure reproduce disease in an
13 experimental model? If you expose as animal to
14 chrysotile, will that animal develop mesothelioma?

15 And an example of this would be the study done by
16 Davis in the early 90's in British Journal of
17 Experimental Pathology where various fibers,
18 chrysotile, amosite, crocidolite, were injected into
19 the peritoneal mesothelial lining of rats. And all
20 fiber types cause mesothelioma. In this animal model,
21 actually chrysotile was the most potent of those three
22 fibers in causing mesothelioma. But this would be an
23 example where direct experimentation with animals
24 reproduces what happens with humans and is strong
25 evidence of the causal association.

1 Q The use of animal studies, is that a principal that is
2 generally accepted and widely used throughout the
3 science and medicine, not just in the asbestos field?

4 A Yes, I would say it is a complement to epidemiologic
5 studies. It allows for control of the environment in
6 a way usually that can't be achieved in human
7 populations.

8 Q And the next Bradford Hill criteria?

9 A There are two remaining Bradford Hill criteria that I
10 generally link or lump together, and Bradford Hill
11 caused these analogy and coherence. And basically
12 what that means is that a similar group of exposures
13 should cause the same disease. And obviously asbestos
14 fibers would be a strong example of this, where
15 various fiber types, though not identical, have
16 similar properties, have overlapping properties, if
17 you will. And certainly the various fiber types,
18 amosite, crocidolite, chrysotile, while may have a
19 variable potency, certainly cause a similar effect in
20 mesothelioma.

21 And the sort of flip side of that is coherence.
22 Is there a compelling reason why one similar exposure
23 shouldn't cause the disease that other similar
24 exposures cause? And I would say in this case, no,
25 that chrysotile from a biologic or medical perspective

1 would be anticipated to cause similar disease as
2 amphibolic fibers.

3 So those are the nine criteria. And I think in
4 the case of asbestos in general and chrysotile
5 specifically, the criteria are well met in terms of
6 cumulative exposure and development of mesothelioma.

7 Q During your testimony today, I think you mentioned
8 epidemiology, you've mentioned lung fiber burden
9 analysis, you've mentioned cellular biology, and you
10 have mentioned animal studies. Are all of those tools
11 generally used and widely accepted throughout the
12 scientific community?

13 A Yes.

14 Q And it's fair to say that certain other experts may
15 look at the information derived from those tools and
16 reach a different conclusion than you have reached?

17 A I think part of the scientific process is discussion
18 and debate about studies. And certainly in the
19 epidemiologic literature, no study is exactly
20 reproduced in another study. And this is an important
21 basis for debate and discussion, and certainly there
22 are disagreements among experts.

23 Q But there's no disagreement that those tools that
24 you've talked about are generally accepted and widely
25 used throughout science?

1 A I don't believe there would be any significant
2 degree -- disagreement that the Bradford Hill criteria
3 are not an appropriate way to look at the scientific
4 evidence in terms of evidence of causation.

5 MR. FROST: Thank you. I don't have
6 any additional questions.

7 MR. OSBURN: Thank you, Your honor.

8
9 CROSS-EXAMINATION

10 BY MR. OSBURN:

11 Q Dr. Brodtkin, what was Mr. Lott's dose of joint -- of
12 chrysotile asbestos from joint compound over his life?

13 A In my review of Mr. Lott's deposition, he described
14 fairly regular use of joint compound between 1973 and
15 1976 when working after school with his father. The
16 specific types of activities would include mixing. He
17 used some pre mix, but also a dry mix joint compound.
18 He applied it during his patching work. He performed
19 sanding procedures and performed clean-up procedures.

20 In addition to that direct exposure, he assisted
21 his father and describes holding drywall at very close
22 proximity to manipulation of joint compounds. So I
23 think there would be a direct and bystander exposure.

24 Certainly joint compound during the mid 1970's
25 when Mr. Lott worked in the apartments is known as an

1 asbestos-containing material generally had a 4 to 12
2 percent asbestos content.

3 Q Maybe you missed my question. How often did he do it
4 and what was the exposure to chrysotile? What are the
5 numbers?

6 A My knowledge of his exposure relates to his
7 deposition. He testified that he worked on apartments
8 during the winter months approximately two to three
9 times weekly, regularly performed manipulations of
10 joint compounds. So those would represent his
11 exposures.

12 In terms of the fiber cc levels, obviously he did
13 not have a dosimeter on, but in terms of the
14 activities he was doing, for instance, mixing in
15 various studies has been shown to result in up to 30
16 fiber per cc exposure.

17 Sanding can result in at least a mean of 5 fiber
18 per cc with maximum 19 to 20 fiber cc exposure.
19 Clean-up activities similarly can result in high fiber
20 cc levels. So those would be the types of exposure
21 that Mr. Lott would experience during that three-year
22 period.

23 Q But you can't quantify his exposure to joint compound,
24 can you?

25 A My knowledge --

1 Q Quantify it?

2 A My knowledge is from his description of the
3 deposition. I'm not aware that specific levels were
4 measured during his work.

5 Q And you can't quantify Mr. Lott's exposure to, say,
6 Bondex joint compound, which is what this trial is
7 about, right?

8 A In the deposition, Mr. Lott described working with
9 several joint compounds, among them Bondex.

10 Q And you can't quantify his exposure to Bondex?

11 A No. Certainly Mr. Lott did not discuss that in his
12 deposition.

13 Q You mentioned the Yano study. That is a high
14 exposure, is it not?

15 A The Yano study would be a high exposure study, yes.
16 Generally about five to seven fiber cc in that cohort
17 setting.

18 Q Was any fiber burden analysis done in this case?

19 A I'm not aware in my review of the materials that any
20 fiber burden analysis was performed.

21 Q Was there any radiological evidence of asbestosis in
22 Mr. Lott?

23 A In my review of Dr. Maddox' report and Dr. Maddox'
24 deposition, there was no clear evidence of pleural or
25 parenchymal fibrosis.

1 Q Was there -- there was no radiological evidence of
2 pleural plaques in Mr. Lott, was there?

3 A I don't believe so. There was unilateral
4 calcification, but the description of it was a
5 development of a calcification fairly acutely, which
6 can happen with mesothelioma. I don't believe it was
7 observed on the first imaging study, which I think
8 would weigh against that being a plaque.

9 Q There isn't any biological or radiological evidence
10 that Mr. Lott's exposure to chrysotile asbestos caused
11 his mesothelioma?

12 A Well, there's strong biological evidence obviously of
13 mesothelioma. Dr. Maddox' pathologic description
14 describes both histology and immunohistochemistry,
15 which were very specific for mesothelioma. There is
16 nothing intrinsic to the pathology of mesothelioma
17 that says it's an asbestos-related mesothelioma.

18 One has to look at various criteria, which can
19 include biologic effects such as plaques and fibrosis,
20 but in most cases involves review of occupational
21 history because at least 60 percent of cases of
22 mesothelioma do not have pleural plaques or evidence
23 of parenchymal fibrosis at the time of presentation.

24 Q So in this case there is only Mr. Lott's history.
25 There's no radiological evidence? There's no

1 biological evidence? Correct?

2 A There is the evidence of mesothelioma and there is the
3 occupational history in my opinion.

4 Q And you can't quantify the occupational history; is
5 that correct?

6 A One has to look really qualitatively at the types of
7 exposure and then look at the science in terms of what
8 that exposure represents.

9 And I think in the case of joint compounds, this
10 is one of the more well-studied exposures where
11 activities such as mixing, sanding, sweeping, have
12 been well quantified in a number of studies
13 consistently in terms of fiber cc levels.

14 Q You are obviously familiar with Dr. Richard Lemen?

15 A Yes.

16 Q Dr. Lemen came up with a criteria when he worked with
17 OSHA for exposure to essentially a threshold level of
18 chrysotile asbestos which caused mesothelioma,
19 correct?

20 A Well, Dr. Lemen has contributed widely to the
21 literature. He has published in numerous areas.

22 Q And one of his conclusions in one of his writings was
23 that he required a 0.1 fiber cc year exposure to
24 attribute mesothelioma to chrysotile asbestos,
25 correct?

1 A I'm not specifically aware of that. In terms of .1
2 fiber per cc, my understanding of that level really
3 relates to Dr. Stayner and Dr. Lemen's work in terms
4 of asbestos-exposed workers in the Carolinas.

5 And basically at that level, .1 fiber per cc,
6 which ultimately became the permissible exposure
7 limit, over a 40-year working career, you would have a
8 four fiber cc year exposure. And at those dose, they
9 saw two cases per thousand of asbestosis and more
10 cases of lung cancer. They didn't characterize
11 mesothelioma as such, even though peritoneal and
12 pleural mesotheliomas were found in that cohort.

13 But I'm not aware of any threshold. In fact, in
14 that study and others published by Stayner, they are
15 very clear in saying there is not a threshold level at
16 which suddenly no risk is encountered with asbestos
17 exposure.

18 Q Actually I'm not asking about Dr. Lemen. You probably
19 haven't read his testimony in the Pell versus Amitek
20 case, have you, Brazoria County?

