File No. 65381

C A N A D A

IN THE PROVINCIAL COURT OF BRITISH COLUMBIA

(BEFORE THE HONOURABLE JUDGE F. HOWARD)

SURREY, B.C.

1997 JANUARY 30

REGINA

V

VICTOR EUGENE CAINE

PROCEEDINGS AT

CHARTER APPLICATION

APPEARANCES:

T. DOHM, A. CHAN, M. HEWITT for the Crown

J. CONROY, P. SMITH GANDER for the Defence

G. LANDRY Court Recorder

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NO. DESCRIPTION

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1997 JANUARY 30

(PROCEEDINGS RECONVENED PURSUANT TO ADJOURNMENT)

MR. DOHM: Recalling Regina v. Caine, Your Honour. Dr. Kalant.

DR. HAROLD KALANT, recalled, testifies as follows:

THE COURT: Yes, sir, you are still under oath, you understand that?

A Yes, I do, Your Honour.

THE COURT: Thank you.

EXAMINATION IN CHIEF BY MR. DOHM continuing:

Q Doctor, let's get now towards the report of the ARFWHO committee and the report of the meeting of 1981. Just before we go into that, there has been a later meeting of that committee?

A Yes, there is an updated report currently in preparation.

Q And that will reflect work done to what period of time?

A Essentially up to 1995.

Q Back to the 1991 report, firstly, can you tell us how cannabis should be classified as a pharmacologist, how would you classify cannabis?

A It's not entirely clear, because cannabis has actions which are somewhat different from those of any other well-recognized class of drugs, but it depends to a large extent on the dose. At the doses that are typically used by most users for social purposes, it's a mild sedative similar in some ways to the effects of alcohol and similar drugs. It's not identical to them. It differs in some respects, but probably it's closer to that than to any other category. In very high doses, it can produce—

Q Excuse me a minute, please—

MR. CONROY: I'm just having a little bit of trouble hearing.

A I'm sorry.

MR. DOHM: Perhaps I will resort to something that has worked in the past and I'll move further away from the witness.

THE COURT: I'm not sure that that microphone amplifies anything. It may just be recording.

MR. CONROY: I'll pull this out of your way.

MR. DOHM: That's fine there. That won't bother me at all.

MR. CONROY: Okay.

MR. DOHM:

Q I'm sorry, before I stopped you there, Doctor, you were telling us that at normal doses the effect of cannabis is that of a mild sedative or a sedative?

A Yes, that's correct. It produces relaxation and elevation of mood, feelings of greater comfort and pleasure and it can also produce at the later stage of the effect drowsiness and it can enhance the sedative effects of alcohol and similar sedative drugs. In high doses it can produce hallucinations or altered perception and for that reason, some books classify it with hallucinogens, but I think it's important to make clear that that's only true at very high doses. It is not hallucinogenic in the levels that are normally used.

Q Thank you. Now, in the committee work, you arrived and for the sake of reference here, looking at page 3 of the ARFWHO report that is found in Exhibit 5 of the—which tab number was that?

THE COURT: Three?

MR. DOHM: 5-3?

MR. CONROY: Yes.

THE COURT: The Crown's brief, Volume 1, tab 3?

MR. DOHM: Tab 1 is where—

A VOICE: I think it's tab 1.

A VOICE: I think it's tab 1.

MR. DOHM: -- it appears, Your Honour. Tab 1 is where it appears in the—

THE COURT: Of what book?

MR. DOHM: Of the first volume of the Crown's brief.

THE COURT: Gotcha.

MR. DOHM:

Q Doctor, you defined—or the committee defined the term adverse effect as being one which may be considered to occur when such use produces impairment of an individual's biological behavioural or social function, is that correct?

A That's correct.

Q Was there a contextual reference to adverse effect?

A I'm afraid I don't understand your—

Q Does the implication of the phrase adverse effect depend upon the context in which it is being used?

A Ahh. Yes, it does, in the sense that as pointed out in that paragraph, there can be disagreement about whether an effect should be called adverse or not, and I think it is quite clear that something which alters perception may have no adverse consequences if a person is simply sitting quietly and enjoying music or whatever; whereas the same effect could be adverse if a person were driving a car, so that the impairment of function does necessarily relate to what a person is doing, what the circumstances are and so on.

Q You also defined intoxication and toxicity?

