ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY ARISING FROM THE USE OF ASBESTOS IN ONTARIO

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180 Dundas Street
Toronto, Ontario
Thursday,
August 13, 1981

Volume XXVI

ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY

ARISING FROM THE USE OF ASBESTOS IN ONTARIO

VOLUME XXVI

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DR. DUPRE: That is my understanding.

MR. LASKIN: Yes. Everything is still a hold in the last week. The Monday and Wednesday, the 24th and 26th, are holds in the afternoon only.

DR. DUPRE: All right.

DR. UFFEN: What about Tuesday, the 25th, as a hold?

MR. LASKIN: All day.

DR. DUPRE: Is a hold, and so is the 27th.

MR. LASKIN: Dr. McDonald is still coming on the 27th, Alison McDonald that is.

DR. DUPRE: Does that establish our holding pattern then, counsel?

MR. LASKIN: It does, indeed.

DR. DUPRE: Do the parties have any further comments or questions?

Very well, I understand that today we greet Dr. Kenneth Crump, is that correct?

MR. HARDY: Dr. Kenny Crump, officially, on the birth certificate, I believe.

DR. DUPRE: Dr. Kenny Crump.

THE WITNESS: Right.

DR. DUPRE: I understand that you will be leading the examination, Mr. Hardy?

MR. HARDY: That's right, Mr. Chairman.

DR. DUPRE: Well, may I on behalf of all of us, welcome you, Dr. Crump, most warmly for agreeing to come and give sworn testimony as an expert witness.

Miss Kahn, would you swear in the witness, please?

DR. KENNY SHARMAN CRUMP, SWORN

EXAMINATION-IN-CHIEF BY MR. HARDY

Q. Dr. Crump, I think it might be helpful for the

Q. (cont'd.) Commission if we first have you describe some of your background and how you got involved in the field of risk assessment and biostatistics. Why don't we start with your educational background?

A. Okay. I have a bachelor's degree in electrical engineering from Louisanna Tech University, and a master's degree in mathematics from the University of Denver, a Ph.D. in mathematics with an emphasis in statistics from Montana State University, and the last year of graduate school I spent at the State University of New York at Buffalo in their statistics department.

Q. I gather after you got your Ph.D. you went into academia?

A. I spent thirteen years as a professor of mathematics at Louisanna Tech, that's right.

Q. In the last year you haven't been a professor, I gather?

A. While I was a professor, I was on leave on a number of occasions working in fields related to human health and eventually in risk assessment. I spent a summer at Oak Ridge, I spent part of the summer at the National Heart and Lung Institute, part of a summer at the National Institute of Environmental Health Sciences, and then later I spent almost a year at the National Institute of Environment Health Sciences. That was in 1974 and 1975, and at that time I became particularly interested in assessing of risk from environmental contaminants and took up research in that field, working in the field since that time.

A little over a year ago I resigned from the university to devote my time totally to research and consulting in the field, and I formed my own consulting company.

Q. Starting with the year when you were a fellow at the National Institute of Environmental Health Sciences, did you begin working on publishing some papers dealing with theories

Q. (cont'd.) of risk assessment?

A. I've published about, I guess half a dozen, six to ten papers in the field of risk assessment, in the literature. One of the early papers dealt with some theoretical study of what the shape of carcinogenesis dose-response curves might be at low dose. Later work dealt with procedures, statistical procedures for estimating low-dose risk from high-dose data. I had a contract from the National Institute of Environmental Health Sciences, while at the university, to work on that particular problem.

As an outgrowth of that project, some of the techniques which we developed have been adopted for use by the EPA in the United States, for setting model criteria for carcinogens.

Q. You've discussed a number of projects you've done consulting for U.S. government health-related agencies. Have you also done some consulting for nongovernmental bodies?

A. I've consulted with Kirknell and Ellis on asbestos for the last year and a half. I've consulted with Battelle Laboratories, Electric Power Research Institute, American Petroleum Institute, some other law firms and some other government agencies as well.

Q. What I would like to do today, Dr. Crump, is to talk to you first about some general concepts of your view of how best to do risk assessment, some relationship as we go through them to the asbestos data, and then perhaps go through some preliminary calculations you have done from some of the existing data - risks predicted by those studies.