21 A I have not.

22 Q Okay. Dr. Lemen was asked, "Do you believe there it
23 been demonstrated that exposure to chrysotile causes
24 peritoneal mesothelioma?" And Dr. Lemen reports that
25 there is very little evidence on chrysotile and

1 peritoneal mesothelioma. He has not seen any
2 epidemiological studies. And he finally says, "I
3 would say there are no epidemiological studies that
4 have been able to demonstrate chrysotile in its
5 greatest form or any other form have caused peritoneal
6 mesothelioma."

7 And we're talking about peritoneal mesothelioma in
8 Mr. Lott's case, aren't we?

9 MR. FROST: Your honor, if he's
10 going to use depositions, we would request copies of
11 those because --

12 THE COURT: That's appropriate.

13 MR. FROST: -- Dr. Lemen has given
14 hundreds of depositions.

15 THE COURT: You will need to provide
16 copies.

17 MR. OSBURN: Certainly. That will
18 be Exhibit 624.

19 THE COURT: Is that a new exhibit?

20 MR. OSBURN: It is a new exhibit.
21 It is for cross-examination only.

22 THE WITNESS: My response to that
23 would be, first of all, to agree that there are, you
24 know, ongoing efforts at studying asbestos-related
25 facts. And certainly the issue of chrysotile exposure

1 and peritoneal mesothelioma, I would agree with
2 Dr. Lemen that, you know, specific epidemiologic
3 studies have not been designed to look only at that
4 question.

5 But I would note that the general issue of
6 mesothelioma and chrysotile has been looked at, and a
7 number of studies such as Yano observed with pure
8 chrysotile exposure both pleural mesothelioma and
9 peritoneal mesothelioma.

10 And a number of the studies looking at
11 mesothelioma such as Suzuki in Annals of New York
12 Academy of Science, 2002, had more than a dozen cases
13 of peritoneal mesothelioma where chrysotile was the
14 dominant mesothelial fiber burden. And in 24 percent
15 of cases in that study, the only identifiable fiber in
16 the lung and the pleura was chrysotile.

17 And I think based on that, it would be an
18 erroneous conclusion to say that chrysotile does not
19 cause peritoneal mesothelioma. I think it's a fair
20 statement to say that specific studies designed to
21 look only at that question have yet to be carried out.
22 I would agree with that.

23 Q And you would agree that 0.1 fiber cc years, after a
24 year of working, which is either 265 or 262 days, I
25 can't remember?

1 A Well, I don't know which it is. It may be more or
2 less depending on the worker. But --

3 Q Roughly that over eight hours a day, correct?

4 A Yes. I would represent full-time employment at the
5 permissible exposure limit for a year, .1 fiber cc
6 year.

7 Q That's well over 2,000 hours exposure?

8 A I haven't calculated the hours, but that is the metric
9 for assessing .1 fiber per cc.

10 Q And the rat studies where chrysotile and other
11 products were injected into the peritoneal cavity,
12 that would directly pass the body's defense
13 mechanisms, which are otherwise quite excellent for
14 all fibers, aren't they?

15 A Well, the experimental studies are really in the
16 nature of two types: One in direct injection, which I
17 described in the Davis study, which certainly would
18 bypass defenses, the other are inhalation studies,
19 such as Dr. Coffin did in Inhalation Toxicology in
20 1992. Those are high level of exposures. They are
21 direct inhalation studies strongly associated with
22 chrysotile-induced mesothelioma.

23 MR. OSBURN: Thank you, Doctor.

24 Nothing further.

25 ///

REDIRECT EXAMINATION

BY MR. FROST:

Q Dr. Brodkin, are you familiar with Dr. Lemen's article, Chrysotile Asbestos As a Cause of Mesothelioma?

A Yes, I believe I am.

Q I wonder if you could just read for the court this highlighted section on Page 237?

A I'm just reading from the highlight here. "However, even when potency on a dose by dose basis is considered, the fact remains that chrysotile is capable of causing mesothelioma and that no safe dose has been identified below which a risk of developing mesothelioma no longer exists."

Q Thank you. Now, Mr. Osburn asked you about what kind of exposures would be necessary to create a .1 fiber cc year of cumulative exposure. And I think his description was it would take 2,000 hours of work at a .1 fiber cc environment to create that exposure?

A Well, he mentioned 2,000 hours. Basically, a year in an eight-hour work shift, five days per week, would result in at permissible exposure limits, .1 fiber per cc.

I do want to emphasize that OSHA in publishing that recommends that exposures be as far below that as

1 possible, but that would be at that limit.

2 Q That's not a safe level of exposure, is it?

3 A It is not a safe level of exposure. In fact, it's
4 clearly stated and Stayner notes in his article in
5 Occupational and Environmental Medicine in 1997 that
6 at that level one can anticipate two cases of
7 asbestosis per thousand and even more cases of
8 malignancy.

9 Q So if it took 2,000 hours at .1 fiber cc of exposure,
10 then 200 hours at 1 fiber cc of exposure would create
11 that same .1 fiber cc year?

12 A Yes. The cumulative exposure metric involves two
13 parameters, intensity and duration. So a shorter
14 duration of exposure at higher intensity certainly can
15 result in the same cumulative exposure as a lower
16 intensity exposure over a longer duration.

17 Q By the same token, it would only take 20 hours at 10
18 fiber cc's to develop a .1 fiber cc year total?

19 A Yes. Basically as intensity increases, the duration
20 would increase -- decrease linearly that would be
21 required to produce the same cumulative exposure.

22 Q And the studies that you relied on show that drywall
23 activities, mixing, sanding, sweeping, often create
24 exposures in excess of 10 fiber cc's?

25 A Frequently. Sanding procedures up to 20 fiber per cc,

1 mixing, particularly dry mixing, up to 40 fiber per
2 cc, even pre mix when you sand it and sweep it will
3 have very significant fiber cc levels.

4 MR. FROST: Nothing additional.

5 Thank you.

6 MR. OSBURN: Very quickly, Your
7 Honor.

8
9 RECROSS-EXAMINATION

10 BY MR. OSBURN:

11 Q But you don't have any idea in any manner how Mr. Lott
12 used Bondex --

13 A My only knowledge is from Mr. Lott's deposition, and
14 he described his use of joint compound generically.
15 He noted he was exposed to a number of different joint
16 compounds, but didn't describe his work with a
17 particular joint compound brand in contra distinction
18 to another.

19 Q Did you read his testimony where he testified that he
20 could not recall working specifically with Bondex
21 joint compound?

22 A I certainly read Mr. Lott's testimony. I can't
23 remember every word of it, and I would trust what it
24 said. But my recollection is that he used a number of
25 different joint compounds, and he did not provide any

1 testimony of specific activity with a particular brand
2 of joint compound. I certainly don't recall that.

3 MR. OSBURN: That would be fine.

4 Thank you, Doctor.

5 MR. FROST: Nothing additional.

6 THE COURT: Thank you, Dr. Brodtkin.

7 THE WITNESS: Thank you, Your Honor.

8 MR. FROST: Plaintiffs call
9 Dr. Samuel Hammar.

10
11 SAMUEL P. HAMMAR, MD, FCCP, having been first duly
12 sworn, testified as
13 follows:

14
15 DIRECT EXAMINATION

16 BY MR. FROST:

17 Q Will you please state your full name and your address
18 and zip code for the record?

19 A Yes, Samuel P. Hammar, Diagnostic Specialties
20 Laboratory, 700 Lebo, L-E-B-O, Boulevard, Bremerton,
21 Washington, 98310.

22 MR. FROST: And, Your Honor, we're
23 going to use a Powerpoint with Dr. Hammar. We had the
24 slides marked as Plaintiff's Exhibit No. 364 for use
25 in this.

1 THE COURT: All right.

2 Q (By Mr. Frost) Now, Dr. Hammar, you understand that
3 we're going to use your CV and supplement the record
4 with your qualifications, but is it fair to say you
5 have written extensively and studied extensively the
6 field of asbestos medicine?

7 A Yes.

8 Q Now, you understand the issue in this case is
9 cumulative exposures and how they relate to causation
10 in mesothelioma cases, correct?

11 A Yes.

12 Q Would you explain to the judge what your opinions are
13 and what those -- what the basis of your opinions are
14 concerning cumulative exposures as they relate to
15 causation of mesothelioma?

16 A I think as already partially stated by Dr. Brodtkin is
17 that asbestos related diseases are all dose response
18 related, which means that the more that one is exposed
19 to asbestos, the greater the risk is of an individual
20 developing one of the asbestos-related diseases.

21 And if you look at it from an incidence point of
22 view, the greater the exposure, the greater the
23 incidence is of the disease. And it's thought that
24 all bystander and occupational exposures to asbestos
25 have the ability to contribute to cause the injury

1 that eventually leads to one of these diseases.

2 Q And, Dr. Hammar, have you had an opportunity to review
3 the deposition of Mr. Lott in this case?

4 A I have, yes.

5 Q And I'm trying to not go over the same areas that we
6 went over with the previous doctor, but in regards to
7 Mr. Lott's explanation of his exposures, would those
8 have been causally related to his mesothelioma?

9 A Yes.

10 Q Now, I want to talk to you a little bit about whether
11 there is a safe level of exposure to asbestos and what
12 the literature says in regards to that.

13 And so the first thing I want to direct your
14 attention to is the statement by Dr. Selikoff that
15 there is no safe level of asbestos. Is that a true
16 and accurate statement? Is that a generally held view
17 in the medical community?

