File No. 65381

C A N A D A

IN THE PROVINCIAL COURT OF BRITISH COLUMBIA

(BEFORE THE HONOURABLE JUDGE F.E. HOWARD)

SURREY, B.C.

1997 JANUARY 31

REGINA

V

VICTOR EUGENE CAINE

PROCEEDINGS AT

CHARTER APPLICATION

APPEARANCES:

T. DOHM/A. CHAN/M. HEWITT for the Federal Crown

J. CONROY/ P. SMITH-GANDER for the Defence

R. WALLS Court recorder

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A VOICE: My apologies, Your Honour. It's my fault.

THE COURT: All right. The Caine matter again.

HAROLD KALANT, recalled, testifies as follows:

THE COURT: Doctor, you are still under oath. You understand that?

THE WITNESS: Yes, I do, Your Honour.

THE COURT: Thank you, sir.

CROSS EXAMINATION BY MR. CONROY continuing:

Q Doctor, yesterday at the end of your evidence you indicated that it was your opinion that the weight of medical opinion, as I understood it, took the view that

use of marihuana was a significant health hazard, is that right?

A Yes, that it posed either recognized problems or the significant risk of problems.

Q And when you say the weight of medical opinion, you were talking about the weight of medical opinion in Canada, were you?

A I would say in Canada, in the United States, in the United Kingdom and in Australia, as represented by the—the findings of the Hall Report.

Q And when you say that, though, are you talking about the weight of the medical people involved in marihuana research, or are you talking about the entire medical professions of each of those countries?

A It—it is really difficult to talk about the entire medical profession, because the only thing that one can use as an index of that would be the expressions of medical societies in the form of statements from the societies after—after extensive discussion, and one really can only form a—at best an impression of that, rather than a hard and fast documented proof.

Q And so am I right then in understanding it was the weight of med—informed medical opinion, if I can put it that way, people who have some knowledge about the acute and chronic effects of marihuana use?

A I think that's a fair way to put it.

Q So we're not talking about the whole professions, but those who have some knowledge?

A I think that's correct.

Q Okay. And when you say Australia, you're obviously relying on the conclusions of the Hall Report?

A That's right, yeah.

Q Of which you—

A And also on the articles that have appeared in the Australian Medical Journal.

Q Now, but when you—when you do that, though, do you say okay, we have so many that have been

prepared that indicate there are risks, and so many other documents or journal articles that have been prepared that indicate that the risk isn't substantial, and you're saying there's more that say it is than aren't, is that what you're saying?

A I wish it were possible to give you a carefully derived answer to that from actual counts, but to my knowledge, that—that really isn't available, so that I would have to come back to what you said before, it is my impression.

Q All right. And are you following this—your impression is—is based on the same type of data when you speak about the United Kingdom or the United States, I take it, or Canada for that matter?

A Yes. Yes.

Q So we're just looking at the—the types of research that have been done and the—the majority of the research, if I can put it that way, indicates the type of risks that are indicated in the Hall Report, for example?

A Yes.

Q Okay. And would you say that the Hall Report and that summary of acute chronic—and these possible effects, provides a fairly good summary of the key concerns, other than I know there was one you disagreed with?

A Yes. I think other than that one, which deals with a very rare occurrence, which is suggested but by no means proven to be related to cannabis, other than that, I would agree that the—the list is a fairly good summary of the areas that have received the most investigation and for which the most concern remains.

Q Now, as I understand it, the only thing that may be more up to date or more current than that is this 1995 W.H.O. Committee prepared report that isn't public yet?

A That's right.

Q Okay. And that contains references, I take it, to research in addition to what's contained or was considered by the people in the Hall Report?

A Yes.

Q Okay. Now, when we talk about significant health hazard, I think you indicated that it's all a question of rates and patterns of use?

A Yes. This is a general principle of all drug-related (indiscernible).

Q And so to determine the scope of the problem, we'd have to know just what the number of users are, and their various levels of use, and how frequently they used, these types of things, wouldn't we?

A Yes.

Q Because as you've told us, if—if it's what we call the occasional user, once a month I think—I think we went to that for the occasional user, --

A Yes.

Q -- it might have been a bit more than that but—but once in a while. It might be once a weekend or something like that, is that fair, --

A Yes.

Q -- for occasional user? And we don't have any significant health concerns about that type of user, do we?

A No, as I said yesterday, other than the potential hazards of being intoxicated while driving, for example.

Q Certainly.

A Other than that, no.

Q The acute effect we talked about?

A That's right.

Q Okay. And the real concern in terms of when we talk about significant health risk, appears from what I'm read—and correct me if I'm wrong—the chronic user?

A Yes.

Q The person who uses, on your definition, one or more marihuana cigarettes a day?

A That's right.

Q Okay. So the—the others, if we can call them the moderate users, again insofar as those people are concerned, apart from the acute effect in terms of driving, their level of use, rate of use, doesn't pose a significant health risk, --

A I would agree with that.

Q -- would that be fair?

A Yes. It's—I could point out the same principle, of course, applies to the use of alcohol or other psychoactive drugs, that the occasional use is not the source of concern. It's regular and—principly regular heavy use that one is concerned about.

Q All right. Now, we know that in the case of alcohol, the numbers of people consuming in relation to the total population are very large, aren't they?

A Yes.

Q And—

A But—

Q -- I think—

A Sorry, may I ask what you mean by very large?

Q Well, a large majority of the population, let's put it that way. I mean, can you give us a figure at all?

A In Canada, the rough figure is about eighty-five percent of adults who use.

Q All right. Which is a very large number, isn't it?

A Yeah, but of course the majority of those are not what would be called heavy users.

Q Yes. Yes. No, I understand. Let's do that. If we say eighty-five percent of the Canadian population consumes alcohol, can you give us a figure about the—the heavy user, what percentage roughly?

A The—the estimates of those who use heavily enough to be considered problem or potential problem drinkers range between five and ten percent.

Q Five to ten percent of—

A Of users.

Q -- that eighty-five percent?

A Of the eighty-five percent, yeah.

Q And again, with alcohol then we don't concern ourselves so much about the remaining percentage. It's—it's that five to ten percent that are the chronic user that are the ones who are predominantly responsible for the—the significant health hazard?

A That's correct.

Q And in turn, in the case of alcohol as I understand it, significant increased medical costs as a result of alcohol usage, fair enough?

A Yes.

Q And other types of impacts on not just the user of the alcohol but the general taxpayer, for example?

A That's correct.

Q Yeah. And with tobacco, can you give us an idea of how many tobacco smokers we have in Canada, percentage-wise?

A I'm not really up to date on that, but my understanding is that it's about thirty percent of adults.

Q Okay. And again the major concern there, I take it, would be the chronic users, --

A Yes.

Q -- the people who were smoking more than a pack a day?

A That's right, or—

Q Or a pack a day actually?

A -- or a pack a day, yes.

Q Now, a pack—

A With—

Q Sorry?

A Sorry, if I may just modify that a little?

Q Yes.

A The—the public health approach, the epidemiological approach, aims to assess the relationship between the risk and the level of smoking by correlating the—the frequency of occurrence of various known medical problems with levels of smoking, and there doesn't appear to be from that approach a—a sort of threshold. There isn't a safe level below which you're not a problem and above which you are. There seems to be a continuous increase in risk with amount smoked and—

Q And—sorry?

A -- and that probably represents the fact that when smoke condensate is deposited in the lungs, it—it remains. The effects are cumulative and therefore what you affect is the speed with which problems develop, but not an absolute threshold or—for safety or for risk.

THE COURT: So you even at one cigarette a day, the—

A The statistics lead—leave you to conclude that if you project the risk backward, you would have to say that even one cigarette a day contributes some extremely small but potential risk. It's the—the problem is essentially a mathematical one that you cannot demonstrate in the relationship a cutoff point.

MR. CONROY:

Q If I understand you, that's because of the nature of cigarette smoking, by taking the cigarette smoke into your lungs, depositing of the tars and the particulates and as you said, accumulating over a period of time?

A Yes.

Q So that if you—it wouldn't be a situation if you were a novice and you only smoked one cigarette. You're talking about a person who smokes one cigarette at least daily over a period of time, and that those

particulates accumulate, is that—am I understanding that correctly?

A That's the—the concept. I have to say that from my own point of view, I find it difficult to think that the—the risk at a level of one cigarette a day would be very significant, but the—the evidence that is available does not permit one to say that there is a safe level below which you run no risk.

And I suppose one would have to say the same thing about cannabis smoking, but if the smoke is treated in the lungs in the same way, then if enough statistics are accumulated over a long enough period of time, as we were discussing yesterday, one would probably come to the same conclusion, that there is no totally safe cutoff point below which there is absolutely no risk, but for practical purposes now, I think we can say that it—it's fair to say that what we're really concerned about is the regular heavy user of either tobacco or cannabis.

Q All right. And the usual or the common user—I think we dealt with this briefly yesterday—smokes, we believe anyway, a pack a day or something like that?

A That would be a fairly typical—

Q Whereas the marihuana smoker, the cannabis smoker, we're talking about—well, various levels of use but in the example, one marihuana cigarette a day, but taken in in a different way than cigarettes?

A Yes.

Q Because if I understood it, the concern or one of the concerns in terms of cannabis smoking is the method of taking it into the lungs and holding one's breath and how that can result in a—a deeper sample, if you will, of the particulates going into your system?

A That's correct.

Q Whereas—

A The—sorry, if I may interrupt here, two concerns. One is the higher content of—of particulates or of solid residue in cannabis smoke than in tobacco smoke. The fact that tobacco cigarettes, the majority are filtered while cannabis cigarettes are not, and the pattern of smoking that you just described, all of those would tend to contribute to a relatively higher deposition of material from cannabis smoke than from the same amount of tobacco.

Q I thought yesterday we—we went over this a little bit, the business of—there was a time when you thought that the—there—there was a greater quantity of particulates in marihuana smoke than tobacco smoke, but that now—

A Oh, no, I think that's—

Q -- they're essentially the same?

A No, no. No, no, I think you must have misunderstood what I'm saying. What I said was that the—the content per cigarette or per gram of material is substantially higher from cannabis than from tobacco, but that what matters is the total amount which is consumed.

What you were asking me about, that I agreed with that had changed, was the concept that there was a higher percentage of carcinogenic material per gram of condensate in the cannabis smoke than in the tobacco smoke, and I think that view has not been borne out by later research, so that to the best of my knowledge, it is still quite true that the yield of—of tar or of condensates per gram of material is significantly higher from cannabis than from tobacco.

Q And the study that says that, a current study that says that, is that referred to in that—in preparation—W.H.O. report?

A No, that was not—my recollection is that that was not dealt with, because that has been well demonstrated in earlier work.

Q Well, what study do you refer to as being the—the final study in relation to that issue?

A Well, I could cite, for example, the paper which you were kind enough to send me last night.

Q Yes.

A The—the Tashkin (phonetic) paper which is in press now, which—

Q Yes.

A -- continues to cite that—that finding as one of the considerations in assessing the risk.

Q Well, that Tashkin—

A In the discussion—

Q Sorry?

A In the discussion of that paper, he uses that as one of the possible considerations in accounting for the disagreements amongst some of the findings, but he accepts that as a—as a given. I think he cites his own work on that—on that count.

Q All right. So you're saying that Tashkin still says that the particulates per gram of material smoked are higher in cannabis smoke—

A Yes.

Q -- than tobacco smoke?

A That's right.

Q But—but the area that's—there's a question mark about is—whether or not there's a difference in the number of carcinogens, is that—do I have that accurately?

A Yes, that was—that's the issue on which I believe the predominant view has changed.

Q Okay.

A It was asserted at one point that the—the percentage of carcinogenic material in the—the tar was higher in cannabis than in tobacco, but I don't think that is generally supported by all of the analytical data.

Q All right. Well, I'll come back to that in more detail because I want to deal with that Tashkin's report at some length, but let's come back to where—what we were talking about. So we have—I'm sorry, you said about thirty percent of the population is tobacco smokers is the—the Canadian population?

A That's the figure that I—

Q And that's—

A -- think is correct.

Q -- substantially less than what it used to be—

A Yes.

Q -- just a few years ago, isn't that right?

A Yes.

Q And my understanding is that reduction has been achieved through education, widespread putting out of material about the damage or the hazards of cigarette smoking and so on, isn't that right?

A Of the various discussions that I've read of the explanation of that decline, several factors are credited. One is education.

Q Yes.

A Another is a general change in lifestyle with an emphasis on more physical fitness and physical activities among the young adult group, and the feeling that tobacco smoking is incompatible with that type of lifestyle. And I suppose, in many cases that I know of, the impact of children on their parents.

Q Yes. That's an interesting one, isn't it?

A Yes, it is.

Q Many people say, "Well, what are you going to tell your children about smoking marihuana?" but the fact seems to be that in relation to tobacco, it's the children that are getting their parents to stop smoking, isn't it?

A Yes.

Q Now, it certainly wasn't the use of the criminal law, didn't make it an offence subject to penalty to someone in order to get people to reduce their smoking?

A No.

Q That's not one of the—

A That is not—

Q -- means resorted to, is it? And in relation to the smoking of tobacco, we know that there's a vast body of medical evidence that attests to substantial devastating health consequences, don't we?

A Yes.

Q And we know it's one of the leading causes of death in Canada, don't we?

A Yes.

Q We know it's one of the leading causes of illness in Canada, don't we?

A Correct.

Q And there's various types of disease and so on that it causes that are well documented, isn't that right?

A Yes.

Q I mean well documented not just from research and statistics and so on, but well documented from people going to see their doctors and telling them that they're having problems with bronchitis or emphysema or any number of different lung-type problems that we attribute to tobacco smoke?

A Yes.

Q And we know that that in turn, given the large number of users and the amount that they use, that that in turn translated into significant costs and medical costs and things of this nature?

A That's correct.

Q All right. So that—and as I understand it, we also know that in relation to tobacco smoke or tobacco smokers, not only was the use of the substance harmful to the user, but—and—and impact on society as a whole in terms of medical costs and so on, but also it could potentially be damaging to somebody nearby the smoker because of—

A Through second-hand smoke.

Q -- contamination of second-hand smoke and things of that nature?

A Yes.

Q Fair enough. And that the—the pattern of use of the cigarette smoker that would impact upon other citizens often would be as a result of people smoking in a bus or an elevator or a restaurant or places where they were—where other people were present, not participating in

the smoking with them or that could be effected simply because it's out there in society in a place where there other people, is that right?

A Yes. Interestingly, your question a moment ago about the government not using legal controls for tobacco, that's the one issue where perhaps it does not apply, because there are local regulations passed in many places to prevent exposure of non-smokers to smoke from smokers, in—

Q Yes.

A -- restaurants for example or—

Q It's quite a recent phenomenon?

A Yes, it is.

Q And it's an example of local government, municipal governments, city governments, things of that nature addressing a local problem, isn't that correct?

A Yes, that's correct.

Q So that they're able to pass non-criminal type laws to try and encourage people to look out for other people that might be affected healthwise?

MR. DOHM: I have to object to that, just for the purpose of saying that it's very difficult to expect most witnesses to know the difference between a control which is criminal, and one which is non-criminal. There is—that's—that's a tough question for many trained in the business, let alone for the witness.

MR. CONROY:

Q Well, I have absolutely no doubt that Dr. Kalant has no difficulty in understanding the difference, do you, Dr. Kalant? I mean you've written papers about social policy in relation to the control of drugs, haven't you?

A Yes, I have.

Q And when you wrote those papers, you took into account the means of control, didn't you?

A Yes.

Q And particularly I'm thinking of the paper that is called—if I can put my hands on it—here it is—Drug Policy: Striking the Right Balance?

A Yes.

Q It's at tab two, I believe, of the Crown's Brandeis brief, and it was written by you and Avrim Goldstein in 1990 and printed in Science Magazine?

A That's correct.

Q Yeah. And in that document, you talk about not just marihuana but all the different types of drugs, including alcohol and—and tobacco, isn't that right?

A That's right.

Q And in fact, you gave examples in one of the footnotes about different means of controlling the conduct of people in terms of some of the things that we use that could impact upon others or—or on ourselves. Do you recall that?

A You're referring perhaps to the footnote about vending machines or things of that sort?

Q The one I had was requiring people to wear motorcycle helmets for example?

A Oh, yes.

Q And you know, that's a provincial Motor Vehicle Act type of statute?

A Yes.

Q Another one was safety belts in cars, again Motor Vehicle Act type stuff?

A Yes.

Q Another one was pasteurization of milk?

A That's right.

Q I'm not sure—

A Fluoridation of water.

Q The fluoridation of water was another one which, if my memory serves me, is something that the province requires or a local municipality may require?

A Yes.

Q In fact, that's what you—you noted was fluoridation of municipal drinking water supplies, and another one is immunization of school children, fair enough?

A Right.

Q And none of those are dealt with by any federal statute like the Narcotic Control Act that we're dealing with here, are they, to your knowledge?

A No, to my knowledge they're—they're regulations rather than criminal.

Q Yeah. Okay. Now, let's come back then to cannabis. We've talked about eighty-five percent of the population being alcohol consuming, about five to ten percent of them being the chronic user. Tobacco is at thirty percent now, with a significant reduction over the last few years. Tobacco smokers—can we put a chronic user figure on that percentage? It would seem from our discussion to be fairly high, a fairly large percentage of the thirty percent?

A Yes. Unfortunately, I—I don't know the exact percentage. I know that the majority of smokers would be considered toward the heavy end rather than the light.

Q Okay. So what then about cannabis smokers, how—what is the percentage of cannabis smokers in Canada, do you—can you tell us that?

A Unfortunately, I don't have the—I'm not knowledgeable on the most recent statistics, but my understanding is that the figure of something like ten or twelve percent of the general population are ever users, that is have at some time used. What the percentage of current users is, I'm afraid I don't know at the moment.

Q But we do need to know that, don't we—

A Yes, we do.

Q -- in order to determine the scope of the risk, don't we?

A Yes.

Q And so if—if you don't know the total or the estimated number of total users in Canada, it's also then difficult to figure out what their pattern of use is, isn't it?

A Well, may I point out here I said I don't know. I don't mean that it is not known.

Q Yes.

A If one wishes to put a—an estimated figure of total risk or total social cost or whatever, you certainly would need to use such figures, and such figures are available from surveys. All I'm saying is that I personally at the moment cannot give you such a figure.

Q Yes, and you can't give me the figure for how many—well, actually yesterday I think you said the chronic user estimate was about—in your view about five percent?

A Of users, yes.

Q But—but don't we need to know what the total number of users is—

A Yes.

Q -- in order to figure out what your five percent means?

A Yes, we do, and such figures, as I say, are available. I'm simply saying that I regret that I'm not in a position to give you those figures right now.

Q But when you say five percent, don't you have in your mind five percent of a figure?

A No. I wish I did, but unfortunately I—at the moment, I cannot think of the most recent figures—

Q Because—because in order to determine the nature and scope of the health problem, we need to know those figures, don't we?

A Yes, certainly.

Q But you don't know them, do you?

A No, I don't.

Q And so you can't tell us what the extent or the nature and scope of the risk is here today?

A No. No. The nature is what I have attempted to outline in—

Q The scope, sorry.

A -- my testimony—scope, but the—the extent I really cannot give you a valid estimate.

Q Now, when somebody has a problem with irritation of the lungs from smoke, bronchitis, emphysema, these sorts of things, they normally go to a physician for help, don't they?

A Yes.

Q Or to a hospital?

A Or to a hospital, yeah.

Q Or a person who feels that they're dependent on—on the substance and wants to stop using it, they might go to a counsellor or to some course or—or something like that in order to—to get some help, isn't that fair?

A Yes.

Q And similar approaches would be taken with alcohol, wouldn't they?

A Yes.

Q If somebody's got a health problem, that's usually what they're going to do?

A That's correct.

Q And the doctors and the hospitals, to the extent that they can persuade people to stop or change their ways -- first of all in relation to tobacco, I think we've already discussed we've been reasonably successful in accomplishing that, haven't we, without some big stick of the criminal law there, haven't we?

A Yes, I think that's—

Q And the same—

MR. DOHM: I'm going to object to the use of criminal—of the term criminal. I continue that objection. The Supreme Court of Canada has said in Howser (phonetic), very clearly, that this is not criminal law and with all respect, I do believe that my learned friend is unwittingly misleading the witness.

THE COURT: It is a loaded term, punitive.

MR. CONROY: Well, I don't—provincial laws can be punitive, too. I—you know I think that that's what this case is all about. It's about whether or not Parliament has exceeded its constitutional jurisdiction and one of the categories that they have power over is criminal law. They don't have power over matters of a purely local and private nature, and let me maybe develop it a little bit further to—in terms of the witness' expertise.

Q As a physician, you're concerned about what the government does or doesn't do that may impact on the health of your patients, aren't you?

A Yes, I think that's reasonable.

Q And so you—that's why you're concerned about the social policy that the governments adapt in relation to one drug or another?

A Yes, I think that's fair.

Q And you agree that different drugs have different effects, depending upon patterns of use and so on that we've discussed?

A Yes.

Q And you think that one policy is appropriate for one, and not for another, depending upon the circumstances and the facts in relation to the particular drug?

A Yes, I think that's true.

Q And you agree that some matters, some things, while posing a significant health risk to people can be dealt with at a local and private—or local, city, municipal, village level?

A Yes, some are.

Q And some require perhaps a province-wide approach?

A I think there I would be getting beyond my competence to discuss what types of powers that different levels of government are—

Q Let's just focus on the health issue. Some health issues can be dealt with at the local city, municipal, village—

A Yes.

Q -- doctor, hospital level, can't they?

A Yes.

Q Some health issues might require something more than that, they might require a provincial policy for the whole province because it's considered to be—require a little more than just the local doctor in the hospital, isn't that right?

A Yes.

Q And some matters, if you take an extreme example, an epidemic of some kind, affects the—Canada as a whole, obviously we might require some action from the federal government, so that all the provinces are—are assisted?

A But—but are you talking about legislative action or action by the Department of Health coordinating some response among provincial Departments of Health?