Perhaps to start we should talk a little about different means of expressing health risks, and maybe you could tell us something about how they are traditionally expressed and your personal preferences on how to express health risks.

A. A traditional measure of risk in an epidemiological study is standard mortality ratio, SMR, or equivalently a relative risk, which is basically a weighted average of relative risks in the various age categories in the cohort, and relative risks which are bigger than one indicate there is some relationship between exposure and a health effect.

That particular measure, although useful for determining whether or not there is an effect, is not particularly useful, in my judgement, for determining the health consequences of an effect.

For one thing, we are talking about a relative risk, and if you have a large relative risk - in other words, you have a much higher incidence of disease in an exposed cohort - if the overall base level of risk, of disease incidence, in the unexposed cohort is low, the amount of..the total amount of disease that you are talking about may still be fairly small.

So, for example, a risk ratio of one point five with respect to something like simple heart disease might be more important as far as human health than a risk ratio of ten for a relatively rare disease.

There are some other measures which can be used for estimating health effects. One of these is the additional risk, additional lifetime risk of a particular disease. For example, if you know the lifetime risk, say of lung cancer, in a standard population, and using some data on exposure to asbestos you could estimate the lifetime risk at a certain exposure pattern of lung cancer in a population exposed to asbestos and just taking the difference of those two estimates, you could estimate the extra risk of lung cancer.

This measure has some advantages over relative risk. I think it also has some drawbacks. For one thing, it doesn't incorporate the amount by which a life is shortened by

A. (cont'd.) death from disease, and I think that's an important issue. Most people, I would guess, are more concerned about when they die more than the specific cause of death, and most people would prefer death to a number of causes at age seventy as opposed to death from any cause at adolescence.

I think the time at which a person dies is an important concept which needs to be taken into account in a risk assessment. One way to do that is to use a measure that incorporates length of life, such as loss-of-life expectancy. This measure incorporates both the risk of acquiring the disease, plus the loss of life resulting from the disease.

I think another advantage it has is that you can apply this one single measure to diseases, to exposures which might cause increases in a number of different diseases. You can compute loss of life expectancy from all diseases combined, and as an overall measure, a single measure, of human risk from a particular exposure.

I think I would...given that you have the data available for making an estimate of loss of life expectancy, I would prefer, I think, using that measure over the other measures I talked about.

I have heard some objections to the use of loss of life expectancy on the grounds that if you estimate a loss of life expectancy of say one month, what that really means is that there are a lot of people that didn't get the disease, but there may be a few that lost twenty years of life, so it doesn't really reflect those twenty years.

However, I feel that any reasonable measure of risk has to reflect both the chance of getting the disease and the resulting loss of life expectancy, and this measure does that.

It's not reasonable just to look at the loss of life in affected persons. If that were the case, we would be concerned

A. (cont'd.) by any risk to children, such as riding in cars, because the ones that do lose their lives lose a great, a major portion of them.

There are some other objections to the use of loss of life expectancy which would also apply to these others in the same way. One objection might be that it doesn't reflect the quality of life. A person may become sick with some disease and live a very low-quality of life for a number of years, but survive.

There are modifications that can take that into account. For example, you might want to look at the loss of life expectancy of a particular quality, and look at the time not until death, but to the time until the diagnosis of a particular debilitating or life-threatening disease, so if one wanted to, you could make that modification to overcome those kinds of objections.

Q. Just to backtrack a little from that means of expressing risk, one other issue I think it would be useful to have you discuss would be the question of whether relative risks stay constant throughout a person's life. I think there has been some discussion of that here previously, and the use of some of the asbestos data, and maybe you could walk us through that issue. It would be helpful.

A. Okay. I've got a slide here, I think, that we can use as the basis for a discussion.

This is a graph of a relative risk of lung cancer in insulation workers, Selikoff's study, plotted against years since onset of work. You see that the relative risk began to rise around a little less, well, at least ten years past exposure on up to a maximum of thirty years past exposure, and from that point on they began to decrease. This pattern is typical. I think it has an important implication for risk assessment.