18 A I think it's definitely an opinion that is held in the
19 medical community, specifically with respect to
20 occupational bystander exposure to asbestos.

21 Q Now, in regards to whether there is a threshold at a
22 minimal lower limit, could you explain how, and I have
23 got the quote from the Hillerdal paper where it talks
24 about there's no proof of a threshold value that is a
25 minimal lower limit below which asbestos fibers cannot

1 cause the tumor. And that's related to the
2 mesothelioma.

3 Could you explain what that means?

4 A Sure. That paper by Dr. Hillerdal had to do with a
5 report of several examples of mesothelioma occurring
6 in people that had nonoccupational exposure to
7 asbestos and lower level dose exposure.

8 And Dr. Hillerdal's opinion was really there isn't
9 any specific background incidence of mesothelioma in
10 the United States or in the world with respect to
11 asbestos causation of mesothelioma. The number that
12 is usually cited is one case per million people per
13 year, which is an incredibly low level of exposure.

14 What this is is just evidence that you can see
15 cases of mesothelioma in people who have incredibly
16 low levels of exposure. And based on that, there
17 really isn't a threshold that you can state that if a
18 person was exposed to a concentration of asbestos
19 below this amount, then they would not get
20 mesothelioma.

21 Q Now, I want to move on to NIOSH. NIOSH has discussed
22 this, whether there's a safe level or not, and NIOSH
23 has indicated in 1980 that excessive cancer risks have
24 been demonstrated in all fiber concentrations and
25 studied to date. The evaluation of all available

1 human data provides no evidence for a threshold or for
2 a safe level of asbestos exposure.

3 Is that a similar finding as the Hillerdal?

4 A That is, yes.

5 Q And as we sit here today, is that still NIOSH's
6 opinion as far as you know?

7 A Yes.

8 Q Now, Dr. Roggli, who has been brought up before, has
9 written a medical textbook on pathology and
10 asbestos-related diseases, correct?

11 A He has, yes.

12 Q And is that textbook, is that generally accepted as --
13 although there may be things in it that folks disagree
14 with, is that generally accepted in the scientific
15 community as a peer-reviewed and scientific book that
16 is relied upon by doctors?

17 A It's a learned treatise, sure. Dr. Roggli is an
18 absolute expert in asbestos-related diseases. That
19 book now is in the second edition.

20 Q Now, in the 1992 edition, Dr. Roggli talked about this
21 linear dose response relationship. And he indicated
22 that there is a linear dose response relationship
23 between the amount of asbestos to which an individual
24 is exposed and the risk of developing mesothelioma.
25 In addition, the threshold level of exposure below

1 which mesothelioma will not occur has not yet been
2 identified.

3 Is that still a correct statement as we sit here
4 today?

5 A I believe it is, yes.

6 Q And is that generally accepted in the scientific
7 community as a statement in regards to the threshold
8 level of exposure in regards to mesothelioma?

9 A Yes.

10 Q Now, besides Dr. Roggli, the Consumer Products Safety
11 Commission has also looked at the scientific
12 literature in regards to whether there's a threshold
13 below which asbestos diseases occur.

14 Are you familiar with the Consumer Products Safety
15 Commission?

16 A Sure.

17 Q And you are familiar with their ban on patching
18 compounds, including joint compounds?

19 A Yes.

20 Q The consumer product -- and is that something that is
21 something you generally looked at and relied upon in
22 the past and are aware of in the scientific
23 literature?

24 A Yes.

25 Q Now, Dr. Hammar, the Consumer Products Safety

1 Commission indicated that the commission noted that in
2 the scientific literature, there is general agreement
3 that there is no known threshold level below which
4 exposure to respirable free-form asbestos would be
5 considered safe. Is that still true today and
6 scientifically valid?

7 A Yes.

8 Q And I believe we already talked about NIOSH and their
9 statement.

10 Now, do you know who Dr. Battifora is?

11 A Sure.

12 Q Who is Dr. Battifora?

13 A Dr. Battifora is a well-known pulmonary pathologist.
14 He was the author of the third series of fascicles on
15 tumors of the serosal membranes, which are basically
16 mesotheliomas and other tumors that look like
17 mesotheliomas. And he and Dr. McCaughey, and
18 Dr. McCaughey was a pathologist in Canada who wrote
19 that fascicle.

20 Q And the fascicle is a pamphlet put out by the Armed
21 Forces Institute of Pathology?

22 A It is. It's about 100 pages long about, I would say
23 close to a half-inch thick. There's one -- a newer
24 one after this one that just was published recently,
25 but this was the third edition or the third series

1 fascicle.

2 Q And in regards to the fascicle, was that generally
3 relied upon by scientists and medical doctors?

4 A I think so, yes.

5 Q And was that a statement of the general consensus in
6 the scientific community?

7 A Yes.

8 Q And in the third series of the fascicle of the Armed
9 Forces Institute of Pathology, Dr. Battifora
10 indicated, "The incidence of diffuse malignant
11 mesothelioma rises with increasing intensity and
12 duration of exposure to asbestos. The dose specific
13 risk data is a linear relationship."

14 Do you agree with that?

15 A I do, yes.

16 Q Could you explain that a little bit to the court?

17 A Sure. What it means is basically is that if you were
18 to plot the incidence of mesothelioma on, say, the X
19 axis and the concentration of asbestos a person was
20 exposed to on the Y axis, you would get this line that
21 would go like that. (Indicating.)

22 Eventually it does level out. It doesn't increase
23 forever, but it's linear for much of the data that has
24 been studied.

25 Q And is that linear relationship, is that something

1 that is generally agreed upon in the scientific
2 literature?

3 A That's what almost all of the articles use when they
4 do any type of studies in trying to assess low levels
5 of exposure or even high levels of exposure. So the
6 answer is yes.

7 Q And why is it important in dealing with low levels of
8 exposure, that linear relationship?

9 A Well, it's important because if you don't know what
10 the low level incidence is of a disease like
11 mesothelioma, you wouldn't know exactly how to protect
12 people from or what concentration of asbestos should
13 people be allowed to be exposed to.

14 Q Now, the American Journal of Industrial Medicine, is
15 that a peer-reviewed publication?

16 A It is, yes.

17 Q And in the year 2001, Rodelsperger published an
18 article called Asbestos in Manmade Vitreous Fibers As
19 Risk Factors For Diffuse Malignant Mesothelioma,
20 Results From a German Hospital-Based Case Control
21 Study.

22 And in that article, they came to the conclusion
23 that our results confirm the previously recorded
24 observation of the distinct dose response relationship
25 even at levels of cumulative exposure below one fiber

1 year.

2 Is that something that you agree with?

3 A I agree with that, yes. I have that article with me
4 today.

5 Q And could you explain to the judge how that relates to
6 your basis of your opinions in this case as to the
7 causation of the mesothelioma in Mr. Lott?

8 A Well, it's additional evidence that mesothelioma can
9 occur in people that are exposed to low concentrations
10 of asbestos.

11 Q Now, the same -- the same article also indicates that
12 they did a further case control analysis based on lung
13 tissue fiber concentrations in addition to the
14 interviews, and they yield similar OR's. Are you
15 familiar with the, in that particular study, the use
16 of lung tissue fiber concentrations in order to
17 supplement the interviews of the subjects?

18 A That's described in that article, yes.

19 Q And why is that important?

20 A Well, what that is looking at is actually the
21 concentration of asbestos you have in the lung tissue
22 which would directly relate to a person's exposure. I
23 guess the only thing that might be better than that
24 would be what the concentration was of asbestos,
25 either the pleura if it was a pleural mesothelioma, or

1 in the peritoneum, if it was a peritoneal
2 mesothelioma.

3 Q Now, in regards to a mesothelioma, in order to
4 attribute a mesothelioma to asbestos, do you need
5 pleural plaques?

6 A No.

7 Q Do you need findings of fibrosis or asbestosis?

8 A No.

9 Q Now, in regards to your opinions as to whether you
10 need pleural plaques, asbestosis or other fibrosis in
11 order to attribute a mesothelioma to asbestos
12 exposure, are those generally accepted in the
13 scientific community?

14 A Absolutely. They are published by the Helsinki
15 Consensus Report Journal, Scandinavian Work and
16 Environmental Health in 1997.

17 Q And those have been throughout the literature, those
18 same type of statements in regards to pleural plaques
19 and mesothelioma and asbestosis?

20 A Yes.

21 Q Is there really any disagreement in regards to that?

22 A I don't think there's any disagreement, no. If you
23 look at plaques, it's kind of an interesting thing
24 about how many you see radiographically versus autopsy
25 versus standard chest radiographs. There's quite a

1 bit of difference based on what technique you use with
2 respect to how many you see.

3 And that's also true for asbestosis in that when
4 Dr. Dodson and I published our paper in 1997 on fiber
5 types of 55 cases of mesothelioma, it turned out
6 clinically there were only three cases of asbestosis.
7 But when we looked pathologically, it turned out there
8 were 29 cases of asbestosis, most of which were
9 CAP-NIOSH Grade 1 asbestosis, which is the least
10 severe form of asbestosis.