A We attempted to define them, but recognized that it was not always possible to differentiate them. Intoxication ordinarily refers to the constellation of effects that a person experiences when taking the drug in the usual way for the usual purpose; whereas toxicity usually refers to unanticipated and undesired harmful effects to the extent that you can separate them. That's the meaning, but sometimes for obvious reasons they can't be separated because intoxication can result in toxicity.

Q The committee also—and when I say committee for the next little while I'll be referring to the Addiction Research Foundation World Health Organization committee.

A Yes.

Q The committee also defined acute to refer to single doses and their effects?

A That's correct.

Q Or to reactions or responses on single occasions of brief duration?

A That's right.

Q Did you have the same level of precision with chronic?

A No. Chronic is used in different senses by different users. I don't mean users of the drug, I mean users of the term. In animal experiments, for example, chronic can mean as few as five or six administrations. Clinicians ordinarily use chronic to mean long-term, months or years and there's a convenient term, subacute or another subchronic that attempt to indicate various stages of difference between the acute and the clearly chronic, but they are not widely used.

Q Did you have success in finding the term rates of use or coming to agreement on what rates of use are?

A No. The trouble is that occasional, frequent and heavy moderate are all judgmental terms in a sense, and it's better, if possible, to avoid those terms and simply specify frequency and amount in terms of actual numbers.

Q Is that what the committee did eventually when they had their meeting and the report was prepared?

A They attempted to. Unfortunately, habits often die hard and the term chronic use does come up frequently without specification but where possible, attempts were made to relate effects to known durations and amounts of use.

Q The next heading on page 4 of that report is General Toxicity of Cannabis Preparations. What can you tell the Court about the general toxicity of cannabis preparations?

A The term general toxicity usually refers to the production of organ damage or life-threatening functional change and in general, I think one can say that the toxicity of cannabis as measured in terms of the

dose required to produce death, for example, which is a standard definition of toxicity for many drugs, such definitions would say that it is not a—not a severely toxic drug. There are no known deaths from cannabis alone and in animals one can cause death with enormous doses, but humans—in human experience there is no such toxicity because people don't use doses of the order that are required to produce that in animals.

Q There are from your answer though, I understand that there are lesser levels of toxicity than lethality, being those which can have an effect on the organism?

A Yes. And most of the committee's report is devoted to consideration of special toxicity in the sense of adverse effects on specific organ systems or functions, rather than on life or death.

Q That takes us to page 7 or reference cite. What is the—were the findings of respiratory toxicity in animals?

A At that time there was not very much experimental work on respiratory toxicity in animals. What was well-recognized was that single doses of cannabis can decrease the frequency and the depth of respiration much as other depressive drugs do and the effect of cannabis was at least additive with the similar effects of other drugs such as alcohol, barbiturates, general anaesthetics and so on. However, the studies on other characterizations of pulmonary pathology were not very extensive. There was some evidence that large amounts of cannabis smoke administered to animals could produce inflammation in the small airways and some changes in the lungs in terms of inflammatory cells collecting in parts of the lungs. That was not given a great deal of attention by the committee because they—again, the parameters of exposure of animals were not terribly clear in terms of how they related to human exposure.

Q Has that situation changed in the science since 1981?

A Yes, it has changed, not in the sense of animal studies but of much more refined studies in humans and perhaps you—I don't know if you want me to go on to the human studies or not at this point.

Q Well, you can certainly do that. What has been learned since 1981 with respect to respiratory effects in humans?

A There has been a continuing series of studies by the group headed by Dr. Tashkin in California, very sophisticated studies with quite refined functional tests and one similar study by Bloom's group in England somewhat later. They have not coincided completely in terms of their findings, but in general there has been very good agreement between them, and what they have shown is that chronic heavy use and by heavy in this case they meant essentially daily use, gives rise to inflammation of the what are called the proximal bronchials, that is the larger branches rather than the very fine terminal branches that lead right into the lung tissue.

Q Proximal are those closer to the mouth.

A Closer to the mouth.

Q And the—

A Distal are the ones—

Q -- distal are those furthest away.

A Closer to the final branching in the lungs, so the more proximal tubes have shown inflammatory changes, excessive production of mucous, alteration of the inflammatory cells, that is the cells that normally scavenge foreign material out of the bronchial tubes and the alveoli, the small air sacs in the lungs, and there have been changes in resistance of air flow through these tubes. The—probably the best term to use is a chronic bronchitis with reduced efficiency of air movement through the bronchial system to the lungs.