First of all, obviously if you...if the data is predominantly drawn from this area and you haven't had enough followup and you use that to estimate your relative risk, you

DR. UFFEN: (cont'd.) attention to it.

THE WITNESS: Well, in answer to that..I'll not answer the first question. I think the answer to that is no, you don't really need to get into it.

DR. UFFEN: All right.

THE WITNESS: Is that good enough?

DR. UFFEN: Yes.

MR. HARDY: I've always wanted to ask that question, too, but we'll do that some other time.

MR. HARDY: Q. We've been talking a lot about principles of risk assessment, and your judgements on how to best do them, and I think you've done some preliminary calculations based on the McDonald data which take us through the steps you would use in making a risk extrapolation from that data. Maybe if you could take us through what you've done, then give them the charts to do it with, we would see how these principles are used in action.

I think these charts are all going to be in order as we go through, so they should be pretty easy to follow.

THE WITNESS: A. They may not be exactly in order, but they are all...

Q. But they are all numbered.

A. I made some calculations based upon the McDonald data. Quite frankly, a lot of the issues that I've discussed here one cannot apply or take account of the way that I would like to, because this had to be based upon the published data. Some of the data that one needs just was not published.

Q. Would this be data that's unavailable, or just not published?

A. It's just...the raw data is not presented in a way that would be appropriate for what I would like to do with the data.

A. (cont'd.) Because I think it's important to consider the effects of smoking, the interactive effects of smoking and asbestos exposure upon lung cancer, I decided to base the analysis and tell you about this table here, which comes from McDonald et al, 1980, paper, in which he has broken the cohort down by degrees of dust exposure accumulated at age forty-five, and by degrees of smoking. Those are the categories he has the observed numbers of lung cancer deaths and the expected number based upon a reference population.

Dust exposure accumulated at age forty-five may not be the most appropriate measure of dust, but this is the only one in which he breaks out the effects of smoking, at least in a prospective type of study.

So the first thing I did was fit some mathematical models to these data to see how well they fit. I can fit a number of things to these data. I'll just mention two things.

One is what I call a multiplicative model...the RR stands for relative risk...the alpha, beta and gamma?...delta. The alpha, beta delta are parameters if you estimate the data. D is asbestos dose, X is cigarette dose.

In the multiplicative model...we'll call it multiplicative because the effect of smoking multiplies the effect of asbestos exposure, and vice versa. There have been some investigations into the interaction of smoking and asbestos to suggest that the effect is indeed multiplicative.

But each of...the effect of each exposure taken by itself is linear. In a fixed level of smoking, the relative risk varies linearly with respect to asbestos exposure.

The additive model is similar, except the effect of smoking adds to the effect of asbestos exposure rather than multiplying it.

So, I took these two models and actually a number

A. (cont'd.) of other models, and fit them to the data to see what happened.

Here are the results of this exercise.

Q. This is table four, for the record.

A. What I have here are the actual numbers predicted by these two models compared to actual observed values, the numbers of lung cancers, I have the parameter estimates, and I have the results of a chi square electron spin test.

The P value for the multiplicative model is point seven six, which indicates that this model provides a very adequate fit to these data. The P value for the additive model is point zero one, which indicates that the fit of the additive model can be rejected by this point zero one level.

The smoking levels were not given by McDonald at all, so I had to just assume the values for the smoking levels. I just gave them nonsmoking, moderate and heavy smoking.

I did change these numbers up in ways that I thought might be reasonable and didn't materially affect the fits of these models to the data.

So the multiplicative model fits quite well. The additive model didn't fit very well at all.

I actually used some other models in which the relative risk varied as a square of smoking or as a square of asbestos exposure. None of those models materially improved the fit of the multiplicative model in which the variation was linear with smoking and with asbestos exposure.

So I decided to use the results of fitting on the multiplicative model to estimate risks of lung cancer.

Essentially all I get out of this fitting is the value of this potency parameter for asbestos. This alpha is the asbestos potency parameter point zero zero one five nine, so that means essentially that the relative risks from exposure to

A. (cont'd.) asbestos will be one plus alpha is point zero zero one five nine, times the dose. That's the model that I'm using.