11 Q Now, even based on those findings, did you still come
12 to the conclusion that you do not need asbestosis or
13 finding of pleural plaques in order to attribute a
14 mesothelioma to asbestos exposure?

15 A That is correct because, again, clinically, I think as
16 Dr. Brodtkin stated, in that most people with
17 mesothelioma, you don't see asbestosis.

18 Q Now, the next article we have is the -- I know I'm
19 going to butcher the name -- Iwatsubo. It's called
20 Pleural Mesothelioma: Dose-Response Relation at Low
21 Levels of Asbestos Exposure in a French
22 Population-based Case-Control Study. Are you familiar
23 with that article?

24 A I am. I have that one with me also.

25 Q And is that a peer-reviewed publication?

1 A It is, yes.

2 Q And is that something that is generally accepted and
3 used by folks in the medical community?

4 A Yes.

5 Q Now, in Iwatsubo, it talks about a significant excess
6 of mesothelioma was observed for levels of cumulative
7 exposure that were probably far below the limits
8 adopted in most industrial countries during the
9 1980's. What are they talking about there?

10 A They are talking about the fact that mesothelioma can
11 occur at very low levels of concentration of exposure,
12 and that they occur at concentrations greater than
13 what the permissible exposure levels that various
14 countries have initiated for asbestos to try to
15 control the diseases that asbestos cause.

16 Q And is that something you've also seen in your
17 practice?

18 A Sure. It's sometimes hard to evaluate exactly how
19 much a person was exposed based on some of the
20 information you get. But I don't think there's any
21 doubt that there are cases of low level exposure to
22 asbestos that cause mesothelioma.

23 Q And is that based on review of the scientific
24 literature and is there statements in the scientific
25 literature regarding low dose exposures causing

1 mesothelioma?

2 A Sure.

3 Q And then again in Iwatsubo it talks about the dose
4 response relationship with cumulative exposures for
5 both intermittent and continuous patterns of exposure.
6 Why is that important?

7 A Again, the more you are exposed to, the more likely
8 you are to get a disease.

9 Q Now, I want to talk a little bit about short or
10 indirect exposures, and we've just hit on that very
11 briefly.

12 A Uh-huh.

13 Q In the Greenberg and Davies study, which is the
14 mesothelioma register from '67 to '68 published in
15 1974, it talks about the duration of exposure was more
16 widely spread ranging from three weeks to over 50
17 years. 12 percent of the cases had been exposed for
18 under five years. A man with only three weeks of
19 exposure died over a half century later.

20 Why is that important when you are considering the
21 issue of causation of mesothelioma in cumulative
22 exposure

23 A Well, I guess what is really important is that you are
24 trying to protect everybody that might ever be exposed
25 to asbestos. And if you know there are examples of

1 people that develop mesothelioma at very low levels of
2 exposure, you would have to try to protect those
3 people also.

4 And the only way you could do that is to make sure
5 that they were either never exposed to asbestos or
6 exposed to an exceedingly small concentration.

7 Q And then I have the next slide, which is also from the
8 Greenberg and Davies, and it talks about hobby
9 exposure where it indicated that there were folks who
10 had a history of exposure to asbestos with their hobby
11 work, including a case of nonoccupational asbestos
12 exposure in a mesothelioma victim exposed for one day
13 sawing asbestos cement sheets to construct two sheds.
14 Are you aware of that study?

15 A I am, yes.

16 Q And how does that relate, and when you talk about the
17 dose response relationship and mesothelioma?

18 A Well, again, it's an example of cases of mesothelioma
19 developing in people that had what appeared to be
20 relatively low exposures. It could turn out that in
21 that case the exposure actually might be higher than
22 you think based on the fact that he was sawing cement
23 board.

24 But, again, it's an example of a short time of
25 exposure resulting in the development of mesothelioma.

1 Q And I'm not sure if previously we talked about peek
2 exposures, but what are peek exposures?

3 A Most people who are exposed to asbestos don't --
4 aren't exposed to a concentration of asbestos that
5 stays the same over their entire working day or entire
6 working life. And you can have people that, say, at
7 one point in time of their working day might be
8 exposed to very low levels and no asbestos, but at
9 other times be exposed to incredibly high
10 concentrations of asbestos.

11 And when you calculate the fiber cc years of
12 exposure, you have to take that information into
13 account.

14 Q Now, I put up the article from Anderson, et al, which
15 is Mesothelioma Among Employees With Likely Contact
16 With In-Place Asbestos-Containing Building Materials.
17 These are basically the teacher studies. Are you
18 aware of those?

19 A I am, yes.

20 Q And it indicates that 75 percent of the school
21 teachers, the only identifiable potential source of
22 asbestos exposure was derived from in-place asbestos-
23 containing materials in schools. One teacher had
24 spent the season in the merchant marine aboard an iron
25 ore hauling ship and two had worked in the residential

1 construction industry.

2 How does that indicate that, again, the low levels
3 of exposure that can be causally related to
4 mesothelioma?

5 A Again, it's just an example of how things happen. I
6 mean, in the Third Wave Book, which was a symposium of
7 information published from a conference held in Ottawa
8 in 1998, there's some more examples of teachers
9 developing mesothelioma. And it's accepted an type of
10 person who can get it, presumably from asbestos in
11 place that became friable where they were exposed to
12 it that way or maybe from some other way in the school
13 where they say they were sweeping up asbestos from
14 asbestos-containing thermal insulation on pipes, for
15 example.

16 Q Now, in the Iwatsubo article, it indicated that
17 background levels are assumed to be as low as one to
18 two per million in habitus, and we are talking about
19 the rate of mesothelioma.

20 Do you agree with that?

21 A Well, that's what the number that is usually given,
22 it's usually one or between one to two cases per
23 million people per year.

24 But Dr. Hillerdal in his article published in
25 1999, he didn't think there was any actual background

1 level of mesothelioma in the population who had not
2 been exposed to asbestos.

3 Q Now, the Iwatsubo article also talks about based on
4 their review of the mesothelioma cases, about
5 cumulative exposures, and it indicates their results
6 indicated that mesothelioma cases occurred below a
7 cumulative exposure of five fibers per milliliter
8 years and perhaps below .5 fibers per milliliter
9 years.

10 Could you explain how those relate to your
11 opinions in this case?

12 A Again, those would be examples of low exposures.
13 Again, the fiber cc years, the way to calculate that
14 is you try to determine the average concentration of
15 asbestos a person was exposed to during their
16 eight-hour day work, and you would try to determine
17 that for the 252 days a year that they worked, and
18 then you would determine what that concentration was
19 over that time period.

20 So, for example, five fiber ML years could
21 potentially be exposed to five fibers per cc of air
22 eight hours a day, 252 days a week -- I mean 252 days
23 a year for one year or it could be something like
24 exposed to, say, one fiber per cc of air eight hours a
25 day for a week and then exposed to maybe 20 the next

1 week and on and on, you could get various combinations
2 that could result. That is a relatively low
3 concentration. And the 0.5 fiber cc's is 10 times
4 less than that. So that's even a much lower
5 concentration of asbestos that resulted in
6 mesothelioma.

7 Q And how does that relate when we're talking about the
8 linear and dose response?

9 A Well, it just shows that asbestos-induced mesothelioma
10 occurs at a very low level of concentration.

11 Q And is it generally accepted in the scientific
12 community?

13 A Sure.

14 Q Now, the US Department of Health and Human Services
15 has also indicated in regards to -- you are familiar
16 with the US Department of Health and Human Services
17 statements on asbestos?

18 A I have seen those before, yes.

19 Q And they have indicated that on their report to
20 commerce on workers' home contamination study, that
21 mesothelioma has occurred following short-term
22 asbestos fibers of only a few weeks and can result
23 from very low levels of exposure. Is that
24 scientifically based?

25 A It is, yes.

1 Q And is that based upon all those articles and things
2 that we talked about so far?

3 A Yes.

4 Q And, in fact, there have been cases of individuals who
5 work at the home and not directly with asbestos where
6 they also get mesothelioma, correct?

7 A Unfortunately, yes.

8 Q And that's what the next slide is in regards to
9 household exposure. And you agree that that's
10 generally accepted in the scientific community that
11 individuals who aren't directly occupationally exposed
12 but could be exposed through secondary sources, that
13 those folks also could causally get mesothelioma?

14 A Right. They could have either what is called
15 household exposure or domestic bystander exposure or
16 what is called neighborhood exposure.

17 And in the abstract that Dr. Roggli and I
18 submitted in 1997 on 103 cases of mesothelioma in
19 women, the most frequent way the women were exposed
20 was from that bystander household or neighborhood
21 exposure.

22 Q And the last area I want to go over with you,
23 Dr. Hammar, is this area concerning each exposure
24 shortens the latency period for mesothelioma.

25 And could you explain to the court what we mean by

1 each exposure shortens the latency period for
2 mesothelioma?

3 A Well, the latency period by definition with respect to
4 an asbestos-related disease is the time when a person
5 was first exposed to asbestos to the time they were
6 either diagnosed with the disease or the time that
7 they developed symptoms for the disease.

8 And for example in mesothelioma, the shortest
9 latency that is recognized by Helsinki is 10 years.
10 The longest is over 60 years. The shortest that I
11 have seen in my own practice as a pathologist, I saw
12 one case of a person who had a 10-year latency. But
13 most of the people have had latencies between,
14 basically, about 20 to 60 years.