Q At the moment, I'm talking about either, the combination?

A Because I can't think of an epidemic, for example, which would require national legislative action.

Q You can't think of one, that is to that extent that would require that kind of a thing, can you?

A I don't know of any instance, I suppose other than—unless under federal legislation the matters of quarantine for example, for people coming—

Q Tuberculosis, something like that. Let's say we had a massive outbreak of tuberculosis or—or have we always been able to deal with that locally in the province?

A That has been traditionally dealt with at local or provincial level.

Q Yes, and that was—that's a pretty significant—very serious risk or health risk type of problem, tuberculosis outbreak, isn't it?

A It was years ago. Then it ceased being. It is now becoming again a matter of concern.

Q So—but in your looking at the social policies that the governments adopt, whether it be local, provincial or federal and so on, you—you do look at it with a critical eye to see if it's doing more harm than good from the perspective of health and your patients, isn't that right?

A That'—that essentially is the argument we put forward, that one has to look at the harm and the good resulting from the medical problem itself and from the measures taken to—to deal with it.

Q Because you don't want the approach taken by government to exacerbate the problem?

A That's correct.

Q To make matters worse, fair enough?

A Yes, that's—yes.

Q To increase the harms that may occur?

A That's correct.

Q Or do we interfere in some way with your ability to, as a physician, to grapple with the problem, --

A Yes, that's correct.

Q -- from a health perspective, to try and help cure the patient or—or another example, to do all of the research that you'd like to do without being hindered by non-disclosure or inaccurate information or unreliable information because of the subject being in the underground economy or in some sort of subculture, fair enough?

A Yes, as a general principle I would agree with that.

Q So it would be a lot easier for you to help your patient or your prospective patients if you're able to get accurate information, open reliable information, in order to conduct your research to see what the full extent of the problem is?

A Yes, certainly.

Q So coming back then for a moment to marihuana or cannabis, you can't tell us what the total percentage of users are in Canada currently?

A No, unfortunately I can't.

Q You can't tell us what the percentage of chronic users would be, other than to say that you estimate it to be about five percent of whatever the total user rate is?

A Yes.

Q And—

A I should point out that's not my estimate. That's what I have understood from the—what I have heard and read from people who work in the epidemiology area.

Q Have you received any recent or current information from people in the epidemiological field, and I'm thinking in terms of input into the W.H.O., recent W.H.O. paper or—

A I looked at that last night to see if there was any concrete information, and I can—let me just turn to that. The most recent figures from—you want the Canadian figures?

Q That's what I'm thinking of.

A Unfortunately, there isn't—what is cited here is not very recent. "A national telephone survey was conducted in Canada in 1989 by Health and Welfare of eleven thousand, six hundred and thirty-four persons aged fifteen years and older. Overall, twenty-three percent of the sample reported that they had ever used cannabis, with higher rates among males than females across all age groups. Prevalence of use declined with age from a high of forty-three percent among those aged twenty to twenty-four years, to ten percent among those aged forty-five to fifty-four years, and two percent among those aged fifty-five to sixty-four years. Rates of discontinuation were substantial with only fourteen percent of those who had ever used cannabis having done so in the past year." That is the figure that I was thinking of when I said something around twelve. Fourteen percent presumably would be classed as—as current users.

Q Twelve to fourteen percent?

A Twelve to fourteen percent.

Q Of the total Canadian population?

A No, of the users, of those who had ever used. If—it says, "Only fourteen percent of those who had ever used cannabis had done so in the past year," so that would mean that that would be fourteen percent of—of twenty-three percent of the population. Twenty-three percent were—describe themselves as having ever used.

Q Yes.

A And of those, fourteen percent had done so in the past year.

Q But that only tells us about the occasional user or—or moderate user?

A Well, it's all—all users. It doesn't tell—that doesn't break it down into—

Q Statistics like this aren't my—

A -- the different levels.

Q -- strong suit, I'm afraid, but can you tell me what—from that, can you tell me what they estimate the total user population to be then in '89, I guess it is?

A That would be fourteen percent of—of twenty-three percent, that would be one-seventh roughly. That would be something in the range of three to four percent of the total population.

Q Three to four percent of the total Canadian population?

A Yes.

Q Okay. And so when we—we say the chronic user, we're talking about roughly five percent of that three to four percent of the total population, is that fair?

A That would—I think that would be it.

Q And so that—I don't know how quick you are at doing those things in your head, but could you give me a—or give us a figure in terms of numbers of chronic users?

A It would be less—it would be less than one percent of the population.

Q Less than one percent of the total Canadian population?

A Yes.

Q Okay. And so those are the—those are the people that have the significant health risks—

A Yes.

Q -- apart from the acute driving issue, fair enough?

A Yes.

Q Okay. And can we break that—

A May—sorry, may I just—

Q -- down—

A -- may I add one further point? That again, one must keep in mind the difference in levels of use at different—in different age groups, so that the figure would obviously be substantially higher among—as a percentage among the—those aged fourteen to say thirty—fifteen to thirty, because the levels of use were substantially higher among them, than among those aged over thirty.

Q All right. But that's when we start to break it down into the specific—

A Age groups.

Q -- high risk groups, if we can call them that, --

A That's right.

Q -- that we talked about?

A Yeah.

Q Just talking about the total—the total scope of the problem for the moment, we're talking less than one percent of the total Canadian population, total users and about five percent of that—

A No.

Q -- sorry, three to four percent of the total Canadian population, but less than one percent, was that the chronic user?

A Would be the heavy—chronic heavy users, yes.

Q Okay. And are we able to—or do we have access to government figures or—or Health and Welfare, Addiction Research, whatever, that tell us how that breaks down across the provinces in terms of percentages of population in the provinces?

A Yes. In—there is a publication put out by Health and Welfare jointly with the Addiction Research Foundation and the Canadian Centre on Substance Abuse called Profile Canada.

Q Yeah.

A The most recent one I know of is for 1994. There may be a more recent one, but I don't have a copy of it, but it gives national and province-by-province breakdown on all drugs, psychoactive drugs including cannabis, alcohol, tobacco and a variety of other things. It gives percen—or at least numbers of hospital admissions or hospital discharges associated with each of the drugs in each of the provinces. It gives a breakdown of—I believe on accidents attributed to them, on poisonings and so on. It's a comprehensive but not—not fine-grained analysis of the data for each of the provinces and the national figures, and it—for most of them it also gives them by—by—by sex and by—for some of them, by age groups.

Q And does it break down for us—I assume it gives us the total user population or estimated user population for each province, to start off with?

A I believe it does.

Q And does it give us the total chronic user, estimated chronic user figure for each province?

A I'm sorry, I—I can't recall whether it gives that breakdown or not. I just—

Q Because the—

A I just don't remember.

Q -- the significance of the health risk, if it's the chronic user, we—we'd want to know how many chronic users there are in each province in order to determine the scope of the problem—of the problem in each province?

A Yes.

Q But relative to tobacco and alcohol, we're talking about a pretty small percentage, aren't we, compared to the percentages we talked about in terms of tobacco and alcohol?

A Oh, without—without doubt.

Q Yeah.

A It's a small percentage compared to the—

Q I mean when we're talking about significant health risk, I take it you'd agree with me that tobacco particularly is a far greater health risk in terms of impact on Canadian society?

A Yes, I—I think there's no question that under present conditions, the problem posed by cannabis is much smaller than the problem posed by alcohol or tobacco. The concern is the ability or inability to predict how that comparison would be under altered conditions. In other words, if the use—if the level of use of cannabis were to increase to a point where it was comparable to the level of use of alcohol or tobacco, what—would we be in a position to estimate now what the health hazard might be then.

Q But that involves quite a large amount of speculation?

A Yes, it does. That's the difficulty.

Q And because so much of it has to do with demographics?

A It has to do with demographics, it has to do with attempts or bases of attempts to estimate how levels might change under different conditions, all of which is part of the problem of forecasting.

Q It's problematic, isn't it?

A Yes.

Q And as we went over, I think yesterday, we've had examples of different countries with different approaches and so on, and the rates of use don't necessarily seem to be affected by whatever the government's position is. Sometimes they are, sometimes they aren't?

A No, I wouldn't agree with that. I would say rather that they are affected not only by what governments do, but by important other considerations such as cultural traditional practices and beliefs and attitudes, and so on, so that one cannot account for all of the differences between societies, purely on the basis of what their legal approach has been.

Q But when it comes to marihuana use, we know that notwithstanding the war on drugs in the United States for example, that during that whole—or during the continuing period in which that approach has been taken, that that has not resulted in a decrease in use, has it?

A I don't believe we're in a position to say that, because there was a period in which there was a decrease. Now in the last few years, there's a period in which there's again an increase in some level—at least in some segments of society.

Q The adolescent?

A What we can't say is how much of the decrease was due to the law, how much was due to changed attitudes, beliefs, knowledge and so on, and I think given the fact that the law did not change during that time, we would have to conclude that the changes were due to factors other than the law.

Q Well, if you could have Exhibit 6, you—I think we've talked about the high school survey of the National Institute on Drug Abuse yesterday, you're familiar with that?

A The high school survey of the—you mean the Johnson -- or Johnston—

Q It may be Johnson, I'm not completely sure of the—all I have is a reference to preliminary estimates from the 1993 National Survey on Drug Abuse, --

A Which—

Q -- Department of Health and Service.

A Sorry, which page is that on?

Q Sorry, the—it's the first claim, so it's page two?

A Page two, right.

Q Yeah.

A Okay. And you're referring to—

Q Do you agree with this? It says, "According to government surveys of the general population, marihuana use began decreasing in 1980 after more than a decade of steady increase." Do you agree with that?

A Yes.

Q "By 1990, the downward trend shows signs of slowing, but use rates remained substantially lower than those recorded in the '70's,"—

A That's correct.

Q -- you'd agree with that?

A Yes.

Q And then it goes on to deal with an example of people of different age groups and it says, "Among twelve to seventeen year olds, past year marihuana use was about eight percent in 1992, compared to twenty-four point one percent in 1979?"

A Yes.

Q A substantial decrease, isn't it?

A That's correct.

Q "Among eighteen to twenty-five year olds, past year use is twenty-three percent in 1992 compared to forty-six point nine percent in 1979?"

A That's right.

Q And then it refers to, "A separate survey of high school students showing similar trends with use rates in

the 1990's being well below those reported in the 1970's," doesn't it?

A Yes. The first figures I believe are from the National Household Survey, --

Q Yes.

A -- and the—the high school figures are from the Johnston surveys.

Q And then it says, "However, after reaching an all-time low in 1992, they increased slightly during the next two years," and it sets out a table showing that first high rates in '76, going up to 1980 and then starting to come off until 1990, and then it—in 1992, in fact, and then this increased to 1994?

A Right.

Q And it then points out that this is a—a N.I.D.A. survey to determine non-pathological drug use, correct?

A Yes. It measures all use.

Q And then it says—this document says, "Adolescence is a time of experimentation with drug use, as well as other activities. Most adolescent drug users do not go on to become drug abusers. Indeed most adolescent drug users after a few years of experimentation cease using illegal drugs altogether." I take it you agree with that?

A To the best of my knowledge, that's true.

Q And then it says, "We will probably never know why marihuana use rates go up and down over time. However, it's worth noting that the recent increase occurred among the same population of young people who had been exposed to a decade long of anti-marihuana campaign in the schools and the media, that campaign based on exaggerations of marihuana's harms, and a 'Just Say No' ideology has clearly failed." I take it you'd agree with that, as well?

A To the—yes, I think that—that is true. The—the one concern I have about those figures is that these are what are called cross-sectional data. In other words, they take the final year of high school—they're high school seniors—and it's a different group of people that are being surveyed every time. Therefore, the fact that in 1994, thirty-eight percent as opposed to thirty-two

point six percent in 1992 said they had ever used, suggests that either that increase from thirty two to six—thirty-two point six to thirty-eight point two meant new users who had begun during that time, or that the increase had begun some time back, and one was now seeing a class with a larger percentage who had ever used at some time, and that would make it a little difficult to know what the factors were that increased the—the percentage of ever users in that group, because it—if it meant an increase in current year use, then that would be a more serious problem than if it meant that sometime back they had used, but were still not currently using, and therefore I—I couldn't hazard an interpretation of what that increase means.

Q Okay. But I guess what I'm coming to, Doctor, is that we don't seem to have a very good handle on just what the scope of the marihuana consuming problem may be, because we don't have clear information about total number of users and how many of them are chronic users, do we?

A Well, as I said before, I—I really don't feel that I'm in any sense the best qualified person to answer that, because I would have to say I don't have the—the most accurate, most recent figures, but figures can be obtained from the people who do these surveys, as to what the most recent levels of use are, at least in defined groups of the population, and I just don't feel competent to answer that.

Q All right. But we do know that in relation to tobacco again, that it kills some thirty-three to forty thousand people a year in Canada, don't we?

A Yes. No, I don't question your statement that the identified problem is very much greater with tobacco and with alcohol.

Q And we know that again, not just from research and studies, but from people going in and telling their doctors that they smoked so much per day and—and that they're having lung problems or whatever, and then ending up contracting cancer, emphysema, whatever it might be, and ending up in hospitals and eventually dying, correct?

A Well, those are the sources from which the public health data come, yes.

Q Yeah. And in terms of alcohol, I understand a recent figure I—I heard was something like it kills about seven

thousand Canadians per year. Does that sound about right to you?

A That sounds reasonable.

Q And again, people go in and see their doctors, get advice, contract diseases, go to hospital and that's how we again determine these public health figures, fair enough?

A Fair enough.

Q And in terms of all other illicit drugs except marihuana, I understand there's about eight hundred deaths per year, cocaine, heroin, these types of things. Do you agree with that?

A That again, I feel not qualified to answer because the types of—when you talk of deaths, they are of many different causes, some of—they can be called drug related for quite different reasons.

Q All right.

A For example, we did a search of the records of the Chief Coroner for Ontario some years back to look for causes of death among amphetamine users and over half of them were violent deaths. Now, you wouldn't call that a death caused by medical hazards related to amphetamine use. On the other hand, you can argue that they are because where violence resulted from paranoid delusions or from over-aggressiveness stimulated by central stimulant drugs, such as amphetamine, then you could argue that that is indeed a—a medical complication, so depending on how you choose to classify them, you might arrive at factors differing by a rate of—of a hundred percent or fifty percent if you're going downwards.

Q So what you're saying is if we take that eight hundred a year number for deaths from all illicit drugs, some of them may be caused directly by consumption of the drug?

A Yes.

Q Provided it's the type of drug that one can overdose on and kill oneself on?

A Yes, exactly.

Q Which is, as I understand it, drugs that have receptors in the brain stem primarily, is that right?

A No, it doesn't need to be receptors, as long as there's an action of some type on the—

Q That impacts the brain?

A -- the—that affects respiratory or cardiac regulation in the brain.

Q That causes a shutdown?

A Yes.

Q So it may be that of that eight hundred, a substantial number of those are caused that way, but a substantial number are caused by what you term drug-related causes?

A That's right.

Q Okay. So it's not—I guess the way to put it is not from the toxicity of the drug, but some other cause that's related to the drug?

A From—related to the drug through behavioural consequences.

Q And in some cases, these deaths can occur as a result of the social policy approach of the government. It might create a prohibition and people shooting each other and so on, and black markets and so on. You're familiar with that, aren't you?

MR. DOHM: Your Honour, that is a very long bow for my learned friend to draw, with respect, especially in light of the qualifications of the witness.

MR. CONROY: Well, my friend put his articles in front of the Court. It seems to me I'm entitled to ask him about his—what he's written about and what my friend has put in front of the Court.

Q You—you talk about this in your paper, don't you, Doctor, about some of the causes of deaths from—in the drug field because of prohibition, is that right?

A Yes, that's true.

Q And that's a concern for a physician, isn't it, that people are dying from whatever the cause, isn't that right?

A Well, it's—I think it's a concern for all citizens including physicians.

Q Yes. But when you're a physician and your primary concern is health, and that you want your patients to get better, you want to make sure that whatever he's done results in them getting better, and not dying ultimately, --

A Yes.

Q -- fair enough?

A Fair enough.

Q Okay. But when it comes to marihuana, we don't have any deaths, any known deaths, do we?

A That's correct.

Q Anywhere in the world, isn't that right?

A Yes, I think that's—that is the accepted view now, that—

Q So—

A -- there are no—no deaths which can be reliably attributed to cannabis.

Q We don't have—we know that tobacco kills, marihuana doesn't, isn't that right?

A Well, I have to qualify the answer I just gave, because I'm—your question I took to be referring to deaths due—directly due to toxicity that is acute toxicity overdose, and there are no deaths of that kind.

Q From tobacco?

A No, from cannabis.

Q All right.

A Now, if you talk of tobacco-related deaths, you're not talking about acute toxicity. You're talking about chronic

toxicity, and there I don't think we are in a position to say that there is no—there are no deaths of that type due to cannabis.

Q You—you think there—you think there might be, but there's none documented, are there?

A That's right.

Q Yeah.

A Well, there are—other than the few case reports which I described, and the demonstrated actions which by analogy with tobacco are grounds for anticipating that there may well be such, and when there are enough public health statistics accumulated.

Q That's—that's what it comes down to, isn't it?

A Yeah.

Q You—you see some things and you think there might be some problem in the future, but we don't have the actual documented deaths or—from marihuana use, whether toxic causes or any other causes at this time, do we?

A No. Again, I cannot agree with that, because if one is talking about traffic deaths, for example, there are as I indicated in previous—in the preceding testimony, there are data which are strongly suggestive of a role of cannabis in traffic accidents. If this is validated by future work, then would—one would have to say yes, there are deaths attributable to cannabis.

In the same way, if the pulmonary changes prove to give rise to things such as respiratory system cancers, then one would have to say again, yes, there will be—one can expect deaths attributable to long term toxicity.

Q Yeah, if those things pan out, --

A Yes.

Q -- then we might see them, that's what you're saying?

A That's right.

Q But we don't see any yet, do we?

A We have no proof of that now, that's right.

Q We do see that for tobacco, don’t we?

A Yes.

Q We do see that for alcohol?

A Yes.

Q We do see that for other illicit drugs?

A Right.

Q We do see it from other causes?

A Yeah.

Q Food, different types of foods and things, right?

A Yes. Right.

Q But we don’t see it for marihuana yet, do we?

A No, we don’t have the same standard of—the same level of proof.

Q Okay. But we don’t have any—we don’t have people going in and seeing their doctors, and saying, "I’ve got this type of a problem from using marihuana,"—

A Well, no, --

Q -- and ending up in a hospital—

A -- no, no, I’m sorry—

Q -- and dying, do we?

A No, I’m sorry, I can’t agree with that.

Q Well, do we?

A We have people who go to doctors complaining of respiratory symptoms.

Q And then going into hospital and dying?

A No, that we don’t.

Q All right. No, we don’t have it?

A But on the other hand, we have the published case reports of upper airways cancer which—

Q I'll come to those. I'll come to those. At the moment I'm talking about deaths. We don't have any of those, do we?

A Well, other than those, because those were deaths.

Q The upper esophagus cancer?

A Yeah, there have—there have been some deaths.

Q In the teenagers?

A Yes, there were a few.

Q Okay.

A It's a small number, but the point is—

Q All right.

A -- that one can't say that there are none or that there is no—that there is no evidence that that may occur.

Q But we don't have the kinds of figures that—

A No.

Q -- we have for these other drugs?

A No, we don't.

Q And we don't have the kinds of numbers of people going in to their doctors saying they've got a health concern and then being in—ending up being put in hospital and then dying from marihuana consumption, as we do with these other drugs?

A No, true. We don't.

Q All right. And marihuana became illegal in Canada, as I understand it, in 1923, did you know that?

A Yes.

Q And so we've had a period what of—what is that, seventy-three years, --

A Seventy-three years, yes.

Q -- then that it's been illegal, correct? And prior to that, we didn't have people dying from marihuana use, did we?

A There are two difficulties with answering that question. One is that cannabis as used prior to 1923, the long—the longest part of the preceding period in which cannabis was used, it was not smoked. It was used medicinally as a tincture of cannabis, an alcoholic solution of cannabis that was prescribed and taken by mouth.

The smoking of cannabis was of relatively short duration before the—before it was made illegal. It was really a phenomenon that began mainly around the beginning of—of this—this century, so that there would have been a little over twenty years of time in which it was smoked or which—in which the major use was by smoking rather than by an orally taken—taken by mouth medicinal solution, and the other problem is that the sorts of public health statistics that we're talking about now, by and large were not collected in those days, so that we—I don't think we can use the period prior to 1923, to say either whether there was or wasn't a significant problem.

Q We can do it for alcohol, can't we?

A We can for alcohol because much better records were kept of alcohol use, for taxation purposes, and also alcoholic cirrhosis was recognized well—well, I suppose about two centuries ago, so that there's a much longer period of clinical experience with it.

Q But marihuana's been around for centuries, too, hasn't it?

A Not in our society.

Q No, but in society generally, in the world, --

A No, but in societies—

Q -- in different societies?

A -- by and large in which medical care was very limited and which medical science was not very good, so that I don't think we can use that experience to draw any conclusions about hazards.

Q So notwithstanding long-term and I'm talking more than seventy-three years use in Africa, or Jamaica, or Malaysia, and the fact that we don't have people going

in throughout this whole time to their doctors or physicians or healers or whatever, and apparently dying from the use of this substance, you say that doesn't tell us anything?

A Well, no. I think your premise is one that can't be accepted. We can't say that there weren't people dying of it because we just don't know.

Q All right. So nothing though has manifested itself in terms of the way it has for other drugs, so that we could see that there's a significant cause of death?

A Let me try to point out a problem in a way that is more meaningful. The traditional use in north Africa, for example, if one looks at the writings of physicians from north Africa, from Egypt or Morocco, or other areas where traditional use has existed for centuries, some are very strongly convinced that it is a serious problem of public health, in the sense that they attribute to cannabis the same effects that alcohol has in our skid row populations and they—they believe that malnutrition, infection, suppression of immunity, effects on motivation and therefore on work and income and therefore on standard of life, are attributable to heavy cannabis use. The problem, as I say, is that their statistics are not simply there to permit one independently to assess whether that's true or not.