It's age independent. It might still happen that these relative risks vary with age as are observed in the Selikoff cohort, but there are no data available in this published data to take that possibility into account, so I am assuming a simple constant relative risk over all ages.

Now, since I want to estimate risks separately in smokers and nonsmokers..let's see, this is table six. I'll show you table five first...I wanted some age-specific mortality rates which could be applied to smokers and nonsmokers separately, and I used two sources for such data. One is the smoking study of U.S. veterans, made a number of years ago, and what I get from the study are the mortality rates in nonsmokers and smokers for all causes and for lung cancer.

Since future populations may have different mortality rates than these U.S. veterans, it's worthwhile to look at other populations in addition to just this one, so I also looked at the smoking data from the British doctors.

Now, the same type of data are available for both sources, the age-specific mortality rates for smokers and nonsmokers taken separately for lung cancer and for all causes.

The estimates that I'm going to come up with are a function of these mortality rates in that potency parameter of alpha which I estimated on the previous slide, and I think I can sort of just generally tell you what I did.

First of all, risk of lung cancer, risk of dying of lung cancer, can be calculated from these lung cancer rates and the total mortality rates. Likewise, a life expectancy can be calculated from total mortality rates. And this gives the mortality rates in nonasbestos-exposed persons, which I'm going to use, and

A. (cont'd.) the only thing that remains to be done is to estimate those rates in asbestos-exposed populations and plug them into the appropriate formulae.

The way I did estimated dose for asbestos exposure is quite simple. Take the one hundred and twenty-six value for lung cancer in the fifty-five to fifty-nine age group - the mortality rate is a hundred and twenty-six with that exposure to asbestos. I estimated the effect of exposure to asbestos by multiplying that value by one plus the alpha, which was estimated in the previous slide, times dose where dose is cumulative exposure to age forty-five.

Once you get those things estimated, you just plug in the appropriate formulae to get extra risk of lung cancer death and loss of life expectancy.

Now, I guess the only remaining question is what dose to plug in there, and I started to ask the question of what risk would result from exposure under a two fiber per milliliter standard. So I had two fibers per milliliter, I wanted to account for the fact that the true exposure might be less than two fibers per milliliter, I decided to divide by two and assume that on average a two fiber per milliliter standard would result in an average exposure of one fiber per milliliter. The data from factory inspectorate of Great Britain indicates this might be a conservative assumption, that true exposures might be less than that.

The measurements which were made, the measurements of fiber counts which were made in the McDonald data, are made by static samplers and the standard might be enforced by personal samplers. There's some indications that there's a difference in the measurements you get from static samplers and personal samplers, and I'm sure the data indicate that effect is quiet variable depending on where you have the static sampler in relation to the

A. (cont'd.) individual.

There is some data in the Simpson Report that indicates that personal samplers give higher readings on average that are greater by a factor of two, so I decided to use that value, so I put another factor of two in there to account for difference in personal versus static sampling.

The most recent paper, McDonald et al, indicates that on average one million particles per cubic foot is equivalent to three point one four fibers per milliliter, so that would be three point one four fibers per milliliter per particles per cubic foot.

So this converts fibers per milliliter to particles, million particles per cubic foot. Now, what we need to plug into the equation is the total exposure through age forty-five. If we assume the exposures we get at age twenty and up to age forty-five, multiply by that, and you get, I think, four point zero million particles per cubic foot years.

Q. So that would be the cumulative dose for a worker working for twenty-five years at a two fiber standard?

A. Yes, that's what that represents.

Q. Put in terms of particle counts?

A. Yes.

Q. In order for you to use the McDonald data to estimate risk?

A. Yes. Cumulative exposure to age forty-five is the appropriate thing to calculate here because that was exposure which was used to estimate the potency.

Okay, so basically what I did was use this for the dose, plug in that linear relative risk model that I showed you, and this next slide shows you the results of that.

MR. HARDY: Table seven.

THE WITNESS: Table seven.