15 But what it is shown is that if you look at the
16 mechanism by which mesothelioma or any other type of
17 cancer induced by a carcinogen is based on, it's a
18 mechanism by which there is continued injury over a
19 period of time in which cells are changed, and
20 eventually these cells will exhibit changes in a lot
21 of the genes that -- or many of the genes that control
22 cell growth, many of the genes that control cell
23 death, the genes that control DNA repair and many of
24 the cell cycle functions.

25 And it's thought that the more of these changes

1 that you have, the more likely one of the cells that
2 has been injured by asbestos or another carcinogen,
3 for example, would become malignant.

4 So the higher the dose is of exposure, you would
5 think that these cellular changes would occur more
6 rapidly and would shorten the latency period for
7 mesothelioma. And that's generally been accepted,
8 although, I would say this in my own experience,
9 there's always going to be exceptions to that rule.

10 Q And is that one of the reasons then in regards to
11 asbestos exposures each and every exposure is causally
12 related to a mesothelioma?

13 A Well, I think that's it. The idea, basically, is that
14 each and every exposure a person has in an
15 occupational or a bystander setting has the ability to
16 contribute to cause that injury. And the way that
17 happens with mesothelioma, for example, is that you
18 inhale air that contains asbestos fibers into your
19 lungs, and a certain concentration of that asbestos is
20 translocated from the lung where it was first
21 deposited to other parts of the body.

22 And with respect to mesothelioma, that would be
23 the pleura that lines the chest cavity in the lung.
24 And in the case of the peritoneal mesothelioma, it
25 would be the peritoneum that covers the organs and

1 lines the abdominal cavity.

2 And the basic idea is that the more asbestos you
3 get to that part of the body, the more likely you are
4 to have this cellular injury that in some individuals
5 will result in the development of a mesothelioma.

6 Q And is this idea of translocation of the asbestos
7 fibers to both pleura and the peritoneum, is that
8 generally accepted in the scientific community?

9 A Well, it's more than generally accepted. It's proven
10 I think at this point in time. In the year 2000,
11 Dr. Dodson and I wrote an article in Chest titled
12 Asbestos in Extrapulmonary Sites, Omentum and
13 Mesentery, which we showed that all types of asbestos
14 that you found in the lung could be found in the
15 abdominal fat tissue and mesentery fat tissue where
16 peritoneal mesotheliomas develop.

17 In 2001, Dr. Suzuki and Dr. Yuen published an
18 article showing that asbestos was translocated to the
19 pleura and that the dominant type of asbestos you
20 found in the pleura was actually short fiber
21 chrysotile, but you did find amphiboles as well.

22 And then there was another article that is not
23 recently -- is not thought of very frequently, but one
24 I think is very important, and that was an article by
25 Hiller, et al, published in 1996, where they were

1 actually looking at ovarian tissue in women and showed
2 that they could find a significant concentration of
3 asbestos, chrysotile asbestos, some amphibole
4 asbestos, in this ovarian tissue, some of the women
5 being -- having bystander exposure to husbands,
6 fathers, who were exposed to asbestos and in some
7 women having no known bystander exposure to asbestos.

8 So there's another example of asbestos being
9 translocated to a target organ where, in this case,
10 peritoneal mesotheliomas develop.

11 Q And is that why in dealing with causation, you have to
12 look at each of the exposures to asbestos to try to
13 determine whether it's causally related?

14 A Yeah. And I think what you know is that in all of the
15 exposures I think a person has to asbestos in an
16 occupational or bystander setting, there is a certain
17 concentration of that asbestos that's going to get
18 into the lung tissue.

19 There's no doubt that a significant amount of it
20 is cleared prior to getting into the lung tissue by
21 the defense mechanisms, which would include a lot of
22 different things, but a certain amount of it does or
23 is deposited in the lung.

24 And once deposited in the lung, then that asbestos
25 has the ability to be translocated to other parts of

1 the body like the pleura and the peritoneum where it
2 can cause mesothelioma.

3 Q And very briefly, Doctor, I don't think we talked
4 about the Bianchi article, Latency Periods in
5 Asbestos-Related Mesothelioma of the Pleura, European
6 Journal of Cancer Prevention from 1997. It indicated
7 that, and we're talking about each exposure shortens
8 the latency period for mesothelioma, that the data for
9 the most heavily exposed people, as well as those as
10 the groups with the lowest exposure, are exactly the
11 same as would be expected. An inverse correlation
12 exists between an intensity of exposure to asbestos
13 and the duration of the latency period.

14 And that's again talking about how the -- as the
15 exposure lessens, the latency period gets longer?

16 A Right, low levels are associated with long latencies,
17 high levels of exposure are associated with short
18 latencies.

19 Q I also think Dr. Churg has indicated in his book that
20 as exposure level decreases, the latency period
21 increases; is that correct?

22 A That is correct, yes.

23 Q Is there any disagreement in the scientific literature
24 on that issue?

25 A I don't think there's any necessarily disagreement. I

1 think, again, with what you can see those is
2 variation, and I think that's what you have to be open
3 to the idea that that's not necessarily going to
4 happen in every case.

5 But I think that what is stated right there is
6 generally what is accepted in the scientific
7 literature.

8 Q Now, Doctor, just so the record is clear, all the
9 opinions that you have given today are within a
10 reasonable degree of medical probability?

11 A Yes.

12 Q And in regards to all the articles that I have shown
13 you on the Powerpoint, these are all articles that you
14 have reviewed and are generally accepted in the
15 scientific community or in the peer-reviewed
16 literature?

17 A Right, articles that I have reviewed or statements
18 that are present in, like, government documents that I
19 have reviewed.

20 Q And are all these the types of materials that you have
21 relied upon in coming to your conclusions in this
22 case?

23 A Yes.

24 MR. FROST: That's all we have, your
25 honor.

1 THE COURT: Mr. Osburn, any
2 questions?

3 MR. OSBURN: I will try and be
4 brief, Your Honor.

5
6 CROSS-EXAMINATION

7 BY MR. OSBURN:

8 Q Dr. Hammar, are you aware that Judge Colville in
9 Allegheny county reviewed a couple of your transcripts
10 in his decision on a Frye Hearing on the same reason
11 we're here this morning?

12 A No.

13 Q The transcript from the Eisen -- I believe you
14 testified in the Eisenreich (sic.) case in 1999?

15 A Yes.

16 Q And in the Kulig case on June 5th, 2003?

17 A I remember those names. I don't think I could have
18 told you the dates.

19 Q Doctor, you've testified many times that exposure
20 below 0.1 fiber cc years is not sufficient to cause a
21 mesothelioma; is that true?

22 A I have stated that statistically there's no evidence
23 until just recently that I thought showed that. That
24 was based on Dr. Nicholson's determination published
25 in the 1986 Federal Register which stated that at .1

1 fiber cc years of exposures, there was seven cases of
2 mesothelioma per 100,000 people. There was no
3 statement of anything lower than that. And that was
4 the number given at that point in time.

5 Q And you've testified that on Nicholson's model, which
6 is 0.1 fiber per cc, which is eight hours a day for a
7 working year, which is 252 days, and that's the
8 exposure he's talking about?

9 A Yes.

10 Q And you testified in the McPhee trial that you were
11 never going to be able to collect a group of people
12 with that low of exposure and, say, compare them with
13 people that had no exposure. It's just not possible
14 to do. So what they do is extrapolate from higher
15 numbers to lower numbers, and higher numbers means the
16 higher dose to a lower dose, does it not?

17 A That's basically what they do. That's correct.

18 Q You have testified, I believe, in the Whitehead case
19 that you cannot quantify a threshold for peritoneal
20 mesothelioma, correct?

21 A I would say that even today. I don't know if anybody
22 knows what the threshold for peritoneal mesothelioma
23 is. I know when Dr. Roggli and I, again, wrote our
24 abstract in 1997, what we found was that there was
25 really a significant variation in the concentration of

1 individuals' lung tissue for asbestos in people who
2 had peritoneal mesothelioma.

3 And I strongly feel that today there's always
4 going to be a range of what you find in concentration
5 of asbestos in lung tissue, pleural tissue, peritoneal
6 tissue where people have mesotheliomas. It's never
7 going to be an absolute number

8 Q You testified that you can't use case reports alone to
9 establish causation (Inaudible) or further study
10 rather to assign (Inaudible), right?

11 A I have stated that. But, you know, it's kind of
12 interesting that there was just a recent article about
13 that where it talked about two incidents in which case
14 reports were used for causation. And the one is
15 really pertinent to mesothelioma, and that was the
16 report by Dr. Wagner, Sleggs and Marchand in the
17 British Journal of Industrial Medicine in 1960 where
18 they reported 33 cases of mesothelioma in the
19 Northwestern Cape Province of South Africa. And it
20 was that article that basically led the world to
21 conclude that mesothelioma was caused by asbestos.

22 The other case report that did a similar thing was
23 five cases of angiosarcoma that occurred in workers
24 who were exposed to vinyl chloride. And it's thought
25 that that case report, again, drew the world's

1 attention to the idea that vinyl chloride was a cause
2 of angiosarcomas in liver.