If I might add one point, yesterday you asked about the composition of the committee and I had difficulty remembering. I did recall that there—now, last night, that there was someone from north Africa. There was a Moroccan psychiatrist who—who was also trained in Spain and therefore who did represent at least that part of the world in which—in which traditional use exists.

But to come back to the question, because of the fact that there simply is not, and has not been, the same level of medical care, the same level of public health statistic gathering, the same level of medical science in the areas of the world where use has been traditional, we simply cannot say whether that—their experience does or doesn't illuminate the—the picture of medical consequences.

THE COURT: If I understand what you're saying correctly, back in 1923, we were in no position to say this drug is killing us, or otherwise destroying the lives of people who use it, not because it wasn't in fact doing so, but because we didn't have the data—

A We just didn't have the data.

THE COURT: -- to—to make that statement?

A There—there—yes. There—there are two—two factors that I think we need to remember. One is that the law which was passed in 1923 was not, in fact, in response to what was perceived as a large—statistically a large problem of use of cannabis in Canada. The evidence, such as it is, doesn't suggest there was widespread use, and secondly, any information about its consequences was not being gathered in a systematic way, so the—whatever the reasons were for passing the law, I don't think we can say they rested on a public health basis.

THE COURT: It's eleven o'clock.

MR. CONROY:

Q That was because of the Americans, wasn't it?

A I can only—I can only—

Q Like many things in Canada?

A I can only conjecture that that was the case. I don't know.

MR. CONROY: This is a convenient time.

THE COURT: All right. We'll take the morning break at this time. Thank you.

(WITNESS STOOD DOWN)

(PROCEEDINGS ADJOURNED)

(PROCEEDINGS RECONVENED)

HAROLD KALANT, recalled, testifies as follows:

CROSS EXAMINATION BY MR. CONROY continuing:

Q I'd just like to carry on in that area that we were at. I think you told us that in 1923 when it became illegal, there wasn't any particular public health problem that we were aware of, and suddenly it became illegal or was made illegal. From my readings, and I'm sure you've probably read many of the same things, there were a number of factors that led to it suddenly becoming illegal. In fact, we—we didn't know what -- what suddenly led to it becoming illegal other than somebody had suddenly added it to the schedule, I think, is the—the stuff I read?

A I—I think that's probably a fair description. It was—all I—all I am qualified to say is that to the best of my knowledge, there was no recognized public health problem that was the—

Q One of the—sorry.

A -- basis of the decision.

Q One of the stories that I read, and I don't know how true it is, is that there was anecdotal evidence of problems in marginal groups in the United States, and that the police chiefs spoke to our—our first woman judge as a matter of fact, Emily Murphy, who wrote something called The Black Candle which then was picked up and serialized in MacLeans magazine, and so on, and that may have been one of the factors. You've heard that story?

A Yes, I have.

Q Okay. In any event, at that time we certainly knew that there were these other large communities in Africa, Jamaica, some of the other countries we've mentioned, Malaysia, where they'd been using the stuff for long periods of time historically and culturally, fair enough?

A Yes.

Q And I think you've told us—I think you said this in chief—it wasn't until the '60's that suddenly there was this focus on it and increased use and therefore government funding to determine what the risks were, --

A Yes, I think that's quite right.

Q -- fair enough?

A The—there was a widespread public concern then about the rapid increase in the—in the use of cannabis in North America, both in Canada and the United States, and that led to government responses which included, among other things, the provision of substantially more money to fund research into the effects.

Q Right, but when that started, we didn't have a significant public health problem. The—the concern was to determine whether we were going to have one?

A I think that's correct, yes.

Q Yes. I mean it wasn't—it was a youth rebellion type of thing that was attributed to the sudden interest in this drug rather than anything else as I recall, would that be fair?

A Or the other way, that the use of the drug was attributed to a youth rebellion.

Q Yeah. Okay. Not that it was caused—

A That's right.

Q All right. In any event, so since—what would we put it, about 1966, or would you put it sooner than that that this started?

A No, I—

Q Mid-'60's?

A Late '60's and early '70's.

Q Okay. The baby boomer generation is considered that group of people who were born in and around 1947, 1948 and until about 1966, isn't that right?

A Yes, I think that's—

Q This is this massive cohort of people that have come on the scene in North America, that is a huge demographic factor that's affected all kinds of things in our society, isn't that right?

A Yeah.

Q It was that particular group that was associated with this sudden interest and increase in use throughout that period, fair enough?

A That seems a reasonable statement, yes.

Q Yeah. And—and we—we know that after that group came a much smaller cohort of people, and I think they call them the baby busters, is that right?

A I'm not familiar with that term.

Q Okay. And after that they say there's a slightly larger demographic group coming up, what they call the echo generation, have you heard that one?

A I've seen it on the title of the book that you're—

Q All right. This sort of thing, large cohorts of people, particularly young people coming up, can affect these types of things we've been talking about in terms of rates of use and things like that, can't they?

A Yes.

Q All right. Now, when the problem started to—not the problem but when the use started and the focus then came about in the mid to late '60's and the funding started, since that time, we still haven't seen a significant number of—well, first of all, we haven't seen again any deaths that can be attributed directly to marihuana toxicity or other related concerns, health concerns?

A That's correct.

Q Nor have we seen any large—well, it's not—we have no evidence that it's a leading cause of illness since that time either, do we?

A No, I would agree with that statement that there—that we do not have the evidence.

Q Right, we don't have any evidence—

A We can't say—we can't say that there is or isn't.

Q Yeah. We don't have the same kinds of public health reports or statistics that we have for alcohol and tobacco of—of people going in to their doctors and to hospitals or health clinics or whatever, amassing a major number of statistics to indicate that it's a leading cause of disease for example in Canada, do we?

A No, we don't.

Q Okay. And for all of these—let's just use maybe tobacco and—and cannabis as the example. When the -- when the person comes in as a patient to a doctor, and talks about having a health complaint—let's use bronchitis as an example—the doctor usually treats the patient or comes up with certain recommendations for the patient in order to try and solve the health problem, isn't that right?

A Yes.

Q That's the usual method, isn't it?

A Yes.

Q Okay. And we've been doing that in relation to cannabis as well as the other drugs, for even well before the 1960's, isn't that correct?

A We have—or physicians have been treating the complaints that patients come with, although I must say to my professional regret, that physicians have not been particularly good until quite recently, in asking patients about the use of substances that might contribute to any problems, even alcohol. The extent of physicians' detailed inquiries of their patients about the frequency and amount of use of alcohol has not been very good, --

Q Yes.

A -- and the—their inquiries about the use of other substances that may contribute to health problems is probably even poorer.

Q Yes. Okay. But in recent years, we've been more successful in educating people and getting them to reduce both alcohol and tobacco, haven't we?

A Yes. There—there has been an improvement.

Q But—but as you point out, notwithstanding that, alcohol and tobacco anyway continue to be the—the leading drug problems in our society, aren't they—

A Yes.

Q -- in terms of harm to health? Okay. Now, you've told us that you were with the Addiction Research Foundation, still are as Professor Emeritus of the Addiction—

A Or Director Emeritus.

Q -- Director Emeritus, sorry, of the Addiction Research Foundation. And you were with them for many, many years when a lot of the studies and so on that you've told us about were going on?

A Yes.

Q In the—in recent years, in the last two or three years, the focus of the foundation has shifted to a harm reduction focus, hasn't it?

A Yes, that’s true.

Q And the harm reduction focus, as I understand it, and you correct me if I’m—I’m wrong on this, is a shift by physicians—health care people, looking at drugs and saying "Well, our first priority has to be reducing the harm caused by the substance or the social policies affecting it?"

A Yes, the—the essence of the harm reduction approach is to concentrate on reducing the harm caused by the use of a drug, rather than simply concentrating on reducing the use itself.

Q Yes. And being concerned about the social policy and how that contributes to the problem or to the—

A Yes, as—

Q -- the harm?

A -- as one of the important elements in the equation.

Q Okay. And that’s because of a recognition by people in the field of some of the problems caused by the existing approach, existing social policy, isn’t that correct?

A Yes, that’s correct.

Q Okay. So that the—there’s a balancing that goes on in the process of looking at the harms from the social policy and the harms from the drug, and trying to look at each individual drug separately in order to come up with an approach that hopefully reduces the harm from all sources?

A Yes, that’s the—that’s the effect.

Q Fair enough?

A Yes.

Q Okay. Oh, incidentally, these—the cross-sectional studies we talked about a bit from Exhibit 6, all of the Addiction Research Foundation cross-section—or studies on usage rates and so on, are cross-sectional as well?

A Yes, they are.

Q Yeah. And it’s very difficult to test for reliability, isn’t it? I mean there’s a telephone call and somebody says something, it’s hard to—to check that out, self-report?

A Yes, that is—that is one of the concerns that is always attached to survey work of any kind, but there -- there was—and again, I’m not an expert in this area—I can only indicate what my understanding is, that the people who have done the survey work have, in fact, attempted to validate answers by cross checking with answers from friends or associates. In other words, someone may be reluctant to say, "I use," but are quite willing to say, "My friend uses," and that type of cross-checking and eliciting information in different ways, by phrasing questions in different ways, suggests that on the whole, the answers given by—in the student surveys and probably in the adult surveys, are generally pretty reliable.

Q The—the more—or the different survey, if I can call it that, that you referred to, is the longitudinal survey, is it?

A The preferred one, if one is attempting to see over the passage of time what particular behaviour—what results particular behaviour produces, the ideal would be longitudinal studies. They are obviously more difficult to do. They require much more investment of personnel and resources, they’re expensive, and keeping subjects in the study over a long period of time is, in itself, one of the problems. You have to track down dropouts, see why they dropped out, whether they dropped out because of some event that is precisely the kind of thing you’re trying to look for, so that they pose problems, but when they can be successfully done, they are unquestionably the best method.

Q Okay. And just to be clear on the differences, the cross-sectional survey, you don’t know who the people are, you—you get a range of people generally, whereas the longitudinal, you’ve got specific individuals that you’re following—

A Over time.

Q -- over a period of time?

A That’s right.

Q All right. Let’s carry on with—and head on to another part of your evidence yesterday. I’m going to come back to the—you talked at length about the lung factor, and I

take it you'd agree that this—the Tashkin Report that you got to read last night is a longitudinal study?

A Yes, that's a—that's a longitudinal study.

Q And so it's probably the best one we have in terms of length of time of following people over—over the period that are cannabis users, isn't it?

A Yes. He followed them for I recall something like eight years and that was—that's probably, if I recall correctly, it's longer than the other major longitudinal study that was done by—

Q Longer than any other one so far, isn't it?

A Yes. Yeah.

Q And so again, a case of him taking individuals who were cannabis users, with different rates of us and so on, and following them over this eight year period and seeing what it was doing to them in the beginning, and see what ended up at the end so far?

A That's right.

Q Okay. And if I can just put it in a nutshell, and I'm going to come back and deal with it in a bit more detail, essentially, as I understand it, he did find—or he didn't find any emphysema results in the cannabis smokers, that other one—one thought might be there from the earlier studies?

A Including his own earlier study.

Q Yes.

A Yeah. Yes.

Q He found the bronchitis a factor?

A He found bronchitis, but he did not find the acceleration of impaired airway flow or resistance or volume that he found in tobacco users, and that he was looking to see whether he could find in the chronic cannabis users.

Q The—when you gave evidence in Hamon in 1991 in Quebec, at that time the—your view based on the research that was available at that time, was that cannabis smoke did affect the smaller—

A Airways.

Q -- airways and the exchange of gasses across the lungs?

A That's correct. And one of the—one of the pieces of evidence that I relied on for that was his earlier study.

Q And since then, we've got this new information that's changed the situation, changed the state of our knowledge I guess I should say, about that to some extent?

A May I comment on this paper?

Q Certainly.

A There are a number of problems with it, and unfortunately one of the difficulties is that the—I don't fault you for this, but the—what you faxed me did not include the tables or figures with the actual data.

Q Yes, I'm afraid it was faxed to me from elsewhere and I didn't get them either. You got everything I got.

A Yeah. And in the absence of the actual data, it's rather difficult to follow how the—

Q To read it?

A -- the reasoning. In part, that's because what you sent me is pre-publication proofs. These are proofs that were sent from the journal to the author to correct, and there are corrections made on them, and other markings, but the paper itself hasn't yet appeared in publication. I was relieved to see that, because what—I was asked yesterday whether I was familiar with it and I said I was not. I was troubled because I had done a computer search of the literature just a few days before coming and had not seen this, and the reason I'm relieved to say is simply that it hasn't actually been published yet, but it has passed peer review, so I presume this is the form in which it will eventually appear.

Q I'm afraid I can't tell you that.

A Well, I—I would think so because if it—when it gets to the stage of proofs, it has already passed peer review.

Q Okay.

A But one—one of the very interesting parts of the discussion is that Tashkin himself or recog—or the authors of the paper—I believe Tashkin is the first author—did point out that their findings are in disagreement with their own previous findings, and also with the findings of the—the group that I mentioned yesterday, Bloom et al, who did a similar longitudinal study of chronic cannabis smokers, chronic tobacco smokers, combined cannabis and tobacco smokers, and non-smokers, and found that there was a significant reduction of volume flow of airway obstruction—an increase of airway obstruction in the cannabis users that was greater than that found in the tobacco-only smokers, and that was combined with the tobacco effect because it was greatest in the combined cannabis and tobacco smokers.

And most of the discussion in this paper is devoted to a—a consideration of what might account for the difference between these findings and the earlier ones, and he comes up really with a conclusion that they're not yet in a position to—to say, but one of the things that he considers as a possible explanation is that the population of subjects used in this study was not really representative of the whole population of cannabis users, because they were recruited in a different way, whereas the—the Bloom et al did—did have a randomly-selected population which in this paper, Dr. Tashkin says is probably more representative of the user population in Bloom's area than his subject population is of the user population in Los Angeles.

So that—that makes it difficult to—to say that this is more conclusive than the other in any way. We simply—he feels he's not yet in a position to say, which is the—what is the real explanation.

Q But the people that he chose or that were selected were people who had been smoking marihuana for fifteen years prior—

A Yes.

Q -- to starting this eight year monitoring, if I can call it that?

A That's correct.

Q So we're talking about people who'd been smoking marihuana at the end—by the time of the end of the study for twenty-three years, aren't we?

A That's right.

Q Yes. And he found that—what they measure for, as I understand it, is something called forced—

A Expiratory volume.

Q -- expiratory volume in one second, the FEV1, is that right?

A That's correct.

Q And essentially, that measures outflow of breath not breathing in, but the breathe out in one second?

A As rapidly as you can. As rapid—as hard and as rapidly as you can.

Q Which gives us some indication of the—what's impacting the narrow or smaller vessels?

A That's correct.

Q Yes. And he found that there was no decline in that measurement for these cannabis smokers, isn't that correct?

A Not strictly speaking. What he measured was the decline which does occur with age in everybody.

Q Okay.

A What he found was that there was no acceleration of that decline in the—in the cannabis group. There was in the tobacco group, and he found no interaction between cannabis and tobacco, but if I can point out, one of the reasons why I have difficulty following the paper without the data, in his discussion he says for example—I'm quoting here, he says—this is what's marked as page twelve of the proof.

Q Yes.

A The bottom paragraph, the one that begins, "Table 4 shows..."—

Q Yes.

A -- partway—about halfway down, he says, "For example, the results for Figure 1 indicate that the reference group (non-smokers) had a 25.3 millilitres per year rate of decline, whereas marihuana smokers had a 35.8 millilitre per year rate of decline, or a difference of 5.5 millilitres per year shown in Table 4 from the reference group. Marihuana and tobacco smokers had a decline of 10.5 millilitres per year greater than did the

non-smokers, which is the sum of the marihuana and tobacco terms and their interaction."

Now, as I read that, that suggests that there was a decline—a greater decline in the marihuana and a still greater decline in the marihuana and tobacco, and that I—I don't know how to reconcile that with the final conclusion that there was no acceleration by cannabis and no interaction with tobacco, so all I can say is I'm—I'm really unable to interpret this paper.

Q If you had this—the diagrams and so on—

A The tables of data and the—

Q -- the tables, --

A -- diagrams, it would be easier to follow what he's saying.

Q It was my understanding, what I've been told and—about it is that he found no decline in this forced expiratory volume and that indicated that amongst the cannabis smokers, the anticipated evidence of emphysema and so on wasn't there in this study?

A Yes, that is what he says in the summary and what he says in his—in his discussion, but I can't—I don't know how to reconcile that with what he says in the description of results.

Q Okay. But he found in the tobacco smokers a yearly decline, didn't he, in this—

A Well, he did—he did in all subjects. What he found was a faster decline in the tobacco subjects than in the non-smokers.

Q And also episodes of dips—dyspnea (phonetic) or breathlessness in the tobacco smokers that he didn't find in the cannabis smokers, isn't that correct?

A Where do—can you refer me to a—

Q I can't I'm afraid. That's just what I'm told because I find it difficult to read as well.

A Yeah. I didn't see that in reading this paper. I did look up some other very recent work of the Tashkin Group. I—I have a set of abstracts of papers that were presented at a 1996 conference of the Cannabinoid Research Society, --

Q Yes.

A -- and may I refer to those, is that permissible?

Q Certainly.

THE COURT: Yes, go ahead.

A There were three abstracts by the Tashkin Group in that set of papers.

MR. CONROY:

Q And this is at what time, sorry?

A This is June of 1996.

Q When—during the committee meetings for the W.H.O. Committee?

A No, no, this is a meeting of what is called the International Cannabinoid Research Society.

Q Okay.

A It was held in Massachusettes in June of '96 and he—they have three papers, one dealing with the effect of the habitual use of marihuana on anti-bacterial and tumorstatic activity of alveliar (phonetic) macrophases (phonetic), the inflammatory cells of the—in the lung, and his conclusion was that—they—they measured this by doing bronchial lavage, that is passing a bronchoscope, washing out an area of lung in groups of non-smokers, tobacco smokers, marihuana smokers and—and combined smokers, and then testing the cells for their ability to phagosatoge (phonetic), essentially to gobble up and—and destroy bacteria. And he looked specifically for their ability to do that with staphylococcus which is a—one of the more important pathogenic bacteria that can produce a pneumonia or a severe bronchitis.

And also he looked at the ability to—to activate the—what are called protoanchogenes (phonetic). They're—they are genes which are believed to play a role in the activation of tumor growths and his conclusion was that habitual marihuana use is associated with substantial defects in the functional activity of the lungs resident in munifector (phonetic) cells, (pulmonary alveliar macrophases), in defending the lung against both bacterial infection and tumor cell growth.

The second one had to do with the deposition of tar in the lung and the bio-availability of Delta 9THC. Well, I won't cite—it's because this deals with a

topic that we haven't discussed, about the question of ability to titrate one's dose.

But the third one is specifically relevant to the question of bronchitis sputum formation, things of that kind, and here again this was—these were groups of non-smokers, tobacco smokers, marihuana smokers, and combined tobacco and marihuana smokers, all of whom had been chronic users and he expresses the—I think the views in terms of pack years for cigarettes—tobacco cigarettes, which is the accepted way of giving total exposure. You multiply the average number of packs by the number of years—that is number of packs a day by the number of years in which that extent of smoking has been going on, and for the marihuana users, he expressed it as joint years, which is similarly joints per day times number of years at that rate of smoking.

And again, each subject underwent bronchoscopy and mucosal injury was evaluated by three techniques: visually using a bronchitis index to measure erythema, that's the reddening; edema, that's the swelling of the tissue, and secretions on a one to three plus scale; pathologically by taking biopsies from the bronchial mucosa and sectioning them, staining them histologically and looking for inflammatory and other changes in the tissue; and third of the molecular level studying the—the protoanchogenes that I referred to in the—in the other abstract.

The results showed that erythema, that is reddening of the mucosa, was common in both non-smokers and smokers, but the mucosal swelling occurred primarily in smokers; for example, sixty percent in tobacco smokers and marihuana smokers alone, and eighty-six percent in combined marihuana and tobacco smokers, versus only twenty percent in non-smokers.

While airway secretions, that is the accumulation of mucous, of phlegm, which produce—contributes to the obstruction, was—airway secretions were not frequent, they occurred only in smokers. At the pathologic level, vascular proliferation was present only in smokers forty to seventy-five percent of all three smoking groups, and none in the non-smokers, suggesting that reddening in the pathologic—in the smoking groups, was due to pathological causes while that in the non-smokers was merely related to the irritation of passing the bronchoscope.

Similarly, mucosal atypia, that means changes in the microscopic features of the individual cells seen in the microscopical sections, was determined by looking for basal cell hyperplasia, that is overgrowth of the basal cells at the bottom of the mucosa; stratification, that is a piling up and layering of cells which should normally be only in a single layer; and cellular disorganization, increased nuclear cytoplasmic ratio, which is indicative of overgrowth of the nuclei; mitosis, that is cell replication activity which should be at a—normally at a low level in—in healthy people; and squimus metaplasia (phonetic) that is change in the character of the cells from mucous-producing cells to cells like the non-mucous producing skin. These changes are of interest because they're believed to be pre-cancerous changes.

And what he found is that these findings were much more prevalent in smokers than in non-smokers, and they were more frequent in the—they were more frequent in—in marihuana smokers than in tobacco smokers, seventy-five percent versus forty percent, and present in almost all of the combined marihuana/tobacco smokers, sixty-seven to a hundred percent for the different changes that were looked for.

Q And this is all part of his ongoing longitudinal study, isn't it?

A This is all part of this, that's right. And his conclusions were that, "Significant mucosal injury occurs in the lungs of young marihuana smokers and tobacco smokers, including inflammation, atypia and activation of protoanchogenes that are felt to be involved in lung carcinogenesis. The injury from marihuana smoking is equal to or worse than that observed from tobacco smoke, and the effects appear additive." So these are not reflected in the paper which you sent me, but they are part of their continuing work.