THE WITNESS: (cont'd.) I estimated risks separately in smokers and nonsmokers. You take the best estimate of the potency parameter, the extra risk of lung cancer - no matter whether you use the British doctors or the veterans - is around the order of one in twenty thousand.

MR. HARDY: Q. That's for nonsmokers?

THE WITNESS: A. For nonsmokers.

If you look at the...if you take the upper and lower confidence limits of that potency parameter it varies from about one in fourteen thousand to one in fifty thousand.

There seems to be relatively good agreement between using the British doctor data and the U.S. veteran data.

That is an area of uncertainty because what you would really like to be using are the mortality rates of some future population, which you don't have available.

For smokers, the risks are on average about twelve times higher, it looks like, on average. The extra risk of lung cancer from the asbestos exposure, the best estimate is like one in two thousand. Confidence limits range from one in thirteen hundred to one in forty-nine hundred.

The next slide shows estimated loss of life expectancy - the same situation as I had in the last slide. For nonsmokers, the best estimate is point one six days for... using the British doctor data...and point two using the U.S. veterans data. That's roughly about three hours.

Q. So what you are saying there is that your calculation says that the average loss of life expectancy for twenty-five years of work at the two fiber standard is about three hours for nonsmokers, for lung cancer?

A. That's what this estimate shows.

Q. And for smokers, about two days?

A. Yes. I really think the estimates might be

A. (cont'd.) applied to not just twenty-five years of exposure, but to longer exposure because the actual McDonald cohort was exposed for longer than twenty-five years. It's just that they only accumulated dose up to twenty-five years.

DR. MUSTARD: This is also based on the kind of fiber and kind of exposure that occurred in the Quebec mines?

THE WITNESS: Your estimating..?

DR. MUSTARD: I mean this estimate applies to that?

THE WITNESS: Yes. I would be reluctant to apply this outside that context.

MR. HARDY: Q. It's a little hard to know what it means to have an average loss of life expectancy of three hours or two days. Do you have some means of putting that in context?

THE WITNESS: A. I have a slide here that estimates loss of life expectancy from other types of endeavors. It might help to put that in context.

Q. This is table fifteen.

A. I have here loss of life expectancy from occupational accidents (few words inaudible) change from the paper by (inaudible)...I'll give you the exact reference, if I have it... the Journal of Health Business, I believe, in which he says...

MR. HARDY: Dr. Crump, I think the reporter is having a little bit of trouble.

THE WITNESS: Oh, excuse me. I don't mind him interrupting if he's having trouble.

Okay, for occupational accidents, these estimates, as you can see, range from thirty days to three hundred and twenty-eight days. It's interesting that the highest risk comes from mining and quarrying. I'm not sure if these can be applied to the Quebec miners or not, but if they can, it's obvious that they are comparing this estimate of loss of life expectancy to that on the previous slide. It's obvious that their risk from

A. (cont'd.) asbestos exposure, given that these estimates are correct, would be a very minor portion of their total occupational risk.

Q. I guess what we are comparing is, when you make that statement, is the average loss of life expectancy among miners is three hundred and twenty-eight days because of accidents, versus the risk we have been talking about of three hours for nonsmokers,

A. ...two days.

Q. ...and two days for smokers.

A. Right.

Another risk which might be related to occupation would be commuting to work by automobile, so I estimated a loss of life expectancy using the U.S. 1976 traffic accident statistics and mortality statistics, and using their estimate that roughly thirty percent of all travel in the United States is to and from work.

If you use that estimate, you get a loss of life expectancy for commuting to work by automobile of seventy-eight days.

I also have three estimates of loss of life expectancy from smoking. There is a problem here because mortality rates from a lot of different diseases are increased among smokers, but some of these may be due to confounding with other types of exposures.

For example, smokers have a higher incidence of cirrhosis of the liver than nonsmokers, but this is probably due to confounding with drinking habits rather than due to smoking. So because of the uncertainty there, I provided three estimates. The first category are mortality from diseases which were fairly certain. This dichotomy is actually based upon what Doll and Peto have in their 1976 paper - that's Richard Peto, Julian's brother, by the way.