3 Q Right. And they draw the world's attention to things
4 by saying here's something we really can't -- we're
5 not sure about, we can't draw conclusions from, so
6 we're going to go ahead and study them, and we are
7 going to go ahead and do a biological study and then
8 we're going to find out?

9 A Well --

10 Q If you just have an isolated case, you can't draw a
11 conclusion from that. That was your testimony in the
12 Whitehead case on December 8th, 2005.

13 A Well, that might well have been. But I would say at
14 the same time is that if you were to ask anybody in
15 mesothelioma medicine, I will put it that way, when
16 they thought that mesothelioma was caused by asbestos,
17 I would suspect the majority would say when the
18 article by Wagner, Sleggs and Marchand was published.

19 If you actually look at the real date, it probably
20 was Merewether's studies from 1940 where he used the
21 word tumors of the pleura, which were probably
22 mesotheliomas. And he did do an epidemiologic study,
23 but unfortunately he didn't use -- did not use the
24 word mesothelioma.

25 Q You mentioned the Iwatsubo study from 1998, correct?

1 A Pardon me?

2 Q You mentioned the Iwatsubo study --

3 A Yes.

4 Q -- on the French population. The Iwatsubo study,
5 right, does not (Inaudible) "Furthermore, since only a
6 few subjects in these cohorts were exposed to low
7 levels of asbestos, there is not enough statistical
8 power to show any significant association with
9 mesothelioma"?

10 A That is a correct statement in that article, yes.

11 Q And you can't quantify Mr. Lott's exposure to
12 asbestos?

13 A I can't, no.

14 Q Let's talk about the Helsinki criteria, one of which
15 refers to diagnosis, one of which is actually
16 attribution of causation, one of which is biological
17 evidence which is not available in Mr. Lott's case?

18 A That is correct.

19 Q The second which is radiological evidence, which is
20 also not available in Mr. Lott's case?

21 A That is correct.

22 Q The third is occupational history, but Helsinki says
23 you need a significant occupational exposure, doesn't
24 it, Dr. Hammar?

25 A No, it doesn't say that. What the Helsinki Consensus

1 Report says, it's on Page 313, and the exact title of
2 the article is Consensus report: Asbestos, Asbestosis
3 and Cancer: The Helsinki Criteria For Diagnosis and
4 Attribution, Scandinavian Journal of Work and
5 Environmental Health, 1997, Volume 23, Pages 311-316.

6 And on Page 313, it says -- it starts out by
7 saying -- talking about lung fiber content, and then
8 it says, "Should be sufficient to relate a case of
9 pleural mesothelioma to asbestos exposure on a
10 probability basis. In the absence of such markers,"
11 and it's talking about plaques and asbestosis, "a
12 history of significant occupational, domestic or
13 environmental exposure to asbestos will suffice for
14 attribution."

15 And then it states that -- in the bullet points
16 it states that, the second once, mesothelioma can
17 occur in cases with low asbestos exposure. However,
18 very low background environmental exposures carry only
19 an extremely low risk.

20 And then it states that an occupational history of
21 brief or low-level exposure should be considered
22 sufficient for mesothelioma to be designated as
23 occupationally related.

24 So it seems to me that that's saying that if you
25 don't have the markers like fiber concentrations,

1 asbestos body concentrations, plaques or asbestosis,
2 you can attribute causation by a history of exposure,
3 and even a very low level of exposure will suffice.

4 Q In the -- you were shown a couple of slides. One was
5 from Rodelsperger?

6 A Yes, mm-hm.

7 Q And it talks about levels of cumulative exposure below
8 one fiber year, and that's one fiber cc year, correct?

9 A Yes, mm-hm.

10 Q Doctor, what -- in the Anderson case, the judge
11 excluded your testimony on causation or at least
12 limited it?

13 A No.

14 Q He didn't?

15 A Pardon me?

16 Q He didn't limit your testimony on causation?

17 A On Anderson? Oh, yes, yeah.

18 Q And how did he limit it?

19 A Pardon me?

20 Q How did he limit it?

21 A He said that we could not talk about anything -- a
22 concentration of asbestos I think it was below .1
23 fiber cc years. And there was another limitation that
24 I can't remember. But it was -- it was limiting. Oh,
25 I know. The other one is that we couldn't talk about

1 all of the exposures that we thought that Mr. Anderson
2 had to asbestos-containing materials from the
3 Caterpillar engine.

4 Q As being causative of mesothelioma?

5 A Yes, mm-hm.

6 Q And didn't he prevent you from giving a range of
7 exposures being causative of mesothelioma without
8 giving a lower threshold, correct?

9 A He did. I will never understand it.

10 MR. OSBURN: Thank you, Doctor.

11 Nothing further.

12 THE WITNESS: Okay.

13
14 REDIRECT EXAMINATION

15 BY MR. FROST:

16 Q Dr. Hammar, is there any scientific basis for that
17 limitation in your testimony?

18 A Well, there certainly wasn't to me. And I will tell
19 you, as a pathologist, I didn't understand it at all,
20 and I still don't, and I never will. And it seemed to
21 me very unfair.

22 Q And, Dr. Hammar, in regards to your testimony, you
23 have testified throughout the country, correct?

24 A I have, yes.

25 Q Has your testimony ever been limited before?

1 A No.

2 Q Now, Dr. Hammar, you also testified in an in re
3 asbestos litigation, what I'm going to call a very
4 large friction motion, that dealt with all these same
5 issues in the state of Delaware, correct?

6 A I did.

7 Q And in that, there's also an opinion in that case that
8 has Dr. Samuel Hammar, and it discusses your
9 testimony, and it discusses other witnesses including
10 Dr. Dodson, correct?

11 A Yes.

12 Q And in that case, in that friction case, which
13 involved the same issues, that judge in Delaware
14 overruled the motion, correct?

15 A That is correct.

16 Q And also in Texas, we have an MDL procedure for the
17 entire state. You are aware of that, that there's one
18 judge that decides all pretrial matters?

19 A Dr. Davidson -- Judge Davidson. Excuse me.

20 Q He might think he's a doctor.

21 MR. FROST: Can that be stricken
22 from the record, Judge?

23 THE COURT: Nothing gets stricken
24 from our record.

25 Q (By Mr. Frost) And Judge Davidson has dealt with

1 these exact same issues. Dr. Hammar, you testified
2 for a number of days, and I think many experts
3 testified in front of Judge Davidson on this exact
4 same issue, correct?

5 A That is correct, yes.

6 Q And in that particular case, in the Texas MDL that
7 involves every asbestos case filed in the state of
8 Texas, you were not excluded, nor was your testimony
9 limited?

10 A That is correct.

11 MR. FROST: That's all we have.

12 THE COURT: Dr. Hammar, I have a
13 question.

14 THE WITNESS: Okay.

15 THE COURT: You were talking about
16 the Nicholson study, and you agreed that you had
17 previously testified that exposure to less than .1
18 fiber cc year isn't sufficient to cause mesothelioma,
19 and then there was an implication that, however, maybe
20 more recently your opinion has changed.

21 Did I just read that in or is that correct?

22 THE WITNESS: That's absolutely
23 correct, Judge.

24 THE COURT: Okay. Could you explain
25 that?

1 THE WITNESS: Well, there have been
2 more articles published and one that I had overlooked
3 that talked about lower levels of asbestos.

4 For example, the Hodgson Darnton article that was
5 published in the American Journal of Occupational
6 Hygiene in 2000 in Table 11, they show concentrations
7 of asbestos.

8 MR. FROST: And, Your Honor, that
9 has previously been marked as Plaintiff's Exhibit 334.

10 THE WITNESS: In Table 11 of that
11 article, they show concentrations of asbestos -- I'm
12 trying to get the exact article -- I mean the exact
13 table. (Peruses document.)

14 But, basically, they show levels as low as .001
15 fiber cc's of asbestos, and this is amphibole
16 asbestos, as causing mesothelioma, extremely low
17 concentrations.

18 And then in the Iwatsubo article, I think their
19 concentration went down to 0.01 fiber cc years, which,
20 again, is lower than Dr. Nicholson's level of .1 fiber
21 cc years.

22 THE COURT: And that was chrysotile?

23 THE WITNESS: In -- in the case of
24 chrysotile in the Hodgson and Darnton article -- and
25 maybe -- let me see if I can find that, and I can read

1 it to you exactly. (Peruses documents.)

2 They stated for one fiber cc years that there
3 were, for crocidolite, they stated estimate 650 deaths
4 per 100,000 exposed, and this is for mesothelioma,
5 highest arguable estimate 1,500, lowest 250. For
6 amosite, it was 90 deaths per 100,000, highest
7 arguable estimate 300, lowest 15 for chrysotile, best
8 estimate five deaths per 100,000 exposed, highest
9 arguable, 20, lowest one.

10 And then they get down to .1 fiber cc years for
11 crocidolite. It was 100 per 100,000 exposed. For
12 amosite, it was 15. And then they said for chrysotile
13 risk, probably insignificant, highest arguable
14 estimate, four deaths per 100,000 exposed.

15 But even at that level, though, if you look at the
16 real background of one case per million people, and if
17 you have four deaths per 100,000 people, that's 40
18 times what you would expect to find in the background,
19 which is an incredible high risk.