Q The paper that I sent you is apparently later in time to those ones that you have there?

A No, I would think it's the other way around. The ones that are—that I'm quoting here are abstracts of presentations at the meeting and in 1996 they probably will not reach publication until sometime in '97.

Q Okay.

A This is work that probably was done a little earlier and therefore submitted earlier for publication and is now in press.

Q Because this—the one that I referred you to is about to be published?

A It's about to be published, that's right.

Q It's gone through the peer review process?

A That's right, and these—

Q And those haven't?

A These have not.

Q Okay. So—all right. Well, let's just go back to the one that I gave you for a moment, and go back to that page

that I think you referred to, which is page -- well, let's give—

A Twelve I think it was marked in handwriting.

Q I have copies of these, and I should have—

THE COURT: Are they filed? Is it filed as an Exhibit yet?

MR. CONROY: I don't think it is, so I think we—it's referred to so extensively that we should do that.

THE COURT: I think the abstracts that Dr. Kalant has referred to should also be—

MR. CONROY: Yes.

THE COURT: -- filed. I don't know if you have extra copies.

MR. CONROY: Maybe—we may have to have some extra copies made of that.

A I don't, unfortunately. Would it be possible to—

THE COURT: All right. Well, we won't take those off your hands right now. During the lunch break, perhaps the Crown can make some copies and—

MR. CONROY: I've got two, Your Honour. One can be an Exhibit and the other one can be one for you to mark up.

THE COURT: Thank you.

THE CLERK: It would be Exhibit 31, Your Honour.

EXHIBIT 31 - EXCERPT OF REPORT

MR. CONROY:

Q You've got one of the Tashkin ones, don't you, Doctor?

A Yes. That's the one that was faxed to me last night.

Q Let me just very quickly before the lunch break take you to—I'm sorry, that was Exhibit—

THE CLERK: 31.

MR. CONROY:

Q -- I believe it’s the page that you read to me. It’s got footnote thirty in the bottom bracket. If—yours probably doesn’t have page numbers on it. I hope the others do in the top right corner, but they’re a little hard to read. It should be page twelve I believe.

A Page twelve.

Q Now, I don’t know if that’s going to help you. Let me count them for you—it would be the eighth page in that starts at the top, "A weakness of the present study ..." is the opening line.

A Yes, I have it.

Q Do you have that?

A Yes, I have it here.

Q Now, if we go down to the paragraph that says, "Our failure to find ..." the last full paragraph, --

A Yes.

Q -- you read to me from there, didn’t you?

A No, I read to you from the page that is marked in handwriting in the upper right-hand corner. It’s marked twelve.

Q Okay. That’s where my—

THE COURT: A whole bunch of them are marked twelve.

MR. DOHM: I think for the record that the—the handwriting—

MR. CONROY: Yeah, there—there’s a lot of—

MR. DOHM: -- shows pages one hundred and twenty through one hundred and thirty with the last digit being missing in many of them.

MR. CONROY: Oh, that’s what it is. Okay. That’s where the confusion comes in.

A Oh, it’s not twelve. It’s one hundred and twenty something.

Q All right. Well, go to the one that—that I've referred you to—

A Yes, sir.

Q -- because that's the part that I'd like to read to you. There he says, "Our failure to find evidence of progressive lung dysfunction in the continuing marihuana smokers who we followed contrasts with our own observations that the proportion of these smokers who reported symptoms of chronic bronchitis was comparable with that of the tobacco smokers in the same cohort." It then goes on, "And with that of the tobacco"—sorry, "And that many of the continuing marihuana smokers have shown as extensive histopathologic alterations on bronchial mucosal biopsies as the tobacco only smokers," right?

A That's correct.

Q But then he says, "However, these similarities between the effects of habitual smoking of marihuana and tobacco on chronic respiratory symptoms and proximal bronchial histopathology do not necessarily imply similar consequences with respect to bronchiolar and alveolar injury that might lead to smoking-related obstructive small airway disease and/or emphysema," correct?

A Yes, that's correct.

Q And then he says, "Although symptoms of chronic bronchitis are believed to be related histopathologically to hypertrophy of the submucosal bronchial mucous glands, alterations in silia—silviated bronchial"—

A Siliated.

Q Sorry?

A Siliated.

Q It's got an extra I in there. "Siliated bronchial epithelial cells and hyperplasia mucous-secreting goblet cells, these symptoms of mucous hypersecretion are not thought to be necessarily linked to the progressive damage to and narrowing of peripheral airways that accompany the evolution of smoking-related chronic obstructive airway disease," correct?

A Yes.

Q Now, if I understand that, he's saying that there's no evidence of progressive lung dysfunction in marihuana smokers, and he contrasts that—he says there were a similar number of tobacco smokers as marihuana smokers who reported symptoms of chronic bronchitis, that many of the continuing marihuana smokers have shown as extensive histopathological alterations on bronchial mucosal biopsies as—as the tobacco only smokers. Although the chronic bronchitis and its symptoms occur in marihuana smokers, these symptoms are not thought to be linked to the evolution of smoking-related chronic obstructive airway disease or emphysema. Is that another way of saying what he said?

A Yes, that's essentially what he's saying. The problem is that in his discussion, he devotes a good deal of time, I think very fairly and very—very appropriately, to the differences between his findings which showed no change in forced expiratory volume one, and those of the Bloom group, which did show forced—such changes—more marked in cannabis alone than in tobacco alone, and additive with the tobacco changes. And the problem is that he's unable to reach a conclusion as to what accounts for the differences.

Q Well, if you go to the next page, he then—and I'll try to just summarize that, he—he contrasts the effects possibly as a result of the disparity in the number of tobacco cigarettes and the number of marihuana cigarettes typically smoked by each type of smoker. That's what he's essentially saying in that first paragraph, correct?

A Mm-hm.

Q And he's saying the average marihuana smoker is 4.1 joints per day, which is awfully high, isn't it?

A Yes, that's high use. Yeah, those are heavy users.

Q And the tobacco cigarette—or yeah, the tobacco cigarettes is 27.5 cigarettes a day, isn't it? So I mean when we talk about—we've talked about heavy chronic users as one or more cigarettes a day, this would be four times—

A These are heavy. Yeah.

Q -- our base level for a chronic user, correct?

A Yes. These—these are certainly heavy users.

Q Now, there's another point on that page, and I just want to get it clear because I think this relates to what we talked about earlier. Is this the reference about the tar in the—in the smoke?

A Mm-hm.

Q Because as I understood this part, he isn't saying that the tar in marihuana smoke is four times greater than in tobacco smoke, but he's saying because of the way you take it in, that—the marihuana smoke that is—and other factors which I'll—I'll list for you, you get more tar when you smoke the marihuana cigarette than you would from the tobacco cigarette. Do you understand what I'm saying?

A Yes, I understand what you're saying.

Q Because as I understand it, he's saying the higher tar in the marihuana joint as opposed to in a cigarette is: (1) because the tobacco is more tightly packed; (2) because of the filters that you mentioned; (3) because the marihuana joints are usually smoked right down to the bottom, to the butt; (4) because of the tendency of people to retain the smoke in their lungs and therefore because of the larger what they call puff volume?

A And longer retention time.

Q Yeah, is that—

A Yeah.

Q -- is that—

A Yeah.

Q That's what that is, eh?

A That's right. That's what he's saying.

Q Okay. All right. So—and doesn't he then conclude further down that there's six times greater reported usage of tobacco narrows the increase in marihuana tar deposition to two times, so that they're qualitative differences between the two types of smoke?

A He says, "This amplification of the exposure of the lungs to the smoke of marihuana narrows the gap

between an approximately sixfold greater quantity of reported usage of tobacco to perhaps only approximately twofold greater exposure.

Q Yes. And then a little further down, I think if I've got the right spot, he says, "Evidence that qualitative differences between the two types of smoke," do you see that part?

A Yes. "Evidence that qualitative differences between the two types of smoke may be more important than quantitative differences with respect to the development of chronic obstructive pulmonary disease derives from animal studies in which the morphologic and physiologic evidence of emphysema was found in rats exposed for six months to tobacco smoke, but not in rats exposed for the same period to smoke from a comparable quantity of marihuana."

Q And then he concludes on the next page that, "The findings from the present long-term followup study of heavy habitual marihuana smokers argue against the concept that continuing heavy use of marihuana is a significant risk factor for the development of C.O.P.D." And C.O.P.D., as I understand it, --

A Chronic Obstructive Pulmonary Disease.

Q -- is Chronic Obstructive Pulmonary Disease. Right. And then he qualifies it by saying, "These negative findings, however, do not imply that regular marihuana smoking is free of harmful pulmonary effects. Habitual marihuana smoking is associated with a higher than expected prevalence of symptoms of chronic bronchitis, as well as a higher incidence of acute bronchitis. Moreover, other evidence suggests that marihuana may be an important risk factor for the development of respiratory infection and possibly respiratory malignancies," and he recommends further studies.

A Yes.

Q Is that fair enough?

A Yes, that—

THE COURT: Those last two items, respiratory function and infection in malignancy would be the topics that he covered in those abstracts?

A In those abstracts.

MR. CONROY:

Q Yes. Okay.

A But I think we do need to be careful to note that that is his conclusion from his own findings, but he did not arrive at any explanation of why his findings differed from those of Bloom et al.

Q Okay.

A And there's no—since we have no reason to reject the findings of Bloom et al, we have to say that—that issue is really not yet resolved.

Q But in either case, we're talking about heavy chronic users?

A Yes.

Q And in this case, really four times what we've described as our starting point for the chronic user?

A Yes, that's right.

MR. CONROY: Thanks. That's fine, Your Honour.

THE COURT: All right. We will adjourn to 1:30.

MR. DOHM: Before we break, Your Honour, I'd just like to address the timing issue. I—because the doctor will have to return to Toronto and I want to make sure that my learned friend will have an opportunity to finish without seeking some adjustments in your normal sitting hours today.

MR. CONROY: Well, what's been going through my head, Your Honour, since hearing the evidence yesterday and considering it overnight, is that because this W.H.O. Report of 1995 -- about to become W.H.O. report of 1995, is so central to my friend's position and the evidence that's been given by Dr. Kalant, that I think in fairness we should have a copy of it provided, together with the reviews, the first stage of reviews and hopefully if it is published soon, the subsequent reviews, before we complete the cross examination of Dr. Kalant, and so we will not be able to complete that today.

THE COURT: All right.

MR. CONROY: And we will have to go to another time to do it, and that doesn't mean there aren't other things that we can't do to—in the interim in order to shorten things down and not delay things further, so my hope would be that the only thing left outstanding would be the completion of Dr. Kalant's cross examination.

THE COURT: All right.

MR. CONROY: But in my submission, my friend and his questioning has brought everything—looked at everything in the past and brought it up to this 1995 report, and it's obvious from even just the evidence this morning there are things referred to there that are specific studies and so on, that we haven't seen or haven't had an opportunity to look at or—or have our experts look at, and I think we—we need to do that before we complete the cross examination.

THE COURT: That report aside, it was my impression that we weren't going to be completing cross examination today in any event.

MR. CONROY: Yeah. Given where we're at, at this hour, I think you're right.

THE COURT: So we should probably reserve another day.

MR. CONROY: Yes.

THE COURT: If you could look to that over the lunch break—

MR. CONROY: I don't know what my friend's feeling is, but -- and I'm assuming that this would make things a lot easier for us to get a copy of the report if we aren't just relying upon Dr. Kalant to persuade them to give us one, if we had a court order that said that as a matter of fairness, the applicants should have a copy of what's being replied upon, I'm sure the World Health Organization would honour the order of the Court.

THE COURT: I'm sure you'd like to point out what jurisdiction I have to make such an order.

MR. CONROY: Well, yes, the jurisdiction you have is called fairness of the trial, Your Honour. It's at 11(d) of the Canadian Charter of Rights and Freedoms, and it's now a constitutional obligation that you have. So says the Supreme Court of Canada.

THE COURT: Do you think Geneva cares?

MR. CONROY: You make the order. All I'm saying is then the doctor—you say this—the witness has appeared and relied on—

THE COURT: I will express the serious desire of the Court to have the document before it, so it can be dealt with fairly as a piece of evidence.

MR. DOHM: The only other alternative to Your Honour would be to direct a stay of my friend's application.

MR. CONROY: I think as a matter of fairness, if my friend produces—if the government produces a witness that relies upon a significant document in the giving of their evidence, and my friend gets up and says questions like, "Now, considering everything you did in 1981, considering everything in the Howell report, considering everything in 1985 Committee," you know and then asks for an opinion, we as a matter of fairness should have that document and everything that it's about.

THE COURT: There are tremendous evidentiary problems associated with the Crown or any other party relying upon evidence of the kind that came from Dr. Kalant, and this is not your problem, sir, which is basically a report of—of meetings and what one expects to find in the volume, the consensus that one expects to find in the volume without putting the volume directly in front of the Court. You yourself has mention—have mentioned the hearsay problems that arise in some of these situations.

MR. DOHM: Well, not in that situation, I didn't, Your Honour, with respect.

THE COURT: Well, I'm going to raise it in—in that situation, because what we're—we're hearing from Dr. Kalant is this is our consensus.

MR. DOHM: I asked him if—what—if in his professional opinion anything had occurred in the development of science which would change his opinion.

THE COURT: Yes, you did ask that and I'm entitled to deal with his evidence in that narrow a respect, but we also have significant references to this particular document in progress.

MR. DOHM: I understand—I understand what Your Honour is saying and I understand Mr. Conroy's position,

and Dr. Kalant has already indicated that he won't attempt to obtain a copy of that report, and—

THE COURT: I—

MR. DOHM: -- if Your Honour—if Your Honour's expression of interest in the—in the report should be conveyed, I will be happy to convey that in conjunction with Dr. Kalant's request.

THE COURT: All right. If there are any concerns that you have or Geneva has with respect to how that document is treated, because it simply is a manuscript and it has yet to be adopted by the World Health Organization, I think within the confines of this proceeding, I can perhaps make certain rulings with respect to how its treated or whether it's even open to public inspection in any way.

MR. DOHM: All right.

THE COURT: So you can relate to them that I'm prepared to -- to deal with concerns they may have with respect to its status, its privilege.

A I'll be happy to do that and to contact them, as soon as I get back to Toronto to explain the—the need for it, and I—I can't predict how the—the responses of the World Health Organization bureaucracy are not always the most rapid but—

THE COURT: I would think that that might be the major stumbling block, because they have yet to put their stamp of approval on it, they may not wish it to be associated in any way, shape or form at this point with them.

A Would it be fair to say to them that we require access to it for evidentiary purposes, without in any sense regarding it as an official view or an official statement of the World Health Organization?

MR. CONROY: That's—that's the evidence. That's the truth of the matter as I understand it.

THE COURT: And I'm—I'm prepared to make a sealing order of some kind if it's necessary to protect it, in that respect.

MR. CONROY: My—I don't imagine they're that concerned about the information and the data. They—

they just can't say it's a World Health Report at this point.

A Yes, I think that's probably the—the main concern, and I suspect that there will be no problem getting permission, but they are not noted for their speed.

MR. CONROY: And just so—so I'm maybe clear on—on -- in terms of what I'd like to see, you know you—you chaired it. I'd like to know who all the members are. I'd like to know who all these eighty experts were that it was sent to. I'd like to—

THE COURT: Those—

MR. CONROY: -- to know what the—

THE COURT: Those are anonymous.

A Those—they're—I have no access to that information.

MR. CONROY: Okay. I'd like to see the responses then, I suppose is what I'm saying.

A Yes, I think we can give you those.

Q And that was incorporated into a revised edition as I understood it?

A Yes.

Q That was then sent out to others for review?

A Which we have not yet seen.

Q And they're anonymous too, these others?

A They presumably will be anonymous as the first one—

Q I see.

A -- first ones were, but we haven't yet seen those second—

Q Okay. And then that's—okay. I see, all right, so that hasn't even got out yet?

A No, I imagine it has gone out, but—

Q You haven't got the response?

A -- we—we haven't had the feedback.

Q Okay. If you get—if the feedback is there, I'd certainly like to see whatever feedback there is?

A Certainly.

Q And I think that's essentially it. There was an indication, I think in your evidence, that it will likely be published this year, if we can get some idea that might speed things up.

A Yes, I—I will ask that. The—I can give you my past experience with another report in which I've—in which I've participated on something quite non-controversial. It had to do with the use of new medications in the treatment of alcohol and drug dependence, and it was well over two years between the time we submitted the report and the time it finally appeared.

Q Okay.

A And that had to do with budgetary reasons and a change of personnel and simply slow response.

Q Just so that I'm—I'm clear, because you've relied on it a lot and because of the interaction and communication with the other people, and particularly those on the committee, I'm concerned to make sure we have the most current information, whether it has their seal of approval on it or not.

A No, I—I understand the requirement and I shall do my very best to get it as promptly as possible.

THE COURT: Can I ask you to see if you are able to obtain a new—a new date over the lunch break? Well, if we come back at twenty to, that will give you some time to meet with the Trial Coordinator. I can advise you—well, first of all, I would like it to be as soon as possible.

MR. CONROY: Okay.

THE COURT: And I can also advise you that as of July 14th of this year, I am moving out of this district into the Vancouver district, so that may affect where you will travel to.

MR. CONROY: We now have a train from Mission, Your Honour.

MR. DOHM: July 15th is looking pretty good for me now.

THE COURT: I thought it might be. We'll have to move all the Exhibits into Vancouver. My only concern with July 15th is it's a long way away. All right.

(WITNESS STOOD DOWN)

(PROCEEDINGS ADJOURNED)

(PROCEEDINGS RECONVENED)

HAROLD KALANT, recalled, testifies as follows:

MR. CONROY: We spoke to the Trial Coordinator, Your Honour, and the suggestion by Mr. Kurtz was that we simply put it over a month to give Dr. Kalant time to try and get what he can, so we can look at it, and then give him a good estimate—he's just worried that if we end up estimating a day and we go two, that's going to cause way more problems for him, so he says put it for a month, have a look at what we have and then fix the date, and then we just have to give him dates and—

THE COURT: All right.

MR. CONROY: -- he'll fix it on your calendar.

THE COURT: We'll do that then.

MR. CONROY: So I'm—I'm suggesting that when we break, subject to my friend, if we go to—let's pick March 5th, is that reasonable?

MR. DOHM: Certainly. I'm—as long as Your Honour expects to be here on the 5th, and if you had all your—

MR. CONROY: Well, I don't think the Court has to be here. I think it's just a matter of fixing—

THE COURT: He's just fixing a date.

MR. CONROY: -- a date.

MR. DOHM: All right. Certainly. That's fine then, sure.

MR. CONROY: Yes.

THE CLERK: March 5th then at 9:30 in courtroom 1, Your Honour.

THE COURT: Yes.

MR. CONROY: Now, Your Honour, we have over the break had copies made of the three extracts that Dr. Kalant referred to, there's two of those, one as an Exhibit and one for the Court, and that will be Exhibit—

THE COURT: 32.

MR. CONROY: -- 32, I think.

THE CLERK: Yes, Your Honour. Exhibit 32.

EXHIBIT 32 - EXTRACT

MR. CONROY: And while we're at it, you'll recall there was some discussion yesterday about other reports to do with the—babies, growth in babies and that sort of thing, and I've managed to get copies of those and I'd like to file those now and that way Dr. Kalant will have a set for consideration between now and the next time, too, instead of having to try and deal with them today. So here. I think what we might do, we could put these all together as one Exhibit. They're all on the same topic, or we could have them separate. I'm in the Court's hands. Maybe we better have them separate, just so they're easy to refer to. Okay. The first one is The Affects of Prenatal Tobacco and Marihuana Use on Offspring Growth from Birth through Three Years of Age.

THE CLERK: That's Exhibit 33, Your Honour.

THE COURT: Yes.

EXHIBIT 33 - AFFECTS OF PRENATAL TOBACCO AND MARIHUANA USE

MR. CONROY: And I'll have an extra set made for the Court because I've made an extra set but I didn't count properly and I gave it to Dr. Kalant.

THE COURT: Okay.

MR. CONROY: But I'll make sure there's an extra set so the Court has one that can be marked up. Do you have one?

A VOICE: (Indiscernible).

MR. CONROY: Okay. We best keep that as ours then. I'm going to hand up this package for the Court. I've already written 33 on it and we've already got a set, so that's just—just an extra set of all four, but I'll give you the—so the top one is the only one we've referred to so far.

THE CLERK: This is Exhibit 33.

MR. CONROY: 33.

THE CLERK: 33 for Her Honour.

MR. CONROY: Okay. The next one is Prenatal Marihuana Exposure and Neonatal Outcomes in Jamaica, an Ethnographic Study, and this was the one that referred to Dreyer.

THE CLERK: Exhibit 34, Your Honour.

THE COURT: Yes.

EXHIBIT 34 - PRENATAL MARIHUANA EXPOSURE AND NEONATAL OUTCOMES

MR. CONROY: Here's an extra one for the Court. The next one is the Association of Marihuana Use with Outcome of Pregnancy, and this is Lynn and others. An extra one for the Court.

THE CLERK: That's Exhibit 35.

THE COURT: Yes.

EXHIBIT 35 - ASSOCIATION OF MARIHUANA USE WITH OUTCOME

OF PREGNANCY

MR. CONROY: And then Prenatal Marihuana Use and Neonatal Outcome.

THE CLERK: 36, Your Honour.

EXHIBIT 36 - PRENATAL MARIHUANA USE AND NEONATAL

OUTCOME

MR. CONROY: Now, there was one other one which we don't have yet, and I think it was Tenes, T-e-n-e-s, and I'll endeavour to get that, and I'll provide copies to everybody ahead of the next time.

THE COURT: This particular copy on Exhibit 35, it's extremely difficult to read in spots. Actually, it's

impossible at spots. I don't know if your copy is better than mine.

MR. CONROY: Is this one better? We can always get—

THE CLERK: That's better.