On the other hand, there has not been evidence, it has been looked for and has been shown not to occur, there is not evidence of impaired diffusion of gases across the surface of the lung into the fine blood vessels. This was in the 1981 report, this was believed to be one of the functional consequences. That has been shown not to be the case now.

The alterations in the inflammatory cells has received a good deal of attention and they have been shown, for example, to have alterations in the nucleic acid which is the chemical constituent of the nuclei that translates into function of these cells. These are changes which resemble those produced by tobacco smoke and are known to be precursors or at least pre-precancerous changes, so that there is some possibility still supported by this evidence that chronic administration of cannabis may give rise to malignant change in the bronchi of the lungs.

Q Has there been studies done, has there been a development in the science which would tell us what the cause of those apparent precancerous cellular changes is? Is it smoke, is it active ingredient in the substance that one would—

A No, that can be answered in the sense that it seems to be fairly clearly the smoke, polycyclichydrocarbons similar to those that occur in tobacco smoke and which produce the inflammatory and precancerous changes in the airways and the lungs. The tetrahydrocannabinol, the active, the psychoactive component of cannabis does not appear to be involved in the production of the precancerous changes or the inflammatory changes.

Q I'd like to just deviate here for a moment. Does there exist data which compares one to the other the risks of cancer between a tobacco smoker and a cannabis smoker?

A No, not specifically with respect to the risks of cancer, because there are not yet data which permit that type of assessment for cannabis. In terms of the inflammatory changes in the airways, yes, because the studies that I referred to have made quite a major point of including as closely as possibly matched groups of tobacco only smokers, of marihuana only smokers, of combined tobacco and marihuana smokers and of non-smokers, so that these studies do give some basis for assessing the relative contributions of the two types of smoke to the production problems and what is clear is that the patterns of change produced by tobacco smoke and by cannabis smoke are not identical but they're additive. In all of the tests that have been examined, for example, the effect of the combined marihuana plus tobacco smoke was greater than the effect of either the marihuana or the tobacco alone and the reason why one cannot draw similar conclusions about the risk of cancer is that the data on which that risk is assessed for tobacco are of a very different type from the immediate clinical study that I've described. Those are public health, based on public health statistics in which one can assess the rates of cancer production according to the duration and the amount of smoking of tobacco and such data accumulated over twenty, twenty-five years or more, in fact over probably several generations of tobacco smokers have been the basis for the conclusions about the risk of cancer production in tobacco smoking and such data simply aren't available yet, at least, for cannabis smoking.

Q And when would one expect those data to become available for cannabis smokers?

A I can only offer a personal opinion here. I think it will be probably—

Q Perhaps you shouldn't.

MR. CONROY: I don't mind.

MR. DOHM:

Q Let's try to stay within your qualifications, Doctor.

MR. CONROY: I'm not objecting.

A I'm sorry. I didn't say—

THE COURT: I think on this issue no one's going to worry. When do you think it might be available?

A I think it will be at least thirty or forty years, for the following reasons. It depends upon the size of the population which is being observed and how accurate the data are concerning the extent of use, in terms of intensity and duration of use. With a legal drug such as tobacco, it's much easier to get such data. With illicit drugs, it's unlikely that one can accumulate such data for the simple reason that patients obviously—or I shouldn't say patients, users are obviously reluctant to provide detailed information if it may be incriminating and also at current levels of use, cannabis is not used nearly as widely as tobacco and therefore it will take a much longer time to accumulate the data. For tobacco, as I said, it took twenty, twenty-five years of accumulation of such data and given the fact that the information on cannabis is much more limited, it will probably take correspondingly longer.

Q The next item in the report is on page 9 and that deals with cardiovascular toxicity in experimental animals. What was the results of the committee's work on that, Doctor? Page 8, excuse me.

A The committee came to the conclusion that there was really minimal evidence to suggest any cardiovascular risk. What is recognized is an acute effect of two kinds. Cannabis tends to—the smoking of cannabis tends to increase the heart rate and therefore in theory someone who has impairment of coronary circulation could have—could experience a risk of heart attack because of an

increase in heart rate elevating the demand for oxygen while the coronary circulation is unable to supply that extra demand. But that's a purely theoretical or a hypothetical suggestion because there were no data to describe cases in which that had actually occurred.

The other general effect that is recognized is that cannabis acutely tends to lower the blood pressure by producing some dilatation of the blood vessels in the limbs, so that if a person stands up suddenly, the blood pressure falls. This is called orthostatic hypotension. It means simply low blood pressure on standing up suddenly. And that can produce dizziness.