20 THE COURT: And the Iwatsubo study,
21 was that chrysotile as well --

22 THE WITNESS: Let me see if I can --

23 THE COURT: -- that .01 fiber?

24 THE WITNESS: Let's see. (Peruses
25 documents.) I don't have that with me. Does anybody

1 have the Iwatsubo article?

2 MR. FROST: I do, Your Honor. Where
3 it is is a different story.

4 THE WITNESS: I thought that I had
5 that with me, but I guess I didn't.

6 MR. OSBURN: I have a copy here. It
7 was one of the submissions by Dr. Maddox in Judge
8 Colville's case.

9 THE COURT: Would you mind handing
10 the article to Dr. Hammar just for a minute?

11 MR. OSBURN: Here you are.

12 THE WITNESS: Thank you.
13 Unfortunately, this doesn't -- this is not the whole
14 article.

15 THE COURT: Oh, it's just the
16 abstract.

17 THE WITNESS: It had the table.

18 MR. OSBURN: It's just the abstract.

19 THE WITNESS: It had the table
20 there. I'm sorry.

21 THE COURT: Okay. But in any event,
22 what was the concentration again in the Iwatsubo?

23 THE WITNESS: It got down to 0 --
24 0.01 fiber cc years.

25 THE COURT: Okay.

1 THE WITNESS: Then at the
2 International Mesothelioma Interest Group which I
3 participated in about two weeks ago in Chicago, there
4 was an abstract presented again at a low level of
5 0.07, which they reported cases of mesothelioma
6 occurring in that low of concentration.

7 And I can furnish the people, the court, I can
8 furnish that to you, Judge, with the abstract.

9 THE COURT: Okay. Thank you very
10 much. Anything further?

11 MR. OSBURN: Just briefly, Your
12 Honor.

13
14 RECROSS-EXAMINATION

15 BY MR. OSBURN:

16 Q The Hodgson and Darnton article was published in 2000?

17 A Yes, mm-hm.

18 Q And Iwatsubo was published in 2002?

19 A Right.

20 Q You testified in the McPhee trial on March 3rd, 2006,
21 right?

22 A Yes.

23 Q You were asked, "You would not attribute -- if a
24 person had an exposure of less than .1 fiber per cc
25 year, you would not attribute that person's disease of

1 mesothelioma to asbestos?" And your answer, "I would
2 say statistically I couldn't do it based on what has
3 been published." Right?

4 A Right. And that was an error that I made of not being
5 aware of those exact concentrations that were present
6 in the Hodgson and Darnton article and the Iwatsubo
7 article. That was my lack of not keeping totally up
8 to date.

9 MR. OSBURN: That's all I have.

10 THE COURT: Thank you, sir. And if
11 you have that abstract, you should probably return
12 that, the yellow sheet.

13 THE WITNESS: I gave that back.

14 MR. OSBURN: I have that, Your
15 Honor.

16 THE COURT: Okay. Good. Thank you.
17 Thank you very much.

18 MR. FROST: Your Honor, I think
19 honestly anything else would be probably cumulative.

20 THE COURT: Let me tell you what I'm
21 really interested in. I would like, and I'm assuming
22 Dr. Maddox may be doing this, I would like somebody to
23 do some math, give a range of possible fiber year
24 exposures for Mr. Lott and tie it into this testimony.

25 I mean, I've been sitting here trying to do the

1 math myself, but I don't know if I'm right.

2 MR. FROST: Your Honor, the problem
3 with that is that's not generally accepted --

4 THE COURT: I'm asking you, it would
5 help me make my decision.

6 MR. FROST: I understand. I'm
7 just --

8 THE COURT: I'm just asking you to
9 do that. So if there's someone who can do that, that
10 would help me.

11 MR. FROST: Your Honor, can we have
12 a few minutes?

13 THE COURT: Sure.

14 (Recess.)

15
16 THE COURT: Please be seated.

17 MR. FROST: Your Honor, we have
18 talked extensively with Dr. Maddox, and one of the --
19 that's not really his expertise, the numbers. That's
20 more of an occupational medicine type issue. That's
21 why we brought the occupational medicine person
22 earlier. He's unable to come back down right now.

23 But, meanwhile, we took the testimony that he gave
24 and kind of put the pen to paper ourselves. And Glenn
25 is willing to explain to the court kind of where --

1 what conclusions we drew on that.

2 THE COURT: That's fine.

3 MR. FROST: Although, to be honest
4 with the court, I don't think that you need to go
5 there.

6 THE COURT: I know that that's your
7 position. Thank you.

8 So let's do argument then, Mr. Draper?

9 MR. DRAPER: Okay. So Mr. Lott's
10 testimony was that he worked during the school year
11 three or four hours a week and that drywall work was
12 one of the major activities that he did, but it
13 certainly wasn't the only activity that he did.

14 During the summer, he worked 40 hours a week.
15 Most of that work was outdoors, landscaping, but he
16 still did occasional drywall work during the summer as
17 well. And then in addition to the work that he did
18 directly, he also had some bystander exposure to the
19 drywall work.

20 So what I assumed was that Mr. Lott did two hours
21 of drywall or back -- background exposure to drywall
22 two hours a week for three years, which I think is
23 well supported by his testimony. So two hours a week
24 times 52 would be 110 hours a year. Three years would
25 be 330 hours.

1 So now we need to figure out what exposures he
2 received during those 330 hours. And, you know, we
3 have looked at the studies with Dr. Brodtkin, and they
4 found exposures anywhere from 10 fiber cc's all the
5 way up to 46 fiber cc's during the sweeping of a joint
6 compound. I just took the lower number. I assumed
7 during that 330 hours, he's exposed to 10 -- asbestos
8 levels of 10 fiber cc's.

9 So you just do the math. That gives you a total
10 of 3,300 fiber cc hours. Basically, 330 hours at 10
11 fiber cc's is the same thing as 3,300 hours at one
12 fiber cc. A fiber cc year I just assumed to be 2,000
13 hours. So divide 3,300 by 2,000, and what you get is
14 1.65 fiber cc years.

15 This is just kind of a back-of-the-envelope sketch
16 obviously. The way you can see is an order of
17 magnitude above the .1 figure that we were talking
18 about as a possible threshold. So even if we're off
19 by a factor of 10 with this calculation, we're still
20 well above what the defendants have argued is a
21 threshold for causation of mesothelioma.

22 THE COURT: Okay. And is there any
23 other argument you want to make on this issue? I
24 mean, I have read both of the briefs.

25 MR. FROST: Your Honor, I think you

1 are well aware of the issue. I don't see a great need
2 for additional argument.

3 THE COURT: Okay.

4 MR. FROST: Unless the court does.

5 THE COURT: No. Thank you.

6 Mr. Osburn?

7 MR. OSBURN: Your Honor, just
8 briefly. I will respond to this as sort of the rough
9 calculations that Mr. Draper did in a moment. Neither
10 Dr. Brodtkin, nor Dr. Hammar, nor Dr. Maddox, because
11 he says so in his deposition, can quantify Mr. Lott's
12 exposure to joint compound. And they certainly can't
13 quantify Mr. Lott's exposure to Bondex joint compound,
14 which is one of the (Inaudible) that he mentioned.
15 With Bondex, he said that he never -- he doesn't
16 remember working with it, and he can't say that he was
17 working around it. He simply saw a can (Inaudible).
18 So that's the kind of exposure that we're talking
19 about.

20 Exhibit 293 are Mr. Lott's social security records
21 which show how much he worked at the Jewel Lake Villa
22 Apartments. And he worked for a number of apartments,
23 but the Jewel Lake Villa is the one he testified
24 that's where he did the joint compound work. The
25 other ones it was all (Inaudible).

1 And we haven't gone over the figures, and Ms.
2 Loftis has been working on those, so she can address
3 that.

4 MS. LOFTIS: So what I did, Your
5 Honor, was I went through the social security records
6 to see how much money he made during the relevant time
7 period. And in 1973, he made during the entire year
8 \$152, \$44 of which was made during the summer months
9 when he testified he predominantly did landscaping
10 work.

11 But even if you calculate the entire amount of
12 work that he did in 1973, he said he was paid \$6 an
13 hour for interior work, \$10 an hour for exterior work.

14 At \$6 an hour to \$10 an hour, the amount that he
15 was paid equates to 15 to 25 hours during the entire
16 1973. And that 15 to 25 hours he told us in his
17 deposition was equally spent among all of the tasks
18 that he did for his father, which include flooring,
19 electrical work, helping the plumber, painting and
20 then landscaping.

21 In 1974, he worked more hours certainly. The
22 total amount of hours, and again using the same
23 calculation, taking the total amount that he's paid
24 and using a range of \$6 an hour to \$10 an hour is 134
25 hours, so 224 hours. In 1975, he didn't work at all.

1 So there's no work in 1975.

2 And I will hand it back to Mr. Osburn to talk
3 about the significance of these numbers.

4 MR. OSBURN: The corresponding theme
5 between Dr. Brodtkin and Dr. Hammar's testimony is that
6 they cannot come up with a study that says that low
7 dose exposures to chrysotile causes mesothelioma, at
8 least the 0.1 articulated by Dr. Nicholson and
9 ascribed to by Dr. Hammar. They don't know what
10 Mr. Lott's dose was.