THE COURT: It's actually blurred.

MR. CONROY: Okay. Well, take that one, Your Honour, and if you could give us—

THE COURT: Oh, yes, this is better.

MR. CONROY: -- that one just so we know what we have to get—we'll get another copy.

THE COURT: This is the Association of Marihuana Use, is that the one you've got?

MR. CONROY: Prenatal Marihuana Use in Neonatal—

THE COURT: No. No, you've given me the one wrong.

THE CLERK: The Association of—

THE COURT: Yes, that's not the one I've got. That one's okay.

THE CLERK: This is the one that's—

THE COURT: Yes.

THE CLERK: It's Exhibit 36.

THE COURT: Exhibit 36, Prenatal Marihuana Use and Neonatal Outcome.

MR. CONROY: All right. Ours, I'm told is just as bad, so let's leave it the way it is, but we'll make a note to get a better copy to everybody. So Exhibit 35 we need to replace.

THE CLERK: 36.

THE COURT: 36.

MR. CONROY: Oh, sorry, this is our 35. Okay. All right.

CROSS EXAMINATION BY MR. CONROY continuing:

Q Doctor, what I'm going to do because I—I don't want to end up going over the same ground again once we get the—hopefully get the information from that Committee—I'm going to carry on and take you through, just to be absolutely sure I understand exactly everything that you were saying yesterday.

So let's move on to the next area in sequence—well, continue dealing with the lung thing for a moment. Clear—it's clear that we're talking about the—the smoke from the smoking of marihuana as being the cause of the—the various lung problems we've been talking about and not THC?

A Not THC.

Q And the same thing with tobacco, it's—it's the smoke not the nicotine?

A That's correct.

Q And it would be true of smoking leaves or just about anything. When you heat them up in this type of a—rolled in anything and take that smoke into your lungs when it's heated up, as I understand it that process is what's likely to cause damage—health damage to various parts of the—of the system?

A That's right. It's the pyrolysis, the destruction by heat of the complex organic materials that make up the—the left.

Q So it's really nothing to do with—with cannabis per se in terms of its active—psychoactive ingredients and so on?

A No. It's the—

Q It's the pyrolysis process?

A That's right.

Q Okay.

A The difference between the smokes generated by different plant materials must depend upon other differences in composition other than the—the psychoactive material.

Q So when we—when you talked about the additive effect, a person smoking—a heavy chronic user say smokes one marihuana cigarette per day. If that person also is a tobacco smoker and smokes twenty cigarettes a day, we're—obviously that person is taking in a huge extra amount of smoke, --

A That’s right.

Q -- which, of course, logically and scientifically apparently obviously would cause more damage?

A That’s correct.

Q The more smoke, the more damage?

A That’s right.

Q All right. And there the ratio between the tobacco smoke and the marihuana smoke may be twenty to one, but it’s all smoke, correct?

A Fundamentally, yes.

Q And so that’s what’s going to do the damage?

A Yeah, other—other than the factors that we already talked about this morning, about the differences—relatively—relatively small differences in the—in the composition of the smoke in terms of the tar content or particulates from different types of plant material, and differences in the manner of smoking but taking account of those, fundamentally, yes. It’s a matter of how much smoke you get in from the sources.

Q All right. And so—but we have no way of, in that example, figuring out how much of the damage is caused by the one marihuana joint that the person smoked that day, and how much is caused by the tobacco or the cigarettes the person smoked, do we?

A No, in an individual case, I don’t think you could. All you could do is in a statistical sense, by looking at studies such as this which analyze separately for the effects of one and the other, get a rough order of magnitude of how much the—what the contribution might be from one another, --

Q But the—

A -- but it would be a rather futile exercise I think.

Q But—but obviously twenty cigarettes to one marihuana cigarette, allowing for the different methods of inhaling, it’s likely that the twenty cigarettes are—are going to contribute the largest amount of smoke over that day?

A Yes. Yes, I think so.

Q Yeah. Now—and the way we would try to minimize or reduce the health risks from taking smoke into the lungs if one is a marihuana smoker, I assume—correct me if I'm wrong—that the logical, natural thing a doctor might say to the patient who he finds smoking marihuana, he would firstly say, "Well, first stop taking deep lung breaths of the smoke," fair enough, that's a first point?

A Yes. The—that would certainly be good advice. The problem would be that if the person then said, "Yes, but if I do that, I don't get the effect I'm after."

Q Well, you know I—I'm told by what I've read that it doesn't make that much difference. Do you know that?

A No, other than that the experience, or at least the practice of smokers, has always been to inhale deeply in order to get the maximum yield from each cigarette.

Q Well, accordingly to Dr. Morgan, his manuscript, and I believe his evidence, he said that it doesn't add that much more psychoactive effect, that process. People think that, but that it doesn't in fact?

A Well, I saw that statement, but I don't know what he bases that on.

Q Okay. You have not seen anything to—

A No.

Q -- say otherwise?

A No.

Q Okay. All right. So—but that obviously is the—a thing a person should do?

A Yes.

Q Maybe they will get less—

A Less high.

Q -- psychoactive or high from it, but nevertheless the object being to reduce the health consequences, that would be a prudent step?

A That would—that would be a prudent step, yes.

Q Another one would be to put a filter in it?

A Yes.

Q Another one would be to—to tell them not to smoke the joint right down to the very end?

A Yes.

Q Because a lot of these tars and everything accumulate in that last part of the joint, isn't that right?

A That's right.

Q In fact, I understand—do you know what the figures are? My understanding is that an awful lot goes into that roach?

A Since we're going to be continuing the discussion later, I'll look back at our own data from the experiments which we did many years ago measuring the smoke production, and see if I can find in our records how much was recovered and how much was—was retained in the—in the butt.

Q I was actually told that—all right. If you took a marihuana cigarette and assume fifteen milligrams of THC in the cigarette, I understand that as you draw in the cigarette, the joint, you lose about fifty percent of the smoke to something called side—sidestream smoke?

A Yes, that's the—

Q Okay.

A -- the smoke which isn't drawn through the cigarette but simply dissipates into the air around the—around the cigarette.

Q So you'll—from the fifteen milligrams—or micrograms—

A No, that would probably be milligrams.

Q Milligrams of THC?

A Yes. Micrograms is a—a microgram is a thousandth of a milligram.

Q Okay. So fifteen milligrams in one marihuana joint, that's pretty high isn't it?

A That would be—no, that would be—

Q It's micrograms per kilo, isn't it? Isn't that the—

A Micrograms per kilo is a measure of dosage.

Q Oh.

A But amount in a cigarette, if you—a cigarette might be, for example, one gram, and if you had a one and a-half percent by weight THC content in the cannabis, that would yield—that would contain fifteen milligrams.

Q You see, my understanding was and we—there was discussion about the Robbe studies in terms of the—a hundred—it was micrograms per kilo—

A No, that was dosage.

Q Yes, that's what I'm talking about.

A Yeah.

Q And there was the three hundred micrograms per kilo?

A No, those were—those were I believe levels. They—did they not have—

Q It was—it was the dose, wasn't it? The dose used in the driving tests?

A Yes, you're right. I'm sorry, it was the dose so it was micrograms per kilo.

Q The average human, as I understand it, is roughly seventy kilograms, another exception?

A That's an average figure, yes.

Q All right. And so if we have fifteen micrograms per kilogram—no that can't be right. It's got to—

A No, it's—

Q -- it's got to be milligrams?

A No, it's milligrams per cigarette.

Q Yes. Okay. So you get roughly five to seven milligrams if fifty percent of it goes away in sidestream smoke on inhaling?

A Well, you would have theoretically seven and a-half milligrams—

Q And a-half—exactly, okay.

A Yeah.

Q And the rest, I understand, migrates down into the bottom of the roach, so that you're—you're not getting exactly a half necessarily. That's why I said to five to seven. If fifty percent goes in sidestream smoke, you get somewhere five to seven milligrams being inhaled, some gets burned off and some goes—and the rest goes down into the—what they call the roach, is that right?

A Yes. The—as you draw the smoke along the cigarette, there is a process of condensation or—or trapping of the smoke on the material that's still left uncombusted, and as you move down the cigarette with the combustion zone, you revolatilize that, and you can therefore continue to breathe it in until you get down to a butt too small to burn any longer without—without having it burning in your lips, --

Q Okay.

A -- so that you would—you would lose a certain amount that had condensed in the butt that was too small to smoke, but the amount of that would depend on how—on the—the efficiency of the smoker, because of course if you take a deep breath, you'll have less opportunity for—for condensation as the smoke is passing through the cigarette.

Q Okay. So coming back then to—to what we were talking about, the—the advice to the marihuana smoker in order to reduce the health consequences from the smoking would be, as we've said, stop taking it deeply into your lungs, inhaling in the—in that way. Don't smoke it down to this small amount. Put in a filter.

A Mm-hm.

Q These—these are the kinds of things that would substantially reduce the health consequences, would they not?

A Yes, that's true.

Q Okay. And that can be done easily by a doctor, educational materials, and so on to smokers, to encourage them to do that, much in the same way as we've been educating people about the consequences of tobacco smoking, fair enough?

A One—one would hope so.

Q Yeah. I mean we've seen good results with tobacco smoking over the last couple of years?

A Yes.

Q Yeah. Okay. All right. Let's carry on then. You talked about being unable to conclude that there was a risk of cancer from marihuana smoking simply because we just don't have the public health statistics for cannabis that we have for tobacco?

A That's true. We have—we have the experimental evidence which raises the possibility, but we don't have the public health statistics to know with certainty whether it happens and if it does, to what extent.

Q And then it was at that point in your evidence that you referred to the size of population and how accurate data we could obtain as to intensity and duration, that we'd need that kind of information in order to predict as I understood it, what really might happen—more likely happen in the future?

A Yes.

Q And you then mentioned that in the terms of a legal drug, you could get the data more easily than an illegal drug, partly because users are reluctant to provide data and you can't rely on it totally?

A Yes. As I indicated this morning in answer to one of your earlier questions the—the data in some types of surveys seem to be fairly reliable, but in terms of establishing the level of use with accuracy so that you can relate risk to level of use, I think that is still more difficult to do when people are maybe afraid to—to indicate the full extent of their use.

Q And the other factor, I think you said was current levels of use have to be determined. We know they're nowhere near as wide as tobacco, and that's why you

gave the estimate of thirty to forty years perhaps before—

A Yes.

Q -- we'd be able to—to tell?

A That's right.

Q Okay. And that's in order to do the kind of research that you do with the control groups and so on over this long period of time in order to be able to be definitive, is that right?

A Yes. I think the—the long-term studies—you're referring to the longitudinal studies?

Q Yes.

A Yes.

Q I assumed that's what you were referring to?

A Well, yes. I was referring to that and also to the—the Public Health type of statistics gathering, statistics over a long period of time, which includes the—the people who continue to use over long periods of time, but in terms of the longitudinal studies of designated groups whom you can follow as individuals to see the consequences of known levels of use, that is difficult for rather obvious reasons. That since you depend upon self-report of the—of the extent of use, it's not as good as if you could give a known amount—

Q Right.

A -- and know that the person was actually using it, and be able to relate quantitatively the definite levels of intake to definite observed levels of outcome. That is difficult and undoubtedly will continue to be difficult for the foreseeable future.

Q And we don't even have a—a start really on these type of public health statistics, do we?

A No, I—I—well, we have—we have a start, but nowhere near as much as one would like to—or need to have in order to be able to draw confident conclusions.

Q Notwithstanding the fact that we've had obvious use throughout the '60's and '70's and up to date, --

A Yes.

Q -- we don't have anywhere near the type of data—

A That we do for—

Q -- that you'd need?

A -- alcohol or tobacco. Yeah, that's right.

Q Okay. Do—how many—can you say how many years we do have of—of adequate data in terms of our beginning or our start?

A No, because in order to answer that, I would really need to know how the levels of use have changed in individual cases over time. In other words, the—the data that we have, as I mentioned earlier, are cross-sectional data from surveys.

Q All right. Now—

A They don't follow how individual users continue to use or change, and that makes it—I don't think yet we can say that we have the—

Q So—

A -- simply enough of the right—

Q But notwithstanding all these years of—of particularly those early years of concern in the mid to late '60's and into the late '70's, and although reduced funding and so on in between, but nevertheless still concerns and so on about marihuana use, people at the Addiction Research Foundation, for example, or is it the International Cannabinoid Society you mentioned earlier?

A International Cannabinoid Research Society.

Q Research Society.

A That's a relatively new group.

Q None of these groups who are involved in research have started that type of a process in Canada, is that right?

A Yes, I—I think that I would say—I would agree with you, because the—as I pointed out, I believe it was this

morning in talking about the statistics, the early surveys which—and by early I mean the first probably fifteen years of surveys, really did not ask the questions with the level of detail needed to define the numbers of users that—what we would now consider heavy levels of use. The—the surveys for years stopped at too low a level of use, instead of asking about the really high levels that are of medical concern.

Q All right. Let's then move on to—you mentioned cardiovascular and my friend was taking you through each topic in the 1981 A.R.F.W.H.O. report, do you remember that?

A Yes.

Q Now, as I understood you, in terms of the cardiovascular situation, your evidence essentially was that the—the evidence is minimal to suggest that there's any kind of risk?

A That's correct.

Q And so in your statement about significant risk, that is no longer an area of major concern?

A That's right.

Q Okay. So in terms of general toxicity, that's not a concern?

A Right.

Q Lungs, respiration is a concern?

A Yes.

Q Heart, cardiovascular is not a concern?

A No.

Q Okay. You did say if somebody had a pre-existing heart problem, I think you said coronary circulatory problem?

A Yes.

Q A person could be more susceptible to risk of heart attack in the acute phase because of the exciting of the heart rate and so on?

A Yes. I said that that is a—a hypothetical possibility because—

Q Because that—sorry.

A -- because if the heart is speeded up, and has a higher blood output per minute, it's doing more work and requires more oxygen and if the person has constriction of the coronary arteries and cannot get more oxygen to the heart, then that would increase the risk of a heart attack, but I pointed out that was a theoretical possibility, but that there were no reports of such.

Q Because that would happen from—if a person had that type of a pre-condition, that could happen from any number of activities such as running or dancing or—

A Oh, yes, certainly.

Q -- anything like that? Yeah. And then you said something about orthostatic blood pressure and I—I'm not sure if I understood that—

A Orthostatic hypotension.

Q Oh, hypotension. And you talked about somebody standing up quickly and feeling dizzy?

A Yes.

Q Now, that's not peculiar to people who've smoked marihuana either, is it?

A Well, no. I don't think there are very many things that marihuana does that are unique to marihuana.

Q Okay.

A The question is what are the things that marihuana does, and this was mentioned simply because some people do experience dizziness if they change position suddenly and the reason for that appears to be that there is a dilatation of blood vessels in the limbs, so that there's a fall in blood pressure, and if the person stands up suddenly, there isn't enough pressure to maintain the flow to the brain, and therefore the person feels dizzy, and that, too, I indicated was not really—could not be considered a serious health problem, because again that would be of significance only if the person was in a situation where dizziness might result in an accident or something like that.

Q But when we were talking about these factors, you were talking about the THC effect, as opposed to the smoking effect?

A Yes.

Q But again, you don't feel that we should be too concerned about this particular area?

A No. This has not—there has been no clinical evidence to suggest that this is a significant source of—of illness.

Q Okay.

THE COURT: Are you saying that the dilation effect that you're talking about and the subsequent dizziness is a consequence of the THC content—

A Yes.

THE COURT: -- as opposed to the smoke?

A As opposed to the smoke.

THE COURT: And is there a similar affect on the consumption of—or the use of tobacco, inhalation of tobacco?

A In—in experienced smokers, yes, but there again, one is faced with the problem that that may be due to the nicotine rather than to the smoker. Nicotine is a—

THE COURT: I think that's what I was—was asking, --

A Yeah.

THE COURT: -- whether the nicotine in a cigarette would have the—a similar sort of effect?

A Yes, it—it could.

MR. CONROY:

Q But again, not something we need to be too concerned about?

A Not really. We used to do a classroom experiment in physiology for the medical students that involved rapid smoking of two tobacco cigarettes and it almost invariably produced marked phaseal dilatation, a fall in

blood pressure, nausea and dizziness, and that was really done simply to show the—to illustrate the action of what are called nicotinic agents on the—on the regulation of blood pressure and blood vessels. So it's not unique to cannabis, but cannabis shares that property.

THE COURT: Thank you.

MR. CONROY:

Q All right. And you then went on and told us that since 1981, there hadn't been any significant changes in this area?

A No, there hasn't—to my knowledge there hasn't been any—

Q All right. The next topic—

A -- report in recent work—

Q Sorry. The next topic then in terms of health consequences that we talked about was growth and body weight, and there, as I understood it, you're saying this is where some of the Freid Studies came in?

A There I must apologize to the Court. I unintentionally misled you yesterday by mentally telescoping work from several different authors. The—I checked again last night and the references that I should have given you for the—the inhibition of fetal growth—sorry, I forgot to flag this. Yes, here we are. The—the references for the inhibition of fetal growth that I should have given you are Hingson et al in 1982.

Q Hinkson?

A Hingson, H-i-n-g-s-o-n, which involved sixteen hundred and ninety mothers and Hatch and Bracken, 1986, that involved three thousand, eight hundred and fifty-seven women.

Q That's two people, Hatch and Bracken?

A Hatch and Bracken.

Q 19 --

A '86.

Q Okay.

A And they both found significantly lower birth rates in the babies of mothers who used marihuana during pregnancy, and the babies were smaller for gestational age, and in those studies, the marihuana effect was greater than that of tobacco smoking, but additive with it, and that—those are the studies, Your Honour, in which—which I was getting confused about yesterday in which they used a statistical correction for the effect of the smoke, so that I apologize for confusing the details of the Freid study with those.

Q We should leave Freid aside on this topic.

A Freid—yes, Freid is—

Q Freid is more this—the other area?

A Freid is more the question of the developmental effect on—on cognitive functions.

Q Yes, okay. All right. And on this growth body weight issue though, I think you said as well that it's transient, it seems to disappear in—

A Yes.

Q -- a relatively short period of time, up to—at a year they seem to be normal?

A They seem to be normal.

Q And the Arkansas study also indicated a tolerance develops the effect inhibitor—the inhibitory effect I think you said?

A The Arkansas study?

Q Well, I think you said there was one good long-term study, Arkansas—

A Oh, the animal—the animal study, yes.

Q Yes, sorry.

A Right, correct.

Q Sorry.

A Yes.

Q And in that study it determined that—

A That there was no residual—or no long-lasting effect.

Q No long-lasting effect, so again that's an area then that we could say, not a significant problem for us to worry about?

A I would be inclined to agree with that, yes.

Q Okay.

A That the retardation of growth is overall not a very important problem. I would be—I think we should devote more attention to the discussion of the effect on cognitive development.

Q Yes, I'm going to come—I want to go through each one just so I see which are the really—which ones you put into the significant category and which ones you don't. The next one was a miscellaneous category and it included—you mentioned this aspergillis (phonetic). Do you remember that?

A Yes.

Q Now, I'm told—and you've said that was pretty rare, but I'm told that that is caused by poor storage of the marihuana, did you know that?

A Yes. It's a—it's a fungus which grows on marihuana.

Q Yeah. So it's got to do with how the person stores the stuff, because if they don't store it properly, there might be some contaminants or this fungus that comes into it, that can lead to this problem, fair enough?

A Correct.

Q So if we had appropriate standards of quality control and packaging and manufacturing and all the rest of it, we would probably eliminate the—or we'd come pretty close to eliminating the chances of that happening, wouldn't we?

A Yes, I think that's probably true. There are two—

Q We'd still be stuck with the individual who may get it from the store—

A There were—

Q -- if it was available and not look—not follow the -- the instructions for storage or something like that?

A Yes. There are two considerations that come into that. One is the contamination of the—of the cannabis, and that certainly theoretically is controllable or remediable, and the other is the question of whether the THC itself, or the smoke of cannabis impairs the immune function of the pulmonary macrophasias to protect against the bacterial or fungal contaminants.

Q Now, we—we talk about that and we get to the immune factor?

A Yes.

Q Fair enough? But just because on this one under miscellaneous you've said it's very rare?

A Yes.

Q It's not nice if it happens to you, --

A That's right.

Q -- but it's pretty rare even under existing circumstances?

A Yes.

Q My point being that if circumstances change so that there was some rules that governed quality control of the product in terms of availability to members of the public, that would go even further to reducing the chance of this happening?

A Yes.

Q Okay. And then—so we can again put that one in the category of not a significant health risk concern at this point?

A I would say certainly not a major health risk.

Q Okay. And then my friend mentioned the gastrointestinal stuff, and again that's not an area that you would put under the significant health risk either?

A No.

Q And then you also talked about at this point toxicity arising from an unusual method such as the example you gave was the person who swallows a balloon because he thinks the police are about to grab him and then the balloon breaks in the stomach or whatever, and that causes a—can cause a serious problem, fair enough?

A Yes.

Q And again, if the law was changed, some people would stop worrying about police coming to—to grab them. It would be far less likely that that would be anything we should worry about?

A Yes, I agree.

Q So we wouldn't put that in the category of significant health risk in those circumstances?

A Correct.

Q Okay. So we could say that that might be a health risk that arises as a result of the decision by others as to what the social control policy is going to be?

A Yes.

Q Okay. Now, you then talked about the cellular toxicity, damage to cells, is that another way to say that?

A Yes.

Q ANd you said in '81 no firm conclusion, and that the results in the human tests were conflicting?

A Yes.

Q And this was an area that you thought we should have longitudinal studies?

A That's correct.

Q Because we just don't know at this point whether it is a significant health risk or not, fair enough?

A Yes. We—we have evidence that it can occur in the test tube, but we don't know whether the levels of drug

which are needed to produce such effects are likely to occur in people smoking cannabis, and if so, how frequently, so—

Q And at this point in time, on the evidence, we have no evidence of it being a public health risk—significant public health risk, I should say, but it's worth continuing to look into?

A Yes.