A. (cont'd.) So you get six hundred and ninety days for those diseases which you are fairly confident are caused by smoking. You add that to category two A, which is likely to be caused by smoking, it doubles it, and there are various other causes which I haven't listed here which are probably, in the words of Doll and Peto, attributable to smoking. If you include those you get sixteen hundred and eighteen days. What's that? Roughly four years?

Q. What sort of smokers do they calculate that for?

A. I think we are talking about the average amount smoked by the smokers in the Doll and Peto study of the British doctors, and their average smoking rates, I believe, is about eighteen cigarettes a day. Not heavy...I wouldn't call that heavy smoking. Average smoking? I don't know. I think heavy smokers smoke more than that.

I think it's instructive to apply, compare this with the risk to smokers that were estimated in the previous slide, which is around two to three days, and if those estimates are valid then we see that the smokers' risk of lung cancer is affected only very slightly by their asbestos exposure in this situation.

Q. I think you talked about the lung cancer risk calculated from the McDonald study, but I believe you've looked at some other possible health effects and accumulated - not only lung cancer effects, but some of the other risks that appear to come out of that data?

Table twelve might be the best.

A. I applied similar procedures to estimate risks of cancer of stomach or esophagus, using some data that was in the McDonald study, and there is no evidence that I know of that relates those diseases to smoking habits. Nevertheless, I made

A. (cont'd.) the risk estimates separately for smokers and nonsmokers because they do have different mortality patterns, and you see that even though in fact the extra risk of death from stomach cancer is greater in nonsmokers, simply due to the fact that they live longer and have longer to contract the disease, it turns out in nonsmokers this procedure estimates the risk of stomach cancer to be greater than that of lung cancer.

But for smokers, the lung cancer risks still dominate.

The pneumoconiosis risk estimates, I feel are even more questionable than the ones I have already presented. I didn't feel like the method I was using was appropriate for disease such as pneumoconiosis, which is not diagnosed unless you have exposure to some type of dust, and what we really would like to have is the extra risk, given exposure versus no exposure to asbestos, and the procedure I have been using was not appropriate because those cases that you see in the general population are not background cases, but they are caused by exposure to some material, whether asbestos or something else. So I use a different approach for pneumoconiosis just in order to be able to generate some numbers.

I looked at the total excess number of cases of pneumoconiosis in the McDonald study, then I looked at the total excess number of cases of stomach cancer, and got that ratio, and I assumed that ratio would hold more widely and actually use that ratio in this situation.

At any rate, down on the bottom line there I have the totals, and everything is linear here so you can get the total risk of all these diseases by adding things up in the columns, and in nonsmokers from all the disease I have considered here, the loss of life expectancy in nonsmokers is estimated at nine-tenths of a day, to four days in smokers.

A. (cont'd.) McDonald, et al, did not give their data on mesothelioma, so that risk is not included. But they had eleven mesotheliomas and about fifty excess lung cancers, so you can..that will give you some idea of how including those would affect these estimates.

Q. Does that mean it might be another half day?

A. I think something on that order of magnitude.

Q. So that if you were to add the data to include mesothelioma risks, you might be talking about something less than a day and a half for nonsmokers, and something slightly less than three days for smokers?

A. I think it would probably be the same for smokers and nonsmokers because I wouldn't assume a smoking effect, but it would be...I don't think it would materially affect these results. I'm sure they would be less than four days...still less than four days for smokers, and less than two days, certainly, or a day and a half for nonsmokers.

But I can't say for sure.

MR. HARDY: Mr. Chairman, there are two other areas that I think Dr. Crump is going to want to address. One is some review we have done of some of the animal inhalation data with respect to risk assessment, and also he wants to talk a little about some of the mesothelioma prediction issues that Mr. Peto mentioned last week. I think the grand total would probably take about a half hour. I wondered whether you would want to take a break now and we'll come back and do that half hour?

DR. DUPRE: I think it might be appropriate to take a break.

Could I ask the counsel this, during the break period, see if you can get some idea on the amount of time that you will need for your questions, as I have to bear in mind our options, if Dr. Crump is willing to let us have these options,

DR. DUPRE: (cont'd.) would be to perhaps either try to sit until about seven o'clock and wrap this up for the day, or alternatively, perhaps break about five-thirty to return at some...