If the user remains seated, however, or is lying down, then this has no functional—no adverse consequences.

Q Now, you were dealing then with humans, you'd gone from experimental animals into humans?

A Yes. Experimental animals show the increased heart rate which is accompanied by an increased work load because of an increased output of blood attributable to the increased rate. There is mention in the animal section of a finding which has subsequently been confirmed in humans, there have been a couple of studies in humans confirming the increased blood flow through the large vessels of the brain. It should be pointed out this is generally considered an interesting finding but it's impossible to say whether it's beneficial or detrimental. The way it's studied in humans is to give a tracer which can be combined with a PET scan or a single positron emission scanning of the brain and the tracer is injected intravenously and then as long as it remains in the circulation you can measure the tracer isotope output, at least the emission from the isotope, over different parts of the brain and thus show an increased flow of blood through those parts of the brain, and that occurs together with the psychoactive effects. It is—in the case of other drugs it is taken to mean that the blood is going through the large vessels and shunting back to the veins, rather than going through the brain tissue itself, but it really—one can use it only as an index of the drug activity but it's impossible yet to attribute either beneficial or harmful effects to that.

Q The next topic dealt with—

THE COURT: Could I just ask one question—

MR. DOHM: Certainly.

THE COURT: -- on this one? Do we know whether these effects are a product of the THC content or the smoking process and the loss of oxygen—

A No, that seems to be attributable to the THC effect itself because it can be seen either with smoking or with administration of the drug by other routes.

MR. DOHM:

Q Have there been—

THE COURT: The same—the same—

MR. DOHM: Sorry.

THE COURT: The same rates of effect?

A Yes, similar effects.

MR. DOHM:

Q Has there been any change in the science on this since 1981, Doctor?

A In terms of cardiovascular?

Q Yes.

A No. The only major change has been the use of these tracers for studying cerebral flow. There has been no further evidence to implicate cannabis in terms of cardiac damage.

Q The next topic that I would like to direct your attention to appears on page 9 of the report under growth and body weight and the conclusion of the committee was that as of 1981 there was little or no information on possible long-term effects of cannabis on growth, development and maturation of humans, is that right?

A Yes. That was the conclusion then.

Q That has changed?

A That has changed. There have been contradictory findings in various studies, but perhaps the best longitudinal studies, that is those which follow subjects over a period of years and re-examine them, relate to the possible—I may be jumping a topic here, but I think this is the most appropriate place to bring it, relate to the effects of prenatal exposure, that is the fetus exposed in the uterus to cannabis smoked by the mother and there have been a number of studies showing that babies born to mothers who have used

cannabis through substantial part of the pregnancy are small for their birth age, that is for their gestational age, the length of time that they've been developing in the uterus. That has been shown in several studies and they, according to perhaps the largest of these studies, that by Freed et al in Ottawa, the infants are small, hyperirritable, don't feed as well after birth, but they recover over the next six months or so and by a year they're back to normal size for—size for age in terms of body weight and growth. The animal studies have consistently shown that high doses of cannabis do inhibit appetite and therefore impair growth over short periods of time, but there is a good possibility that tolerance develops to that effect, as it does to the similar effect of morphine, for example, and long terms studies therefore would be needed and have not been carried out in this -- no, I must retract that. There has been one good long term study by the toxicology group in Arkansas which showed that in—after a year's exposure, there was no appreciable impairment of the growth or development of animals exposed regularly to cannabis, so the implication must be that there was tolerance developed to the initial inhibitory effect on appetite.

Q Have there been studies done on the additive effects of the use of cannabis with other drugs at the prenatal stage?

A Yes. The same studies that I referred to have shown that the effects of cannabis are similar to those of the effects of tobacco smoking. It -- the evidence is suggestive of an additive effect of the two. It's more difficult to state with certainty that that is the case compared to the respiratory studies in which the populations are well-matched. In the prenatal studies, the matching is more difficult because many of the mothers who used cannabis also used other drugs for which good controls are not as easy to obtain.

THE COURT: Were they controlled for whether or not they smoked tobacco—

A Yeah, but they were controlled with respect to smoking.

THE COURT: What is the—what was it—when they say there's a lower birth weight, how much lower than the average?

A I'm afraid I don't recall the exact figure. I would have to refer to the Freed papers, which unfortunately I don't have here, but my recollection is that it was of the order of ten per cent or so.

THE COURT: Is that a significant drop from a medical perspective?