Q And that's—you talked about that in '81 and '95 and that's still the position?

A That's still the case.

Q And it was on that topic that you said we just don't know what the functional implications are for humans, is that—did I get that right?

A I don't recall that, but I would agree with the—

Q Okay. Then the next one was carcinogenicity, am I saying that right?

A Yes. Yes.

Q And this is the one in which you talk about these scattered case reports, as I understand it that have come to light since '81?

A That's correct.

Q And are these particular reports detailed then in the materials that were available to the 1995 Committee?

A They—yes, they were available to the Committee. I don't recall whether specific reference was made to them or not, but we'll see that on the—

Q Is this something that we could get the actual studies or reports that relate to these scattered events so that there's something written about them that we could look at?

A Yes, certainly. Yes, because they're on—

Q And can you get those independently from the—

A Yes, they're the open literature and I can easily get those.

Q Okay. Are they well known articles?

A Reasonably well known. They've been—they are in journals which are respectable journals and are available in the library.

Q Can you give us the names of the authors, because we could maybe get them ourselves?

A Yes. I apologize for fumbling through this. It's not very well marked.

Q Well, let's do this. I can get them from you later or through Mr. Dohm. As long as they're available and we can have reference to them, that we can then deal with them between now and the next time. My—my note was that there were scattered case reports. They related primarily to marihuana and tobacco smokers?

A Yes, that's true.

Q The ones with problems, it was a very small group of marihuana smokers—marihuana only smokers I should say?

A Yes. I have the references here now. I can read those.

Q Okay.

A Yeah. One is Morris, 1985. That's not carcinoma but pulmonary tissue changes, lung tissue changes in—in accident victims who were all known to be marihuana users, and which were described as being of a degree not seen until considerably later in life in those who smoke only tobacco.

Q The marihuana only users?

A No, I think they were—my recollection is that they were combined—

Q Both.

A -- marihuana and tobacco users.

Q Okay.

A And then there's a report by Ferguson et al, 1989, about a metasticizing lung cancer in a twenty-seven year old man that was attributed by the clinicians to his heavy use of cannabis, and he was an extraordinarily heavy user, twenty or more cigarettes a day of cannabis.

Q He was smoke—and doing deep lung—

A And since the age of eleven. So—and then there were a series of additional cases described by Donald—

Q Oh, just before we leave that one then, Ferguson, a tobacco smoker as well?

A I believe this one was a cannabis only smoker.

Q Cannabis only, twenty—

A Twenty or more cigarettes a day.

Q -- joints per day?

A And he'd been smoking it since the age of eleven.

Q So he was doing it—

A Truly an extraordinarily heavy user.

Q -- it to a—a huge degree, especially allowing for the method of intake, smoking as many as a cigarette smoker?

A Yes.

Q Okay.

A And then Donald, 1991, described a series of cases, most of which were—most of whom were combined cannabis and tobacco use—users, but one was exclusively cannabis. That was in a publication called Advances in Experimental Medicine and Biology, 1991.

Q All right. And those are the primary ones?

A Yes.

Q Okay. All right. And my note was then that it was a very, very small number that were marihuana only and it sounds like we're talking one very exceptional case, and a few others?

A Yes.

Q Okay. And these people were in their late teens or early twenties and you saw some—or they found actual—in some of the cases anyway, cancer of the upper airways, the pharynx and upper esophagus is what you said?

A That's right.

Q And you said that this was unusual because these people were so young—

A Yes.

Q -- to see that in such a short period of time?

A That's correct.

Q Because I have a note that the Court then asked you about well, wouldn't we see this then from people coming from the '60's and '70's, if this was going to happen regularly as opposed to being exceptional, and I think my note was you said well, it usually—or it needs from smoke—you need years of heavy exposure to produce a result like this usually?

A And—yes, and that's why I think one is now beginning to see reports in the late 1980's and the early 1990's where there wouldn't have been any before, because the duration of smoking had not been long enough before.

Q But these people were quite young?

A Yes, they were young but they had years of use.

Q Well, the—the one did from age eleven but—

A Yeah, and the others would probably have had up to ten years of use anyway.

Q Okay.

A So we're starting to see reports which are probably indicative of the fact that the levels of use and the duration of use has increased.

Q But still bearing in mind the size of that cohort back in 19 -- the baby boomer cohort if I can call it that, this is a minute amount of cases that we're getting, isn't it?

And we're talking about a period that goes back thirty years—well, twenty to thirty years. Let's put it that way.

A Twenty.

Q Fair enough?

A Yes, it is a minute group. The problem is to—is to attempt to predict from that what the course of accumulation of data will be over coming years.

Q Because you—you did say early North American use was not as heavy as we see in a fraction of users now, --

A That's correct.

Q -- that the frequency of use has increased and therefore you expect to see more?

A That's right.

Q Have I got that right?

A I—I think one would expect that if the cannabis is a major contributor to these—to the production of these tumors, we should start to see increasing numbers of them over the next few years.

Q But—so when you say frequency of users has increased, am I right in understanding though, that you're only talking about this '91/'92 slight increase that's mostly in adolescents?

A No, what I'm thinking of is that as people were recruited into use during the period when levels of use were going up in the cross-sectional studies, we were starting then to recruit a cohort of people who by now may have long enough use and heavy enough use to start showing these—these problems, so if that is the case, we should see more of these over the next few years.

Q Okay. So we're talking about the people in the '60's and '70's when we had the high rates of use?

A Yes, we're talking—

Q It's those people that you expect that we might see—if it is a problem—

A Yes.

Q -- you expect that from that cohort, we should start to see some—

A We should now be—

Q -- over the next three or four years?

A Yes, we should see more of this occurring—

Q Because—

A -- over the next—

Q -- that group's all pushing fifty or starting to, I should say starting to, those from '47, '48 certainly are?

A Yes, but they—they weren't cannabis users. Oh, you mean their age now?

Q Yes.

A Yes. They're—yes, they're—the oldest ones in the group --

Q Are starting to—

A -- would be nudging fifty.

Q All right. So you're not talking about the—when you said early North American use not as heavy as we see in a fraction of users now, that reference was to the adolescents, was it?

A Yes.

Q But when you said the frequency of users increased, you were talking about the older group?

A Yes, the—the period of roughly ten to fifteen years in which there was a—a steady increase in use, which indicated that more people were using.

Q Yes.

A Because these were—these were not accurate figures on amount of individual use, but on numbers of people using.

Q All right. And again, it's the chronic heavy user—

A Yes.

Q -- that we're talking about?

A That's right.

Q We're not talking about the occasional or the moderate, we're talking about the one or more marihuana cigarettes a day type person who comes out of that period in the mid '60's to now, that you might see—you expect that you might see this?

A Yes.

Q So we've got this twenty to thirty year period that we've talked about?

A Yes.

Q So in terms of the overall general marihuana using population, if I can put it that way, the ninety-five percent—I think we said five percent are the high users estimated—again, it's not a significant health risk for the ninety-five percent. It is for that five percent?

A Yes.

Q Okay. The next area was biomechanical, I believe it was—maybe it was biochemical—no, it was biochemical, and I think it was a brief reference to the receptors this anandinligen (phonetic) or—

A Oh, yes, but that was not a—that was just discussion of current knowledge, not—

Q Yes.

A -- health hazards.

Q Nothing of concern in the biochemical, as I understand?

A No, that—our—our discussion of that was not related specifically to health hazards, but just to new knowledge on how the drug acts.

Q But I thought that in '81 there were some potential concerns referred to in the report, but they didn't pan out, is that right?

A I better check back to see what you're referring to.

Q I just made a brief note and that was page thirteen?

A Oh, I—yes, now I know what you're referring to.

Q Do you remember that?

A Yes, that was the Isidoritis (phonetic) et al, the Greek study which describes or put forward a—a hypothetical mechanism of action that was offered as a possible basis of—of cell damage.

Q All right.

A Isidoritis, yes.

Q And that—

A That did not pan out.

Q So we wouldn't put that as a significant health risk?

A No. No.

Q Okay. Then we went on to deal with the immune system and now I've got a note before that to do with allergies. In terms of allergies, there's not a concern?

A That's right.

Q Okay. But on the immune system, that was one where again you felt we need some further study, some further investigation?

A Yes.

Q In '81, there was a suggestion of some impairment?

A Yes.

Q And since '81 there's been some indications that we could have less concern but the doubts haven't been resolved, is that—did I understand that—

A No, I wouldn’t say that we have reason for less concern. I think we are still left with the same concerns.

Q My note was not enough to resolve doubts completely. Did I get that wrong?

A I think what I would probably have said is not enough to resolve the issue.

Q Okay.

A Not to—not to eliminate the doubts, but to have a definite answer one way or the other.

Q Okay. And the work—the more work was defining the effects of cannabinols, is that—or cannabinoids?

A No, cannabinoids.

Q Cannabinoids, sorry. Okay. Now, just to touch on this business of the receptor and so on, as I understand that, though, that is a significant bit of scientific information for us, --

A Yes.

Q -- to the extent we know now where the receptors—where the major receptors are in the brain, correct?

A Yes.

Q And we know that the main one is in the frontal lobe, as I understand?

A No, it’s in various parts of the brain.

Q But the major ones in terms of the impact—

A No, I don’t think we can say that yet, because the—the receptors in brain stem and in—in hypothalamus and so on, are probably all involved in—in different pharmacological actions of the—of THC.

Q Are you familiar with the most recent investigations in that area?

A Which are you referring to?

Q The business of these receptors and are they—

A No, but I mean which studies are you thinking of?

Q I'm—I'm going on information I received from Dr. Morgan.

A Because I—I also there are quite a number of them in this 1996 symposium on cannabinoid receptors and andamoid (phonetic) receptors as they're more properly called, I guess.

Q My understanding from Dr. Morgan was that one of the receptors—and you can tell me in case I get it wrong, but the receptor in—the part of the brain that would deal with short-term memory for example, if a cannabis—

A No, I would say that's an oversimplification.

Q Okay.

A Short-term memory or memory mechanisms in general involve very heavily the hypocampus (phonetic) but not exclusively. Other parts of the brain are also involved because it's the general axiom of brain function that if you intervene anywhere in the brain, you affect things in all kinds of other parts of the brain, because there's so much interconnection, but I'm just looking to see what I have here about the recep—the distribution of receptors in the brain.

No, they—they're involved in various parts of the brain and one of—Dr. Martin, who was one of the participants in Geneva and who is very actively engaged in research on the receptors indicated that the receptor localization in the brain is consistent with roles in thought processes, memory, reward, pain perception and motor coordination, as well as some of the temporary endocrine effects, plus others as yet inadequately explored, and the conclusion was that at present, it remains to be determined whether and to what extent the use of cannabis will alter processes which regulate the endogenous cannabinoid system and anandoid (phonetic) system, so that I don't think we're really nearly far enough along yet to be able to say that a particular receptor is specifically involved with memory or with—with thought processes.

Q But having found the receptor, that did mean that we have—our bodies create a form of THC?

A No.

Q That binds on that receptor?

A No. Our bodies create a substance which is a—a fatty acid derivative that is related to compounds known as

prostaglandins, which have very important physiological regulatory effects on various organs.

Q Yes.

A And which the THC and possibly other cannabinoids resemble enough in some respects to be able to bind the same receptors. You see, I can give you an analogy that may be more familiar. Morphine and codeine, heroin, the opiod drugs, bind to what are called opiod receptors and produce effects on pain perception, on endocrine function, on—on mood, on motivation and so on, at various points in the nervous system, in the spinal cord and in the brain stem, and the forebrain. Yet, there's nothing in the human body which even vaguely resembles morphine or heroin or codeine and the mystery has always been, what are receptors doing there for substances that don't exist in the body? And that was answered by the discovery of protein substances or polypeptides which are fragments of protein, that are the—the naturally occurring substances that are synthesized in the brain and are released from nerve cells and bind to these receptors and initiate actions, and the opiods are, in a sense, usurping the receptors that are for these other substances.

Q There's more substance being introduced into the system than happens naturally, is that another way to put it?

A Well, not only more but different.

Q More, but acting on the same receptors?

A But acting on the same receptors.

Q And as I understand it in terms of the opiods, those receptors are in the brain stem primarily?

A No, they are in various parts of the brain also, in the brain stem, in the—in the thalamus and the hypothalamus, in the cortex. There are quite a number of different sites at which there are opiod receptors of different types.

Q My understanding is, and I think the evidence here has been that one of the reasons why people can overdose on those types of drugs and cause a shutdown to the system, is because of the location of those receptors that are in the brain stem?

A No. What they're talking about is the suppression of breathing.

Q Yes.

A Fatality due to suppression of breathing is due to receptors in the brain stem.

Q And that can happen with the opiods?

A Yes.

Q It can't happen with marihuana?

A Well, you'll perhaps recall I mentioned the cases of extreme overdose, the ones of the children who accidentally got access to—or unintentionally—

Q Eating it or something?

A Yeah, eating it, and the cases were—there's been a sudden massive overdose from a burst container in the -- in the intestinal tract. Those did have a very marked suppression of breathing, but not fatal.

Q Yes. Again, by an oral administration or it finding itself in the stomach because of the balloon or whatever?

A Well—that's right, but I mean the action was not in the stomach, the action was in the brain.

Q Yes, but we—that can't happen by the smoking of marihuana, can it?

A No. I think that would be extremely unlikely because if you put yourself that deeply under the affect, you would have fallen asleep before you took enough to get there.

Q Before you ever got there, yeah. Is that right? Okay. And my understanding is that tobacco is fairly similar in that respect. Again, tobacco, you're not going to have that happen by smoking a huge amount of tobacco?

A No.

Q Or even by eating a huge amount of tobacco? You're going to get sick probably and—and vomit before you'd ever get to the stage where that would happen?

A No, there have been a very small number of fatalities in children from eating tobacco, or in—in adults from working with concentrated nicotine solutions that are used as insecticides in agriculture, and the accidental absorption or ingestion of even a moderately small amount of such concentrated solutions has resulted in fatality.

Q Okay. So—but when we talk about this overall area, the immune system, we're still in much the same way as a number of the other areas. You feel because of some things that have been found, we should do some more studies, correct?

A Yes.

Q And again the concern relates to that heavy chronic user, that five percent of the user population we've been talking about?

A Yes.

Q Again, the rest—it's not a significant health risk for the rest. It may well be for that five percent?

A Yes, I would agree.

Q Is that a fair way to put it?

A Yes.

Q Okay.

THE COURT: Before you leave that field, I'm just looking at Exhibit 32 which are the—the three abstracts that you provided us with.

A Yes.

THE COURT: And the first one refers to the effect of habitual use of marihuana on antibacterial, etcetera?

A Yes, that was the first of three.

THE COURT: Is that an immune system problem?

A Yes, that's an immune system function because the—as I explained yesterday, the immune system has two major types of function to defend the body against infections or against tumors or against foreign chemicals. What is called a humeral function, which is the formation

and release of antibodies and the ephagocitic (phonetic) or cellular function which is the function of macrophasias to engulf the—the foreign organisms or particles, substances, whatever they are and destroy them by enzyme action, so that this ability to ingest staphylococci and destroy them is a function of the cellular immune system.

THE COURT: All right. And it—according to this research appears to disrupt the functional activity of—of the immune affector cells to—

A Yes, defects in the functional activity of the lungs immunifactor cells.

THE COURT: And do we know if that's a product of the THC content or the smoking, just like tobacco smoke?

A Well, this was—this study doesn't—it doesn't clarify that because these were macrophasias obtained by washing out the—the lungs of smokers, but in the cellulars—or at least the invitro studies, in test tube studies that have been done by other groups, they have added THC itself to the incubation medium in which the cells were growing, so that that appears to be a function of the THC.

MR. CONROY:

Q But are these not studies again by Tashkin's group involving the—the people who had been smoking for fifteen years, followed for the eight year period?

A Yes, these are but they—as I said there are other studies by—most prominently by Klein's group, --

Q Yes.

A -- which are done by the addition of THC to the cells in cell culture.

Q And that's in the lab—in the laboratory?

A Yes, in the laboratory.

Q All right. But the—but the concerns that emanate out—emanate from Exhibit 32 are again Tashkin's longitudinal study—

A That's correct.

Q -- of heavy chronic users?

A That's correct.

Q A twenty-three year period basically, eight years in monitoring, twenty—

A Well, these are not—probably not the same subjects because the numbers—

Q I see.

A -- the numbers in each group are different from the numbers in the—given in the paper on—on the longitudinal study, so this must be either a subset of the longitudinal study, or a different group.

Q Okay. But it's an—again done in a longitudinal study method?

A Using the methods that were—

Q Yeah.

A -- done in the longitudinal study.

Q All right. So again, in terms of the immune—or I think you may have already answered this—our concern for the future in terms of further studies is because of this heavy chronic user group, not the occasional or the moderate?

A That's right. There—there are a few public health type studies to see whether cannabis users or long term cannabis users have a higher incidence of infections and they have not been very conclusive because again of the need to separate the effect of cannabis from the effect of tobacco and possibly of other drugs that can affect the immune system and the conclusion was when statistical methods were used to—to try to parcel out the responsibility or the contribution of these other factors, that the cannabis users had a slightly but not dramatically higher incidence of—of infections.

Q Nothing we can—we'd put then as a significant health risk?

A Well, again—

Q That—at this point?

A -- perhaps I don't want to quibble about terminology but it's—I would prefer to say that nothing that represents a major public health problem?

Q All right. Fair enough. Okay. We dealt with allergies. Endocrinal aspects was the next one, and my note of your evidence there was that no evidence to conclude that there's any long term problems, but we need again to keep monitoring it and checking it out?

A That's correct.

Q The—and also it's been determined that there's tolerance that develops in this area and so the committee with—the—since '81, if I can put it that way, there's less concern than there was in '81, but still a need to monitor and check it out?

A I think that's correct, yes. The—the—

Q And again we're talking about the concern arising from the chronic user?

A That's right.

Q Yeah. Okay. And then we went to reproduction and development and you told us there's nothing to suggest any long term effect on fertility and that area?

A That's correct.

Q And so that area is not considered a significant risk, a major risk I guess I should say at this point?

A No.

Q But that's where we got into the discussion about Dr. Freid's—is it Dr. Freid?

A Yes.

Q Studies to do with the prenatal and postnatal development?

A That's right.

Q Okay. Now, I'm going to—we'll just touch on what you said yesterday, but these other reports that I've just filed obviously go to that issue?

A Yes.

Q And will need to have you review those at some point so we can deal with that maybe next time, but my understanding is that even considering all of the Freid studies, you're saying that it's not dramatic in degree but it's statistically significant and—and raises some concern and that's why you think there has to be continued monitoring and study?

A Yes. The—it was not dramatic in degree. The concern is principly as to whether or not even a—a relatively small but consistent impairment of verbal functions and abstract problem-solving and so on will impair the academic and intellectual progress of the children, and therefore influence their long-term career possibilities, things of this kind.

Q Okay. Your—my note, and again I may well be wrong on this, but it—I thought Her Honour asked you about whether there'd been any tests on tobacco smokers—mother tobacco smokers and that you mentioned—maybe I got it wrong. Maybe it wasn't you who said this, maybe it was the judge asking you this, but I've got a note, marihuana smokers, tobacco smokers and then combinations of the two?

A Yes. This was the area in which—

Q We got—

A I was telescoping different—

Q Okay.

A -- studies from different authors and this is something which I would prefer to answer again when the—

Q Okay.

A -- when the court proceedings resume because I'll have a chance then to get the—the Freid studies and see exactly what the experimental design was.

Q And would you—would you check, because I think you said that the marihuana smokers alone had the more significant impairment to the other groups and—

A That's—yes, that's what I want to check and I want to make sure whether they were separate tobacco and marihuana and combined tobacco and marihuana groups,

or whether they used a statistical method for sorting out the contributions of the two.

Q Okay. All right. Again, though, the concern is the chronic user, --

A Yeah.

Q -- the children of chronic users?

A Yes, because I think it's fair to say that only chronic users would be likely to be smoking enough to make pregnancy to—to have an affect on the fetus.

Q All right. Okay. All right. Nervous system was next and again, in terms of nervous system the concern arises from the short-term memory effect and the panic anxiety type reactions that people get in the acute phase, or some people get in the acute phase, that's part of it?

A That's part of it. Yes, the—the acute problems are those.

Q Do you agree though that the panic and paranoia are fairly rare, but it's more this panic—it's more of the anxiety dysphoria—am I pronouncing that right?

A Yes, that's correct.

Q Okay.

A Yes, they—I would say that it is—it has been decreasing steadily in—in frequency because as experience among users has grown, they're less likely to be frightened by unexpected sensations and the perceptions.

Q The fear—fear of the unknown?

A Yes.

Q And so—and as I understand it, when that happens, it's usually the very inexperienced user?

A That's correct.

Q And also it's—it's a very transient condition that can be resolved quickly by—with help from somebody to calm them or reassure them or whatever?

A Yes. It's—it's a matter in most cases of a couple of days at most. In rather rare cases, it may last longer but we're still talking about short-term transitory states.

Q Right. And there obviously we're talking about the novice inexperienced user as opposed to the chronic?

A Or as a—as I mentioned yesterday, or someone who suddenly has a much larger dose than that person's accustomed to.

Q Well, sure. Okay. But in the chronic user, the concerns were more to do with the learning process and things like that?

A With learning, memory, thought processes, motivation.

Q All right. And in that respect, the concern is with short-term memory as opposed to long-term memory you told us?

A Yes. Long-term memory has not proven to be affected by it.

Q And so again, it's a fairly—it's a fairly brief duration. It—it's during the acute phase, is it not, that the short-term memory loss is there?

A No. I think you're confounding two different things that I was talking about.

Q Okay.

A In the acute actions, yes. The—that's a transitory thing which is only for—of a few hours duration while the drug action is in effect.

Q Yes.

A What I was talking about in relation to the chronic or long-term damage is the—or functional impairment, is the—the long-lasting impairment. This may sound strange, but it's not. Long-lasting impairment of short-term memory, and I—I can explain to you what I mean by that.