MR. HARDY: We'll pool our ideas and hope we come up with a good compromise.

DR. DUPRE ; Let us rise until quarter to five.

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

DR. DUPRE: Will you proceed, counsel, please?

MR. HARDY: Fine.

(few words missing due to technical problems)

MR. HARDY: Q. ...asbestos textile factories, and maybe you can just briefly tell us what you were able to learn from those studies?

THE WITNESS: A. I did apply the same general procedures to the Rochdale data and the Dement data, which are both textile operations.

Q. We are looking at table fourteen now, which is in the middle of the addenda. It was, unfortunately, out of order.

A. I didn't go into as much detail with these estimates as I did with the McDonald estimates. I essentially accepted everything the authors said at face value, and just expressed their estimates in different forms.

For example, Peto very crudely estimated relative risk for lung cancer, I think between two and three, from certain crude average exposures, and I just simply took the midrange of those numbers he gave and translated those into additional risk and loss of life expectancy. There was no data on smokers. I just...I didn't fit any smoking data, or anything like that.

A. (cont'd.) For Dement, he did have some table of cumulative exposure versus relative risk. I did actually fit a line to that data and used that fitted line as a basis for estimating these numbers.

Q. With the Dement data, I gather, you accepted the exposure information the way he presented it in his preliminary report?

A. Right. Right. And there is one feature about the Dement study which is different from some of the other studies in that he moves people from different exposure categories as the exposure increases, which other studies didn't do. Whatever their total exposure is, they go into that category and all their person-years of experience fall into that same category.

He moves people from one category, from a low-exposure category to a higher-exposure category as their exposure increases over time, and I took that into account in making these estimates. If one does not do that, but simply does the same thing he does with the other kinds of studies, it would lead to somewhat overestimates, higher estimates at least, of these numbers for risks and loss of life expectancy.

We see here that the same relative pattern between smokers and nonsmokers holds as held for the McDonald study, but the absolute numbers are much larger. Where we had three hours for the McDonald study - loss of life expectancy - the comparable number for Peto's study is three days, three and a half days.

Even though on the surface these studies look very similar, they are both primarily chrysotile and they are both textile operations, the risks of the Dement study are much higher than they are from the Peto study.

DR. UFFEN: When you say you took it into account that he would move people from one category to another, how did you do that? Did you put them back?

TABLE 7
ADDITIONAL RISK OF LUNG CANCER MORTALITY FROM
LIFETIME OCCUPATIONAL EXPOSURE UNDER A 2 f/ml STANDARD[a]

Non-smokers	Using Data From British Doctors	Using Data From U.S. Veterans
α = .00159 (MLE)	1/25,000	1/19,000
α = .00248 (95% Upper Limit)	1/16,000	1/12,000
α = .00070 (95% Lower Limit)	1/56,000	1/43,000
Smokers	British Doctors (Ave. 18.3 cigs/day)	U.S. Veterans (21-39 cigs/day)
α = .00159 (MLE)	1/2,100	1/2,200
α = .00248 (95% Upper Limit)	1/1,300	1/1,400
α = .00070 (95% Lower Limit)	1/4,800	1/4,900

[a] Based upon data of McDonald et al. (1980a).

TABLE 8
LOSS OF LIFE EXPECTANCY BECAUSE OF LUNG CANCER FROM LIFETIME OCCUPATIONAL EXPOSURE UNDER A 2 f/ml STANDARD[a]

	Loss of Life Expectancy (days)	
Non-smokers	Using Data From	
	British Doctors	U.S. Veterans
α = .00159 (MLE)	0.16	0.20
α = .00248 (95% Upper Limit)	0.25	0.31
α = .00070 (95% Lower Limit)	0.07	0.09
Smokers	British Doctors (ave. 18.3 cigs/day)	U.S. Veterans (21-39 cigs/day)
α = .000159 (MLE)	1.9	2.3
α = .00248 (95% Upper Limit)	3.0	3.5
α = .00070 (95% Lower Limit)	0.83	1.0

[a] Based upon data of McDonald et al. (1980a).