11 So in this case, they cannot testify that his
12 exposure to joint was compound was then to the one
13 tenth or one eighth of that to Bondex caused what was
14 a substantial contributing factor to his disease.
15 They simply cannot do it. There's no fiber burden
16 analysis, so we can't tell if there were deposition of
17 asbestos fibers in the lungs, and there's no
18 radiographic evidence there was exposure to asbestos.
19 All you have is this occupational history.

20 I didn't hear any support for low dose from
21 Dr. Brodtkin, just chrysotile causation in general,
22 nothing on low dose exposures. All he was doing was
23 what Dr. Hammar really had to do and what Judge
24 Colville found and what Dr. Maddox is doing is taking
25 a high dose, studies that say there are high doses

1 that cause mesothelioma, and extrapolating down.

2 That's exactly what Judge Colville said you can't do.

3 Dr. Hammar likes to point to certain cases that
4 sort of explain but don't result in epidemiological
5 studies, which is what you have to rely on for
6 causation. He simply says you need a significant
7 occupational exposure.

8 No one has testified or will testify in this case
9 that this was a significant occupational exposure.
10 All they have are generalized statements and case
11 examples of chrysotile causes mesothelioma, asbestos
12 causes mesothelioma. No actual support for their case
13 in this case with Mr. Lott. So low exposures to
14 spouses and children, they carefully didn't mention
15 chrysotile because those were not chrysotile
16 exposures. Those are amphibole exposures from the
17 shipyards to insulation.

18 So what we have in this case is exactly -- it's
19 actually even an even tougher case for the plaintiffs'
20 experts than Judge Colville faced because we have
21 peritoneal mesothelioma for which there are no studies
22 that show that chrysotile is associated with it or at
23 least low doses or as Dr. (Inaudible) states in his
24 testimony ultra low doses, which we have in this case.

25 So we would move the court to strike Dr. Hammar's

1 testimony generally on the fact -- or his testimony
2 that all exposures to disease in this case and -- or
3 failure to demonstrate in this case a causal link
4 between Mr. Lott's exposure and a threshold level of
5 exposure required for peritoneal mesothelioma.

6 THE COURT: Thank you. Mr. Frost,
7 do you have the Hodgson Darnton -- Dartson?

8 MR. FROST: Your Honor, I don't
9 believe I have a full copy. There is one additional
10 article, Your Honor, that I would like to --

11 THE COURT: Actually, I'm really
12 interested in that article. Does it say what the base
13 measurement was? Dr. Hammar referred to Table 11,
14 which indicated a certain number of cases per "X" of
15 population, and I can't remember what the base number
16 was, what the denominator was. Was it 1,000?

17 MR. FROST: It is 1,000.

18 THE COURT: Is it 1,000 or was it
19 more than that?

20 MR. FROST: Your Honor, at .1 fibers
21 per milliliter years, it says the highest estimate is
22 four deaths per 100,000 --

23 THE COURT: 100,000.

24 MR. FROST: -- for chrysotile, for
25 chrysotile. Now, for the others it has -- and that's

1 the article --

2 THE COURT: Let me just ask this
3 question: What is the translation of milliliter into
4 cubic centimeter?

5 MR. DRAPER: I think they are
6 approximately equal.

7 MR. FROST: Well --

8 THE COURT: I didn't have any
9 expressed testimony on that.

10 MR. FROST: There's a differing of
11 opinions as to whether you can do that.

12 THE COURT: Is one generally bigger
13 than the other?

14 MR. FROST: I'm not an industrial
15 hygienist, Judge, so I don't know for sure. I just
16 know there is a dispute in the literature whether you
17 can transpose back and forth.

18 Dr. Maddox says a milliliter is a cc, so they
19 should be exactly the same.

20 THE COURT: Okay. Good.

21 MR. FROST: I'm glad somebody knows.

22 THE COURT: I am ready to give the
23 decision unless you want to make more arguments?

24 MR. FROST: Well, Your Honor, I have
25 one additional paper that talks about low dose

1 exposure to peritoneal mesothelioma that I think --
2 and Dr. Maddox was going to testify about this.

3 MR. DRAPER: I think Dr. Brodkin
4 referred to it as well.

5 THE COURT: Which one is it?

6 MR. FROST: It's asbestos And
7 Peritoneal Mesothelioma Among College Educated Men.
8 It's Plaintiff's Exhibit No. 357.

9 THE COURT: Who is the author?

10 MR. FROST: It's Welch and
11 Sugarbaker.

12 THE COURT: He talked about it.

13 MR. FROST: Okay. And, Your Honor,
14 here is the article. And it deals, Your Honor,
15 specifically with low dose exposure, peritoneal meso's
16 and also deals with individuals that work with brakes,
17 which are chrysotile. And there's no doubt that every
18 single individual that has dealt with brake mechanics
19 and brake linings, that those are all
20 chrysotile-containing products, and there are eight
21 peritoneal mesotheliomas in that group.

22 THE COURT: Is there case studies
23 or --

24 MR. FROST: It is a case study.
25 And, Your Honor, that's the only thing in regards to

1 the statement from the defense that somehow we need
2 epi studies on every single thing, that is just not
3 the law. And that's a 2005 article, Your Honor.
4 That's one of the latest writings in regards to
5 peritoneal mesothelioma and low dose.

6 (Pause in proceedings.)
7

8 THE COURT: And I'm trying to figure
9 out the amount of exposure here.

10 MR. FROST: And, Your Honor, that's
11 why you have to look at the methodology and what they
12 were looking at in that particular study because
13 that's the important part when we're dealing with Frye
14 issues is whether the methodology that folks are using
15 are generally accepted in the scientific community.

16 And all these arguments about what is on your SSPO
17 are great for the jury or for summary judgment, but
18 not for whether the basis of scientific opinions are
19 based on the Hill criteria and based on valid
20 scientific method.

21 That particular cohort, what they did was they
22 looked at individuals who were college educated, and
23 they determined that because they were college
24 educated, much like Mr. Lott, their exposures to
25 asbestos were earlier and very slight, but they still

1 developed peritoneal mesothelioma later.

2 THE COURT: Okay. Thank you. Well,
3 I have read the briefing, and I, of course, read the
4 opinion of the Allegheny County judge. I think it is
5 well established that mesothelioma occurs at 0.1 fiber
6 cc years, and Dr. Hammar seems to think there's
7 evidence that it occurs at below that.

8 I think there is a plausible calculation that
9 Mr. Lott was exposed to that amount or more. And I
10 understand the defense calculation, but I'm not sure
11 that that is dispositive as to the number of hours he
12 actually worked or was exposed, and I'm relying
13 instead on his deposition testimony as to what he did.
14 I think a reasonable jury could accept that as the
15 basis for his number of hours exposed.

16 Assuming that to be true, there's certainly
17 sufficient scientific evidence to prevent Dr. Maddox
18 to testify that this type of exposure, and even if,
19 you know, the Bondex exposure presented some fraction
20 of the total, that Bondex's product could have been a
21 substantial contributing factor to his development of
22 mesothelioma.

23 I certainly do respect Judge Erlick. I just think
24 I come out differently on the issue.

25 The other thing that is of some significance to me

1 is that we have a Court of Appeals decision in
2 Muprutis (sic.) and while this precise issue wasn't
3 presented to that court, they have accepted the type
4 of testimony that Dr. Maddox and Dr. Hammar give to
5 the effect that every exposure contributes, that it is
6 a dose response disease and that the cumulative effect
7 is what ultimately causes his illness.

8 I think that may be a different legal frame work
9 than what we saw in Pennsylvania. So I am denying the
10 motion to exclude Dr. Maddox. Dr. Maddox will be
11 permitted to testify. And I'm not imposing any
12 restrictions on the testimony in the manner that Judge
13 Erlick did.

14 Do you all need a break or can we bring in our
15 jurors? They've been very patient.

16 MR. FROST: If we could just have a
17 short break --

18 THE COURT: Let's have a five-minute
19 break. They've waited for two hours and 15 minutes,
20 so we need to --

21 MR. FROST: And I apologize for the
22 length of the hearing, Your Honor. I just wanted --

23 THE COURT: That's all right. We
24 now have a complete record I think. And I'm giving
25 you back this one.

1 MR. FROST: Thank you, Your Honor.

2 I'm trying to be better with the exhibits this time.

3 THE COURT: Okay.

4 MR. FROST: I'm trying.

5 THE COURT: Good. Thank you.

6 (Proceedings concluded.)

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4 SUPERIOR COURT OF WASHINGTON FOR PIERCE COUNTY
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6

7 SANDRA LOTT,)
8)
9 Plaintiff,)
10) No.
11 vs.) 05-2-06955-4 SEA
12)
13 BONDEX INTERNATIONAL,)
14)
15 Defendant.)
16)

17
18 CERTIFICATE
19

20 STATE OF WASHINGTON)
21) ss
22 COUNTY OF KING)

23 I, BARBARA CASTROW, Certified Court Reporter in the
24 State of Washington, County of Pierce, do hereby certify
25 that the foregoing transcript was transcribed from an audio
recording to the best of my ability.

Dated this 6th day of December, 2006.

BARBARA CASTROW
Certified Court Reporter
CCR #2395