Q Well, this was the business about the levels—

A But this isn't the person who—this isn't the person who smokes chronically and therefore remains under some measurable drug effect most of the time, and what

I mean by long-lasting impairment of short term memory is that the—when you acquire a new memory, to lay down a memory there's an immediate retention which depends upon—purely upon a temporary transient function and nerve cells, and then there's a transition through an intermediate period when new proteins are being laid down that store that memory. And then there's a long term—a longer term process involving interside (phonetic) of their connections that is necessary for the ability to recall that memory, and what you find in long term heavy users is that their long term memories from a long time back are not affected. They still remember things from a long time back, but they have an ongoing difficulty in remembering things that have happened recently and in retaining those memories, which is part of the learning process. And this is why intellectual functions of various kinds, school learning, job learning and so on, are at risk of being seriously impaired in people who are regular heavy users.

Q Okay.

THE COURT: And who stay.

A And who stay as users.

THE COURT: Or as habitual users.

A Now, I did also refer to the possibility that some cases do not recover if they stop using it. In other words, the majority, even of those with this type of long term impairment of—of short term memory do recover if they stop smoking for whatever reason, but some—those who have been smoking probably most heavily and for the longest time may not recover. And there are clinical papers from many years back describing persistence of changes—of cognitive changes in people who had been heavy smokers, had stopped, had been off for many months or years, and retained some level of functional impairment. And it was at that point that I mentioned our own experimental studies in animals which showed impaired learning ability, as much as a-quarter or even a-half of the animal's lifespan, after having stopped the cannabis administration.

MR. CONROY:

Q The concern that goes through my mind trying to look at this logically is that if you can have long term impairment of short term memory, sooner or later all memory would disappear because it was all short term at one point. Pretty soon it would—it would accumulate and there'd be no memory left at all.

A No, because the things that were—that were laid down before you started to smoke cannabis would remain.

Q So there'd be this huge gap that would be there?

A Yes, there's difficulty, a cloudiness and the users themselves, when—when—and there are a significant number of users who do come to, for example, the youth clinic at the A.R.F. for treatment, and among the most common reasons given for coming for treatment are loss of memory, inability to remember things, inability to keep track of what they're supposed to be doing, running into problems of that kind, so that I think this is—although it may sound funny, it is in fact a very serious inconvenience for the person.

THE COURT: When—when you refer to some clinical papers from way back, I think you said, dealing with patients or users who had—had stopped using marihuana, but seemed not to be able to recover their short term memory skills, do you know if the users involved there were purely marihuana users, or are we talking about people who have mixed drug consumption habits?

A That—that is a—a very legitimate question, and I suspect that in most cases, there was more than one drug involved, because this was from the period of the early '70's when the youth clinic was set up in the trailer in Yorkville, for example, in Toronto, and most of the people who were heavy users at that time were also using LSD—loss of short term memory.

THE COURT: This is long term.

A That's even worse. That probably means organic brain damage, but no—mescaline, that's what I was trying to recall and—and other synthetic drugs of the amphetamine-derivative type, so that I think it's a legitimate question as to how much of that was exclusively due to marihuana and how much was due to the combined use of a range of different drugs. I don't think one can ever answer that from post—I mean retrospective clinical histories. One only gets leads that one can worry about and try to—try to think of some way of sorting it out, but obviously experimental solutions would be impossible. You can't do that to human beings. You really depend on—depend on animal studies to see if you can produce similar things with cannabis alone, and that is what we were doing with the—with the rat studies.

MR. CONROY:

Q I keep wondering what happened to the last ten years so I guess that's a combination of long and short term memory, isn't it?

A Yes.

THE COURT: It's shortly after three. Would this be a convenient time?

MR. CONROY:

Q One last little point on that. I think you did say that this was common from alcohol and other sedative-type drugs as well?

A Yes. And that there's better knowledge of what the cellular changes are in the brain that's responsible for the memory loss in, for example, patients with Corsicoff's Syndrome in chronic alcoholism. There—that has been linked to a specific loss of cells in particular parts of the brain and this has not been the case with cannabis.

MR. CONROY: Yes. Thank you, Doctor.

THE COURT: All right. We'll take fifteen minutes.

(WITNESS STOOD DOWN)

(PROCEEDINGS ADJOURNED)

(PROCEEDINGS RECONVENED)

HAROLD KALANT, recalled, testifies as follows:

CROSS EXAMINATION BY MR. CONROY continuing:

Q The next topic area that I have noted was the—it was actually other affects, but the first one talked about was this amotivational syndrome. Now, that's a—a topic that has been tossed around in terms of cannabis use for a long time, particularly in the press and so on, wasn't it?

A Yes, not only in the press, --

Q Not only in the press.

A -- it was in the clinical record, as well.

Q And—but it was used a lot even as I recall it—I don't know if your memory is the same as mine, in terms of media reports about cannabis and so on, that people would get lazy and not work and this sort of thing, fair enough?

A Yeah.

Q And anyway, the position in '81 and certainly today is that there's just no evidence to support that. It's a specific syndrome.

A That's correct.

Q But again in the chronic heavy user that's using it over a long period of time, there are some behaviours exhibited by people who do that that indicates in your opinion that we should be at least looking at that to see if there's a causal relationship to the chronic use?

A Well, yes. I think that's still valid because it is seen imperative motivation and loss of purposeful orientation towards long term activities and so on. It is seen in—in many chronic heavy users, but the question has always—continues to be is that due to cannabis use, or is it due to personality problems that preceded and contributed to the cannabis use, and I think there's still a need for ongoing careful studies of individuals and their course of change over time, including those who stopped using and those who continue using, because it would—I think it would be important to resolve once and for all, whether that is a consequence of chronic intoxication or whether it's a causal factor of chronic intoxication.

Q And so I think we—you said that we banished the term amotivational syndrome and use the term chronic intoxication or to see what the effects—

A Yes.

Q -- of chronic intoxication are?

A That—that was the view of the 1981 committee, and I think continues to be the—the predominant view.

Q So again, in terms of the great majority of marihuana users, the '95 percent that aren't the chronic user, we don't have any significant concern there, but in terms of the chronic user, we should be monitoring that and looking at that first?

A Yes.

Q Okay.

A There—there was one recent paper in which this comes up as a—a relatively minor observation in the course of an examination of the psychic features and performance of long-term users, and it just mentions that the chronic users themselves did complain of memory deficits and of loss of motivation, but it's not the focus of a paper, so I—

Q Okay. You—

A -- I don't think one can gain much from it.

Q You did say in this area at one point, unless I've got it down wrong, you've mentioned other drugs, stimulant drugs, alcohol, having a similar effect?

A Yes.

Q But they clear from the body quickly?

A Yes.

Q You mentioned that with marihuana, it doesn't clear as quickly—

A That's correct.

Q -- the psychoactive ingredients. Again, we've heard the evidence of the metabolites that can linger for long periods of time. You're not talking about—

A But those—those—

Q -- the metabolites?

A No, the metabolites are not active, not pharmacologically active. I'm talking about the—the half-life of the active material itself, of THC, which is considerably longer than that of most of the other drugs that we're talking about.

Q And again we're not—and we're talking about the chronic user and am I right in understanding that as part of that, there's a question of this being so regular that there's an ongoing accumulation of these ingredients, is that right?

A That's right.

Q Okay. Now, you then—I noted you to say it suggested a bigger effect on employment than alcohol, did I—

A A bigger effect?

Q On employment, loss of employment, interruption, these sorts of things, than the studies—

A Yes, that's right.

Q -- in relation to alcohol?

A Simply because the person who gets drunk can recover from that more quickly and therefore someone who abuses alcohol uses enough to be creating health problems or social problems or whatever, can continue to work because recovering enough the next morning to be able to go into work even though their performance may—may be less than optimal, but someone who feels draggy and can't be motivated to get up and go to work is more likely to be fired early.

Q Again we're talking about the chronic user?

A Yes.

Q And are there specific studies to deal with—that deal with that in terms of impact on the work place and things like that?

A There are but that's not an area which I have—which I have myself searched.

Q Okay. You've seen the ones that are specific to alcohol I take it from your experience with the alcohol?

A Yes. Yeah.

Q Okay. Do you know the names of—of the people who've done the major studies in that area?

A I would—I would be—in cannabis you mean?

Q Yes.

A Yeah, I—I don't really think that I can provide that. I can look that up, but I—it's not an area in which I have particular expertise or—

Q Would it be fair to say that again, because we're talking then about the five percent chronic user, you're not saying that this is a major impact on society, you're saying simply that the chronic user has more—

A Yes.

Q -- problems than the chronic alcohol user in terms of his or her employment?

A That's right.

Q Okay. So we're not—just to be clear, we're not suggesting that it's a factor that's resulting in masses of unemployment in Canada because people are smoking marihuana?

A No. I think the concern has been whether it's resulting in significant numbers of school dropouts who don't continue to develop their potential, and don't develop career opportunities that overwise could, and who therefore are—represent a problem to themselves and also possibly to others who might be dependent on them.

Q But we could focus on that particular group under this heading of one of the high risk groups, --

A Yes.

Q -- the adolescents as opposed to just the chronic heavy users generally, would that be fair?

A I—no, I think it would apply to adults too, if they become chronic heavy users, because the same considerations that apply to school performance would also apply to employment in industry or business or whatever. It—to be quite honest, I think you would get better information on that from someone who is a sociologist.

Q All right. Okay. And in that area, too, I think you said the studies haven't added much since '81, but there—and there is some evidence in later studies, animal studies of people recovering skills after they stop using?

A In—

Q Recovering after they stop using, if they stop being chronic users?

A Well, yes. What I mentioned was that—whether they recover or not appears to depend on the duration or dosage of the—of the period in which the drug is given.

Q How long they did it and how much they used?

A In our studies, we gave twenty milligrams per kilo, which was in—allowing for the difference between the rat and the human, --

Q Yes.

A -- which we estimated to be comparable to someone who would be smoking at least one or two cigarettes a day, and we gave it for three months which in human terms would be comparable to—see that's about an eighth of the rat's lifespan, so we're talking about probably about five to ten years.

Q Okay. The next heading was specific brain damage, and at an earlier time there were specific concerns about that. As I understand it, those concerns weren't born out, --

A That's right.

Q -- and haven't been born out by subsequent investigation, so we can say that that's not a major health risk, even in the chronic user?

A Well, what I said was that the type of brain damage which they were originally looking for, gross loss of brain cells, atrophy of different parts of the brain did not appear to be the case, and that the concern now was more with the long-term or permanent—potentially permanent alteration of microscopic features of cell structure that involved cell-to-cell contact and the synapse formation, the transmission of information between cells.

Q Right. I'm going to come to that, but as far as the original concerns about atrophy and so on, --

A Yeah, those—

Q -- we don't need to worry about that?

A -- have not been born out.

Q But on that other part, the synapse part, the—there's only—you only referred anyway to one study by a former student, Fehr (phonetic), unpublished that hasn't been replicated, correct?

A Yes, that's right. I raised that simply as one of the things that requires—

Q Yeah. And is—

A -- requires closer examination.

Q -- is that—is that really the only research on this—current research on this area?

A Well, that's not even current any longer.

Q Okay. From what time was that?

A That's—Dr. Fehr was a student of—a graduate student of mine in the late '70's.

Q Oh, I see, so this was done quite—

A This was blind, which was not continued unfortunately.

Q I see.

A We didn't have the opportunity to continue it, because that required facilities that we didn't have, or collaboration with someone who was more interested in something else.

Q And I think you said again, this was a—well, the indications are, anyway, this is something in the THC as opposed to the smoke?

A Yes, because that was produced by administration by routes other than smoking.

Q But no further studies have been done to see if there's any—anything to this?

A I have a vague recollection of one study in the past few years that attempted to look at synapses and didn't have any very clear-cut findings. I would have to look that up to—to try to see in greater detail what they found.

Q So—

A It's not a major topic in the literature.

Q So can we classify that into the group 2 then, that at least as far as the majority of users are concerned, it's a non-issue. We—we're not concerned with it as a major significant health risk?

A That's right. It's something which requires further study and would be pertinent only to regular heavy users.

Q And at the moment, even in relation to regular heavy users, we simply don't have the evidence?

A No, we don't. We don't have any evidence in humans.

Q All right. The next thing we get to the driving one, and the driving one, if I understand your evidence, the driving one is the one that is the most significant insofar as potential impact on other non-users, because the person is taking in a substance that impairs and is driving, and that can obviously hurt or cause harm to others in society, is that fair?

A Yes, I think that's fair.

Q It's—it's the most significant one—

A It's the most obvious one certainly.

Q -- of that type, isn't it?

A Yes.

Q I mean we—we've talked about the other high risk categories and so on, and they're adolescence, mentally ill, those sorts of categories. This one could be anybody who takes the stuff and drives?

A That's correct.

Q And it's the acute effects?

A That's right.

Q So again, it's different to many of these other things. We're not talking about the chronic heavy user?

A No, we're talking about—

Q We're talking about any type of user?

A That's right.

Q Okay. Now, Robbe is, as I understand it, the—not the last word but the—the most recent word if I can put it, on that—

A Yes.

Q -- topic, isn't it? And you've only had a chance to look a little at the Robbe study, you haven't had a chance to really thoroughly look at and digest it?

A That's correct.

Q What I've done is I've made some copies just of the—I'm going to try to get more copies of the entire publication, but just so that we have the basic stuff, I made copies of the—sort of conclusion parts of it.

MR. CONROY: I'll give you one of those, two for the Court, if we could just mark that as pages 168 to 177 of a book called Influence of Marihuana on Driving by H.W.J. Robbe, R-o-b-b-e.

THE COURT: Was that filed as an Exhibit?

MR. CONROY: I'd ask that that be the next Exhibit.

THE CLERK: Exhibit 37, Your Honour.

EXHIBIT 37 - EXCERPT FROM BOOK

MR. CONROY:

Q Now, I'm just going to take you to, if I could, I start at 269 and it's the topic Effects of THC on Driving Performance, and simply there's a paragraph and I won't go through it, results of the present studies which—a brief summary of what they actually did, fair enough?

A Yes.

Q There's then a reference to Drug Plasma Concentrations on Driving Performance and that part, as I understand it, was an effort to see if they could test afterwards to see how much THC was in the blood, blood plasma?

A Yes.

Q Then it goes on to the topic, Cannabis versus Alcohol and other Psychotropic Drugs, so there's a comparison to a summary of—of their work in terms of the comparisons to the effects of other drugs. and—and amongst those, it even includes such things as Valium, Atovan, fairly well-known prescribed drugs?

A Yes.

Q Okay. So we're talking there about not just alcohol but also a lot of these other types of drugs that are often prescribed and people may drive under the influence of, fair enough?

A Yes.

Q And then the—the next section immediately after that is Why are THC's Effects on Driving Performance Relatively Small, correct? And just from a heading we can see that the authors of this study, the results that they came up with were based on their study that the—the effects were small, fair enough?

A Yes.

Q And then some concluding remarks, followed then by a summary of the conclusions, page 178?

A Right.

Q All right? I want to go through this—the conclusions to some extent and maybe have you comment how this is different to the other studies and so on that you've looked at and referred to. In '81 it was Klonoff (phonetic) and I don't know if in '95, were there—were there others besides Robbe that were considered by the Committee in '95?

A No. The others were more studies which attempted to identify whether or not cannabis use was a significant contributory factor to accidents occurring in real life driving.

Q Right. And was Robbe discussed by the Committee in 1995?

A My recollection is no, I don't believe it was available.

Q So this would be a new—

A This is new, I believe.

Q Yes, okay. All right. Now, here the major conclusion—but first of all, the current users of marihuana prefer THC doses of about three hundred and that's micrograms per kilo, is it?

A That's correct.

Q To achieve the desired high. Now, I—I take it you're not—or are you—from the little bit of time that you did have to look at this, were you able to familiarize yourself with the discussion about that part of the study?

A Yes. I looked at the section which gave the basis of their choice, --

Q Yeah.

A -- and they did—they tested various doses to see what constituted a dose that a user would consider an appropriate dose to get a satisfactory high.

Q All right.

A And they concluded that three hundred micrograms per kilo was the—the sort of preferred dose—

Q And there was surprise?

A -- and then used one-third and two-thirds of that in addition.

Q They were surprised at that result, weren't they?

A I don't remember their being surprised. I didn't have a chance to read it enough detail to see that.

Q All right. It was a situation, though, of people coming in, volunteering to be involved in a test, and being asked how much marihuana they would like and all of them, or a lot of them asked for a larger amount than what was expected to my recollection. You don't remember that?

A Yes, they—

Q It was given to them for free?

A Yes. I believe—I can't recall, but I believe they did comment or said that it seemed that possibly previous driving studies had used too little.

Q All right. It then says next it's possible to safely study the effects of marihuana on driving on highways or city streets in the presence of other traffic, and I take it that's because that's what they did in the circumstances, and Klonoff did that, as well?

A Klonoff had—Klonoff had already done that.

Q Just before we—just come back to the three hundred. Can we convert that three hundred micrograms per kilogram to—to numbers of cigarettes roughly?

A Three hundred micrograms per kilo, we'll say the—the mythical average seventy kilo person, that would be—let me just get a calculator out. It will be better than doing it mentally. That would mean twenty-one milligrams. If the figure is correct that one may lose roughly half or up to half in sidestream smoke, that would mean that to deliver twenty-one milligrams, you would want to give forty-two milligrams and that would mean—now, they were using I believe—I think it said that their marihuana was a concentration of approximately two and a-half percent tetrahydro—THC content.

Q Let's assume that, yeah.

A So if that's the case, forty-two milligrams—let's see—that would be—that would be approximately two—roughly two cigarettes or a little less of the potency that they were using.

Q A reasonably high amount, isn't it?

A Yeah.

Q Okay. And so the—the hundred milligrams that was used—or micrograms per kilogram that was used in the highway driving, you remember commenting on that?

A No, I don't remember the comment on that.

Q Okay.

A That would be—that would be in their terms a rather low dose.

Q Yes.

A Do you remember the discussion that—were you able to read that part of the material that—that discussed how they came up with the hundred microgram per kilogram dose and—and why?

A No, I—

Q Okay.

A I read only the section—very brief section at the end which—in the discussion which explained how they

picked on the three hundred and then they used two doses below that, two hundred and one hundred in order to provide a picture of the connection between the dose and the effect.

Q All right. My understanding, and correct me if I'm wrong, is that a lot of marihuana smokers get sufficiently high, as far as they're concerned, on a fifty microgram per kilogram dose, and that some of this was born out in the studies by Moscovitz (phonetic) and problems of undivided or divided attention in driving?

A Yes. Moscovitz generally did use small—smaller doses than this and used rather sharply defined laboratory tests. He was looking at driving skills rather than driving, and was looking for quantifiable changes in performance, but he—my recollection is that he did point out that being able to measure a change in a driving skill was not necessarily the same as being able to show that that produced a—a significant impact on actual driving, and therefore he—he reviewed that work by others, rather than rely on his own work for the conclusions about the possible contribution of—

Q Certainly—

A -- cannabis to actual driving.

Q But the fifty micrograms were sufficient to get the subjects high on the marihuana?

A It would be—yes, that would be a—a mild effect, but measurable.

Q And so a hundred micrograms, double that, as I understand it, that was certainly effect—effective to give the subjects the—all the typical symptoms of smoking marihuana, in terms of heart rate and being speeded up and all those kinds of things, is that fair?

A Yes.

Q You'd expect that, wouldn't you?

A Yes.

Q Okay. All right. So a hundred microgram dose is an adequate dose in order to show the effects of consumption of marihuana for most users, would that be fair?

A No, no, I don’t think you could say that. It’s certainly enough to show that there are demonstrable effects, but I don’t think you could say that it’s enough to show effects for most purposes.

Q For the—for the occasional or moderate user, wouldn’t you think that even for many of them, just a few—

A Yes, for—

Q -- draws on the marihuana cigarette would be adequate to get them high?

A Well, oddly enough, no. It’s enough to show a change in—in heart rate, for example, but for naive users it, initially at least, it’s not enough to get them high, because it was well recognized in the early years with relatively weak preparations that you had to learn to recognize the high because in inexperienced users, anxiety often overrode it.

Q Let’s leave—let’s leave the naive or inexperienced aside for the moment and assume that they hopefully aren’t the ones who would do and experience something like that and get behind the wheel of a car. Let’s take the ordinary occasional user who might do that. I take it you’d agree with me that if that user smoked the complete hundred microgram marihuana joint, that that’s going to get them high sufficiently for us to test how they’re affected when they drive?

A Yes, probably so.

Q Okay. So it’s not too small a dose, is it?

A No, for the occasional user probably not.

Q Okay. The more experienced chronic user, you might need more?

A Yes.

Q Okay. All right. Let’s go back to the conclusions. The third one, "Marihuana smoking impairs fundamental road tracking ability with the degree of impairment increasing as a function of the consumed THC dose." That’s not surprising, is it?

A No, that—that fits with the laboratory studies as well.

Q And—and fundamental road tracking ability, as I understand it, is something to do with something called lateral sway, staying between the lines on the road?

A Yes.

Q Okay.

A It’s ability to keep on a straight line.

Q Right. But what the—

A Or to keep on a—on a—

Q -- the people measure is this lateral sway factor, isn’t it?

A That’s right.

Q Okay. Next, "Marihuana smoking which delivers THC up to three hundred micrograms per kilogram dose slightly impairs the ability to maintain a constant headway while following another car?"

A Yes.

Q Okay.

A It’s the ability to keep a—a fixed distance—

Q Behind the car?

A -- behind the car ahead.

Q Okay. Next, "A low THC dose, a hundred micrograms per kilogram, does not impair driving ability in urban traffic to the same extent as blood alcohol concentrations of .04 grams percent," am I—

A Yes.

Q -- is that grams percent?

A Yes.

Q And so you don’t have any difficulty with that conclusion, I take it? You don’t have any difficulty with that conclusion?