TABLE 9
OBSERVED DEATHS AND SMRs FOR CANCER OF THE STOMACH AND ESOPHAGUS
IN RELATION TO DUST EXPOSURE TO AGE 45

Dust exposure (mpcf-y) Accumulated to age 45[a]	(assumed average exposure)	SMR[a]	Observed Deaths[a]	Expected Deaths Under Model (5)
0-30	(15)	122	68	56.6
30-300	(100)	114	42	40.7
> 300	(600)	158	26	26.8

[a] Source: McDonald et al. (1980), Table 8.

TABLE 10
ADDITIONAL RISK AND LOSS OF LIFE EXPECTANCY FROM CANCER OF THE STOMACH AND ESOPHAGUS CAUSED BY LIFETIME EXPOSURE UNDER A 2 f/ml STANDARD[a]

	Additional Risk	Loss of Life Expectancy (Days)
Non-Smokers		
α_s = .00105 (MLE)	1/13,000	0.30
α_s = .00188 (95% Upper Limit)	1/7,400	0.54
α_s = .00021 (95% Lower Limit)	1/69,000	0.06
Smokers		
α_s = .00105 (MLE)	1/21,000	0.20
α_s = .00188 (95% Upper Limit)	1/11,000	0.36
α_s = .00021 (95% Lower Limit)	1/105,000	0.04

[a] Based upon data from McDonald et al. (1980a).

TABLE 11
OBSERVED DEATHS AND SMRs FOR PNEUMOCONIOSIS
IN RELATION TO DUST EXPOSURE TO AGE 45

Dust exposure (mpcf-y) Accumulated to age 45	(assumed average exposure)	SMR	Observed Deaths
0-30	(15)	298	5
30-300	(100)	1081	12
>300	(600)	5400	27

Source: McDonald et al. (1980), Table 8.

TABLE 12
ADDITIONAL RISK AND LOSS OF LIFE EXPECTANCY
FROM VARIOUS ASBESTOS-RELATED DISEASES
CAUSED BY LIFETIME EXPOSURE UNDER
A 2 f/ml STANDARD[a]

	Additional Risk		Loss of Life Expectancy (days)	
	Non-smokers	Smokers	Non-smokers	Smokers
Lung cancer	1/25,000	1/2,100	0.16	1.9
Cancer of Stomach or Esophagus	1/13,000	1/21,000	0.30	0.20
Pneumoconiosis	1/8,100	1/13,000	0.43	0.26
Lung cancer & cancer of stomach or esophagus	1/8,600	1/1,900	0.47	2.1
Lung cancer, cancer of stomach or esophagus, and pneumoconiosis	1/4,200	1/1,700	0.89	2.4

[a] Based upon data from McDonald et al. (1980a).

TABLE 14
ADDITIONAL RISK AND LOSS OF LIFE EXPECTANCY FROM LUNG CANCER
RESULTING FROM LIFETIME EMPLOYMENT IN A TEXTILE MILL
SUBJECT TO A 2 f/ml STANDARD

	Additional Risk		Loss of Life Expectancy (days)	
	Non-smokers	Smokers	Non-smokers	Smokers
Estimated from Peto (1980)	1/1,200	1/100	3.5	41
Estimated from Dement, et al. (1980)	1/150	1/14	26	290

TABLE 15
LOSS OF LIFE EXPECTANCY FROM VARIOUS CAUSES

Cause	Loss of Life Expectancy (days)
Occupational accidents[a]	
Trade	30
Manufacturing	43
Service	47
Government	55
Transportation and public utilities	164
Agriculture	277
Construction	302
Mining, quarrying	328
Commuting to work by automobile[b]	78
Smoking[c]	
(1) Cancer of lung, esophagus, and other respiratory sites; chronic bronchitis and emphysema; and pulmonary heart disease	690
(2A) All causes in (1) plus ischaemic heart disease	1355
(2B) All causes in (1) and (2A) plus various other causes probably attributable to smoking	1618

[a] From: Cohen and Lee (1979).
[b] Based upon 1976 U. S. traffic and mortality statistics.
[c] Based upon data of Doll and Peto (1976).