A Well, I don’t, since I haven’t really had a chance to read the book. I don’t know what findings that rests on. I did glance through—through the book yesterday and I was a bit puzzled because it seemed to me that at one point they were saying that they compared the results with—obtained with cannabis with the results that had been obtained in a different study with different subjects with alcohol, and if—if that is the case, then I would not be too happy with that.

Q That’s not the technique that one should use?

A No. If you’re going to make a comparison between drugs, you should be comparing the same—the two different drugs in the same subjects, so that differences between individuals are—are properly taken into account.

Q All right.

A And also one would need to know the—if these subjects were well experienced with—with cannabis and had acquired a—a level of tolerance to it that would not be the case in occasional users, what would -- what was the experience of the alcohol subjects with alcohol, so I—I would reserve judgment on that until I have a chance to see the—

Q See the whole thing. Okay. Next, "Drivers under the influence of marihuana tend to overestimate the adverse effects of the drug on their driving quality and compensate when they can, for example, by increasing effort to accomplish the task, increasing headway or slowing down or a combination of these." That’s an interesting finding, isn’t it?

A Yes, and that is consistent with an earlier finding in the literature by Dr. Smiley (phonetic), and I did discuss this with her in preparation for the Geneva meeting, and she said yes, that the—that cannabis and alcohol had both impaired performance on the driving tasks that she used, but that one difference was that the alcohol tended to make the drivers rather more reckless and aggressive, and therefore to take more chances, while the cannabis tended to make them more cautious and not to try to pass, for example, when there was a—a limited space in which to do so.

Q She was a member of the Committee, was she?

A No, she wasn’t. She was a consultant for someone who was writing part of the—part of the report.

Q Okay. All right. Next, "Drivers under the influence of alcohol tend to underestimate the adverse effects of the drug on their driving quality and do not invest compensat—compensatory effort." That seems to flow from what you've just said Dr. Smiley was saying?

A Yes. I did mention to her one study that I was familiar with in the literature from the 1960's by Drew—I believe it was Drew and Colquhoun that appeared in the British Medical Journal, concerning the effects of alcohol on driving ability in people of different personality types, and the rather remarkable thing that that study reported was that whether people became more reckless under alcohol or extremely cautious under alcohol, depended on whether they were basically extrovert or introverts in personality, that the extroverts tended to become reckless to increase their speed, to make very wide, poorly-controlled movements on the steering wheel, while the introverts tended to be extremely self-critical and to compensate for their recognized inability to drive accurately by slowing down, and refusing to do the drive if they were forced to speed up. And I asked her what she thought of that, and she—for reasons which I'm not really very clear on, she was not very ready to accept those findings. She had doubts about them and I really would—would welcome a chance to discuss with her why she questioned that, but if that study is valid, it would indicate that this statement is of—not of universal truth, but of limited truth.

Q Do you remember the name of that earlier study?

A Yes, the authors were Drew and Colquhoun, Col-qu-houn, and it was in the British Medical Journal, and if I recall correctly it was 1963.

Q All right. Okay. Next it says, "The maximum road tracking impairment after the highest THC dose, the hundred micrograms per kilogram, was within a range of effects produced by many commonly-used medicinal drugs and less than that associated with a blood alcohol concentration of .08 grams percent in previous studies employing the same test." As I understand that, it's saying that when they run the higher dose, the three hundred micrograms, that the effects were similar to things like Valium, Librium, Atovan, but less than what you'd have in somebody driving at what we have as our legal limit, .08?

A The problem I have with that is that for those other drugs the effect is also dose-dependent, --

Q Yes.

A -- so that you can't say the effect is less than with Valium. You need to say less than how much Valium.

Q Yes. So I—I can't judge the—

A Assuming they did that, you'd accept it, but that's a critical thing that one has to—

A That's something which one needs to know, yes.

Q Okay. All right.

A And also the same comment that I made before. They were comparing the three hundred microgram per kilo dose of THC with a blood alcohol level of .08 in different subjects. It was previous studies employing the same tests. I gathered, in fact, from reading—glance—really just glancing at that part in the book, that those studies were not done by themselves, but by others using this test.

Q The alcohol one?

A Yes.

Q Okay.

A So that would be another—another thing that I would want to reserve judgment on, because if they're not the same subjects and if they're not the same experimenters, I'm not sure how much reliance—within what range of—of reliability you can make this type of comparison.

Q Okay. So the better course of action would be to make sure that you get a chance to look at this whole thing before we're here next time so that—

A I would welcome that, yes.

Q All right. Okay. I'll just deal with the last one. "It's not possible to conclude anything about a driver's impairment on the basis of his or her plasma concentrations of THC, and THC-COOH, determined in a single sample." Am I understanding that correctly to mean that a blood sample taken from a suspected impaired marihuana driver won't tell us the concentration of the THC in the blood?

A No. No, it's saying that measuring the concentration of the blood—in the blood—

Q Yes.

A -- in a single sample taken at an unspecified time, --

Q Yes.

A -- doesn't allow you to say what the effect on performance will be.

Q Okay. And it's—do we know if you could simply by blood sample, if the officer say stops a person driving and under appropriate circumstances can have—take a blood sample, would that be—or take more than one blood sample, I understand, would you be able to tell what—

A Yes, this is—this is—this is what this point is getting to.

Q You'd need to take more than one?

A You would need to take more than one, because you would need to know how it relates to the time course of onset and disappearance of action.

This point, I should mention, was made previously in the 1970's by a Swiss group that was investigating the effects of cannabis on—on road performance and simulator performance, and they came to the same conclusion, that a single—a single blood measurement does not permit you to estimate the degree of impairment in the same way that they felt that you could with a single measurement of alcohol. I think, though, the view that you can do that with a single measurement of alcohol has changed since then.

I think it's also recognized that you—in order to make a prediction, you really need more than just that single measurement. You need to know when it was measured in relation to the time of drinking and the time of the performance, because of the existence of a phenomenon that's known as acute tolerance, which means that you change in sensitivity to the level of alcohol or of THC with the passage of time. And therefore, a single measurement at an unspecified times doesn't really permit you to make an accurate prediction of what the impairment will be.

Q So if you took two blood samples at a certain time period in between, is that going to help at all, or are you still stuck with the same—

A Not a great deal. What you really would like would be three, four or five samples tracing the rise and fall of the levels so that you know where on the course of

adaptation the subject is. The law sets .08 as a prohibited level, more on statistical grounds than on grounds of observation in the individual, and the problem has been that there are a small but significant number of reports describing individuals with much higher blood levels who in the eyes of experienced observers were not grossly impaired. And this raises some concern about the need for a better way of relating the blood level, not only of cannabis but of alcohol, and probably of other drugs as well.

Q Alcohol as well. But the—in—rather than me trying to explain this, I'll have you do this, because I'm sure you can do it better than I. The—there's a difference, isn't there, in terms of what happens when you consume alcohol, in terms of the—the curve if you can put, as opposed to cannabis?

A Yes.

Q And the cannabis—

A One—I'm sorry, you say as—as opposed to cannabis?

Q Or compared to cannabis?

A Well, I think the same applies to both of them.

Q No, but in terms of—if somebody consumes cannabis, what is the—the curve—how does the curve operate, does it operate much the same way, in terms of the—the effects of the drug in the system over time and levelling off and so on, is it much the same effect as with alcohol?

A It's comparable. It differs somewhat in—in time.

Q Okay.

A The—to begin with, the fact that cannabis is smoked means that it has a faster onset. When alcohol is drunk, it takes a longer time for the alcohol concentration to build up in the blood.

Q All right. But isn't there a—a more rapid dropping off—

A Yes.

Q -- from cannabis?

A Yes, there is.

Q And so it may come in quicker, but it drops off quicker, doesn't it?

A It drops off faster, too, yes.

Q And then it levels off and stays at a certain level?

A That's right.

Q Okay.

A And that is rather—that is different from the typical time course after alcohol.

Q And it's something called a dose response curve or something like that?

A No, that's a time—a time concentration curve.

Q Yes?

A A dose response curve, the usual way would be to say at a fixed time after administration, so that you are at a comparable point on that time curve. At a fixed time after administration, you measure the effect after having given one, two, three, four or more different doses, so that you can plot the effect at that constant time against the dose that was given, and that is the way in which most dose response curves are constructed, but the problem is that they apply to a particular time, and the dose response curve can be shifted towards greater or lesser effect according to the time after the dose.

Q Okay. Now, when we talk about the driving part of it, of course we're going—we're adding in a whole additional factor, aren't we? We're not talking about somebody—somebody who simply possesses it and uses it, we're talking about this extra factor of the person possessing it, using it and then going out and getting involved in some type of conduct that could impact on others?

A That's correct.

Q And the concern there primarily is the acute effects on psychomotor performance?

A Yes.

Q And so a social policy that prohibits a person from driving while their ability to do so is impaired by alcohol

or cannabis or any other drug, specifically targets the concern that you have in relation to intoxication by cannabis, isn't that right?

A Yes, that's true.

Q And if you—so—so a policeman, for example, who observes erratic driving, that erratic driving could be caused by any number of factors, fair enough?

A Yes.

Q And if the policeman then stops the vehicle and observes the person to start off with, there may be, depending on whether the person smoked cannabis recently, some smell, reddening of the eyes, things like that that the officer might observe, fair enough, to start off with?

A Yeah. Those would be—they would be suggestive. They—they could arouse suspicion. They wouldn't be sufficient to prove the—

Q No, no, I'm—I've got more facts to add to—and after that, though, an officer could ask a person to step out of the vehicle and to have that person go through certain psychomotor skill-type tests, in order to see what their ability, their psychomotor skills are like at that particular point in time, such as the finger—

A As is—as is done when a police officer stops someone suspected of driving while under the influence of alcohol.

Q In order to try and see if their suspicion can be elevated to a level where they have concerns—

A Yes.

Q -- that the person's ability to drive is impaired?

A Yes.

Q You should—you can do that with cannabis as well, can't you?

A Yes, I can't see any reason why not.

Q Okay. I read and I've misplaced a—a newspaper clipping about an approach in Germany where they're measuring factors, but I'm told that it doesn't help us in terms of the amount of THC in the blood. It simply

measures or indicates that you've had some contact with it, and how long ago it was. Are you familiar with any processes that they're trying to develop like the breathalyzer machine that we have for alcohol, for cannabis?

A The—the only one that I have seen that—that appears to be relatively feasible is the measurement of THC in saliva, because that would represent THC from smoke that had been inhaled fairly recently, and a positive test, and a measurable quantity in the saliva would be presumably a better indication of being under the influence at the time than is the finding of metabolites in the urine, which may have nothing to do with the—

Q It may be weeks for—for metabolites?

A Or days certainly.

Q All right. Well, that's interesting. The saliva test. Would you take a small—just need a small amount of saliva, is this a—like taking one of these pieces of paper that changes different colours which tells you how much there is, or what are we talking about here?

A No. It's—a person has to spit into a bottle and—and then the saliva is analyzed usually by gas liquid chromatography and mass spectrography, and that is highly precise and very sensitive for identifying the individual compounds and measuring the exact amounts present.

Q So as long as an officer had reasonable grounds to demand a saliva sample, they could put it through a spectrometer?

A A mass—

Q Is that the right—a mass spectros—

MR. DOHM: Assuming the law changed rather drastically.

THE COURT: I'm not sure the police would relish the idea of inviting them to spit.

MR. CONROY: Well, whether the police—

THE COURT: It may lead to unfortunate consequences.

MR. CONROY: Well, sometimes in law enforcement they have to balance these things with their objectives.

Q But it doesn't sound that difficult to—to take a sample, and if you've got the machine to—to do the analysis, you could determine what the level of THC in that person's blood is?

A No, not in the blood. That's in the—that's in the saliva.

Q In the system—okay, in the saliva which—from which you could find out how much is in the person's system in—

A No.

Q -- terms of toxic—

A No. The—the reasoning is not that. The reasoning is simply that it's known from laboratory studies that it disappears from the saliva within a reasonably short time after the smoking.

Q I see.

A So that if you find it in the saliva, --

Q Recent—

A -- it's an indication that the person has smoked recently and is therefore presumably still under the influence of THC present in the blood.

Q So, if I understand you, that would show the recency of the consumption—

A That's right.

Q -- and you could then take a blood sample or several blood samples?

A Yes, that—that would be the—from a scientific point of view, that would be the ideal.

Q And that should then give us some idea whether the person's ability to drive is impaired?

A Yes, I think one would probably find a much better correlation of the blood level with the ability to drive properly, if it could be combined with saliva testing. It would prove that the THC in the blood was of relatively recent ingestion.

Q All right.

THE COURT: When you say that the presence of THC in the saliva disappears very quickly, does it disappear before the THC reaches the brain and has the effects?

A No. THC is—I think many people now know is highly soluble in fatty materials. It's—it is not readily water-soluble. It's highly soluble in fat. That means that it has the ability to cross cell membranes which are to a large extent fat in—in composition, very quickly.

Now, when it gets into the blood by crossing the cell membranes of the lung and the capillaries in the lung, it's then carried to all organs in proportion to their relative amounts of blood flow, and the brain has a very high rate of blood flow, so that it's delivered very promptly to the brain after it gets into the blood, and being very fat soluble, it can rapidly cross through the cell membranes into the brain, but then it also passes into other tissues, not as rapidly, but reasonably rapidly, so that the blood level drops off and it doesn't tell you how much is still in the brain. That's the problem. That's why it's been difficult to correlate the level in the blood with the effect on performance, unless it is of very recent origin.

MR. CONROY:

Q All right. So recent origin, you could go through the process that we've described a moment ago, but if it's—if it's not of recent origin, --

A It doesn't help you.

Q -- you—and you'd be left then really with the blood sampling process as being the only way to try and determine the level in the blood?

A That's—well, you'd be left with a problem of trying to correlate the blood level, the measured blood level with the affect on performance, which as they point out is not very good.

Q Yes.

A Okay. It's—if I may illustrate by analogy with a different drug, for inducing anaesthesia for surgery, or for performing very minor operations under a short-lasting anaesthesia, it's common to give a very fat-soluble barbiturate, like Pentholthiopental (phonetic) and it's injected, it induces sleep within seconds, but then it wears off extremely rapidly while there's still an awful lot of it present in the body, because it goes into the brain and then the level falls off very rapidly as it goes

into other tissues, and then it starts to move out of the brain again. So that it's a question of the distribution, the relative distribution between the brain, the blood, and other tissues, and that in turn reflects the time that has elapsed since the drug was put into the system.

Q Okay. And all of the effects that we're talking about here in particular are again dose-related?

A Yes.

Q It depends on just how much the person has taken—

A That's right.

Q -- in terms of the impact? Okay. There was some reference I had to small doses that you talked about in terms of Klonoff versus Robbe, but you couldn't remember the doses in Klonoff. You haven't had a chance to—

A No, I—I don't recall those doses. I don't believe they're in the 1981 report. I know they were in the 1981 accompanying volume of background papers, but I don't think the details were in the summary report, so that I can certainly get that information from the background on them.

Q Your recollection though was that the doses were comparable to Robbe?

A Yes, they were—they were not out of line with those of Robbe.

Q All right. Would you then agree with this statement. First of all—and I haven't gone over with you the studies to do with traffic accidents and fatalities, but would you agree that there's no convincing evidence at this point that marihuana contributes substantially to traffic accidents and fatalities?

A Yes, I would say that's correct in the sense that what these studies show is that it has the capability to impair performance, but we don't yet have clear evidence of how much in practice it actually contributes to accidents on highways.

Q Would you agree also with this, that at some doses marihuana affects perceptions and psychomotor performance which could impair driving ability?

A Yes.

Q But also that in driving studies using typical social doses, that marihuana produces little or no car handling impairment?

A I—I would avoid such a statement because I'm not quite sure what little means. The no is wrong.

Q Okay.

A Little—little depends on what you're expecting or what you're willing to accept but—

Q All right.

A -- this study shows that there is measurable and statistically significant effects of so-called social doses on—on the ability to do some features of driving. What this didn't—what this summary doesn't mention and which I think one needs to know, is effects on attention, on level of monitoring of other traffic, of pedestrians or cars coming from the side into potential collision of thinking of other things, rather than paying attention to the road and so on.

Q It recommends things like that, doesn't it, in the Summary of Recommendations, where it suggests other types of studies to—to check for those kinds of things? I'm thinking for example of the—

A Yes.

Q -- fifth one down?

A The fifth one down, right. Exactly.

Q Okay. Let me read this whole statement though to you, because I want to see to what extent you disagree or agree with this. I should have—there was an added part to that sentence. "However, in driving studies using typical social doses, marihuana produces little or no car-handling impairment, consistently less than low to moderate doses of alcohol," would you agree with that?

A Well, no. As I said before, --

Q It depends on—

A -- it depends at how—

Q -- looking at—

A -- the—yes, I really need to see how the alcohol studies were done, on which subjects, by whom, when.

Q Okay.

A Because I—the validity of such a comparison rests on their being done in the same or in very closely comparable subjects by the same techniques, by the same observers. I'm reluctant to say that I agree or disagree with it until I have a chance to see what that—what that information is based—what that statement is based on.

Q You would—you'd agree with this. Based on the study and doctor—the information from Dr. Smiley, that, "While alcohol increases the likelihood that subjects will engage in risky driving practices, marihuana tends to make subjects more careful?"

A That—that appears to be consistent with other—other findings.

Q Okay. And how about this. "Surveys of fatally injured drivers show that in the vast majority of cases where THC is detected in the blood, alcohol is detected as well?"

A Yes, that's true in the—certainly in the majority of cases.

Q And finally, "For some individuals, marihuana may play a role in bad driving, but the overall rate of highway accidents appears to be unaffected by marihuana's widespread use in society."

A The second half of the statement I—again I cannot—I cannot say yes or no, because one really needs to know—there are studies as I mentioned in earlier testimony that there are studies which suggest that THC in the—in the blood or in the plasma is found in a significant number of drivers stopped for impaired driving or accidents who were—had no alcohol present, and unless one has a way of saying how many that represents of a total number of accidents, and how it has changed in—in relation to the change in cannabis consumption, it would be impossible to justify such a statement.

Q Okay. All right.

THE COURT: It is 4:30.

MR. CONROY: Well, --

THE COURT: If you're about to go onto a new topic, --

MR. CONROY: -- yes, I was. I was going to—

THE COURT: Thank you very much, sir. It looks like we will be having you back. I don't know when.

MR. CONROY: Your Honour, can I just hand you up some things so that we have the amended memorandum of agreement and the Narcotic Control Act Regulations, and that's amended only in the sense that I've incorporated into it the—some references in quotations from the R.G.R. McDonald Tobacco case and Heywood, both Supreme Court of Canada cases, Heywood being on the over-breath principle.

The other thing I have for you is a miscellaneous book of authorities which is basically health legislation and some tobacco legislation. So I will have to add a—a case book, I guess, that adds those cases to the other case books, and actually there is another case that I have referred to in the new argument and that's Nova Scotia Pharmaceutical Society, but I'll provide that with the other ones, as well.

The other document, and this one I suppose we should mark, is simply the Hansard Record or when marihuana was put onto the schedule. And I have that, my friends have that. I had two of that, if we could make that the next Exhibit?

THE CLERK: That's Exhibit 38, Your Honour.

EXHIBIT 38 - HANSARD RECORD

MR. CONROY: Now, the only other things that I would have, and I could either give them to you now or next time is we do have copies of some of the books that are part of our Brandeis brief and just the duplicates we have two copies of—do you already have (indiscernible) --

MR. DOHM: We have your office copy, the one with your stamp on it, so you can (indiscernible) --

MR. CONROY: So we have a copy of that.

THE COURT: Do you wish that marked as an Exhibit?

MR. CONROY: Well, it's part of the Brandeis brief, which I guess—

THE COURT: Is the brief marked as an Exhibit?

MR. CONROY: -- we did mark some—yes, I—it's part of it, so it's listed in our index actually already, so it really should be part of Exhibit 18.

THE COURT: Do you know what number it is on the—

MR. CONROY: Yes, that's 18(18.)

THE COURT: All right. So it should be marked Exhibit 18(18) then.

THE CLERK: I'm sorry, Your Honour?

THE COURT: 18. It's Exhibit 18 and then in brackets (18).

THE CLERK: Yes, Your Honour. It's got 21 -- Exhibit 18 has got 21 tabs in it.

THE COURT: All right. This is tab 18 of—of Exhibit 18.

MR. CONROY: And I believe we gave you already—

THE CLERK: (Indiscernible) --

THE COURT: No, it's a blank.

MR. CONROY: -- a new (indiscernible) book, and I think -- behind the additional—

THE COURT: Dr. Kalant, you're most welcome to step down and depart if you wish. You don't have to sit through this.

A Thank you very much, Your Honour.

(WITNESS STOOD DOWN)

MR. CONROY: The Cannabis Cart from Ledeign (phonetic). I don't believe I've given you that yet, and that's part of the Exhibit 13.

MR. DOHM: Has Your Honour been in touch with Mr. Justice Creaver lately?

THE COURT: Who?

MR. DOHM: Mr. Justice Creaver?

THE COURT: No.

MR. DOHM: You both may find some comfort in the tasks that you're undertaking.

THE CLERK: I'm sorry, I don't understand. This is 18?

MR. CONROY: Yes.

THE COURT: Are you telling me this is—there's a parallel case going on in Quebec?

MR. DOHM: No, that's a—the blood inquiry that's been going on for years.

THE COURT: Oh, right.

THE CLERK: And that will be 18(19). And this is ...

(COURTROOM RECORDING EQUIPMENT MALFUNCTIONS)

THE COURT: There may be some bad guys in that case which may make it rewarding.

(DISCUSSION REGARDING DOCUMENTS)

MR. CONROY: All right. I think that's all we had for you at this point, Your Honour.

THE COURT: All right. We're adjourning then.

MR. CONROY: March 5th, 9:30 to fix a date.

THE COURT: All right. Mr. Caine, March 5th, 1997, 9:30 courtroom 1.

MR. DOHM: Thank you.

(PROCEEDINGS ADJOURNED TO 1997 MARCH 05 at 9:30 a.m.)