A No, I think the fact that they recovered by six months to a year would indicate that in overall effects it's not a significant impairment. The real significance of it lies in the possibility that other functions may also have been impaired at the time that—due to the same exposure that resulted in the gross retardation and we will undoubtedly come to that shortly.

MR. DOHM:

Q You have a heading on page 10 of the report entitled Miscellaneous and Toxic Manifestations. Are they so rare as to be inconsiderable?

A I have to recall here something that our professor of pathology said when we were medical students. This is the famous Professor William Boyd, who talked about his experience as a medical student in Edinborough when there his professor of medicine took the group to the window and said, "What are those birds on the roof over there?" And one of the students said, "Sparrows." And he said, "Of course they're sparrows. You don't expect to see canaries in Edinborough. But remember that canaries would be rare except when they happen to you." So that something like a—well, reference is made here to pathogenic fungi such as Aspergillus which produces a very serious disease in the lungs which has been described in AIDS victims smoking contaminated cannabis, that would be rare but I suppose to the person to whom that happens, it's not insignificant. Other than that, I would say yes, in statistical terms they are insignificant.

Q I skipped over a gastrointestinal section. I understand that there does not appear to be any adverse effects in the gastrointestinal—

A That's correct.

Q -- situation.

A And that's—that has not changed in the years since that first report.

Q Going back then to the miscellaneous toxic manifestations, has there been any change since 1981 on that topic?

A No, not really. The other thing that was mentioned there was allergic phenomena and there have not been any additional reports to suggest that this is in any way a significant problem.

Q The next heading on page 10 is toxicity related to unusual methods of cannabis exposure, and what do you or would the committee have described as an unusual method of exposure or administration?

A Well, the one which was given the greatest attention was the accidental exposure to very large amounts by people who swallowed balloons or condoms filled with hashish or hashish oil to avoid detection by the police and in the very few cases in which the container ruptured inside the gastrointestinal tract, the person was then exposed to sudden administration of a very large amount and the toxic reactions that are referred to in that paragraph refer to such episodes. Those are obviously purely random events and cannot be considered meaningful in terms of public health considerations.

Q What type of reactions would those individuals typically suffer?

A They suffered a sharp fall in blood pressure, coma, fallen body temperature, similar to those described in the toxicity studies in experimental animals with very large doses, but it's again the case that none of those resulted in fatality.

Q Let's proceed then to the topic cellular toxicity on page 10 and what was the view of the committee on the cellular toxicity of cannabis?

A The committee at that time was unable to come to any firm conclusion. There were reports that cannabis tested on isolated cells in a test tube was mutagenic, that is produced mutations and drugs which are mutagenic are by definition harmful to the nuclear material of the cell and many mutagens are also carcinogenic, that is they can produce cancer. That's not invariably true, but many mutagens are carcinogens. The problem is that these studies were done in isolated cells and when one looked to see if there was evidence for that happening in humans who smoked cannabis, the results are conflicting. Some studies reported increased frequencies of anomalies in such as what are called chromatid exchange, a breakage and then rejoining in an abnormal fashion, that is not the original fashion in that cell of chromosomal fragments or other changes in cell function by isolated inflammatory cells and some studies showed that there were such changes in increased numbers and others did not. Therefore, the conclusion was that this is an area which requires further study, it requires more precise examination of frequencies and doses used to

give rise to such findings and preferably longitudinal studies following smokers to see whether such changes occurred with increasing frequency or not, as they continued to use the drug.

Q The committee studied reports, experiments on among others for rodents on this, did they not?

A Yes. Yes.

Q And what types of dose would be given to rodents typically in studies of this nature?

A The doses given to rodents are always very substantially larger than the doses given to humans but the—

Q Why is that? Why is that?

A Well, I was just going to explain that there are two considerations that come in. The first is that the dose that you give to—to any species to produce comparable degrees of exposure has to be related to the body size and shape because to produce equivalent effects, the dosage should not be expressed in terms of body weight, but ratio of body surface to body mass. The reason for that is that the smaller the animal, the higher the ratio of the surface to body mass, the greater the metabolic rate, the faster the drug is eliminated and the more drug you need to produce a comparable effect, so that with any drug you can find, for example, that in terms of body surface to mass ratio, if you equate them you're giving an elephant a much smaller dose per unit of body weight to produce the same change that you would produce in a human with a larger dose per unit of body weight and in a rat or a mouse with a very much larger dose per unit of body weight, but all would be comparable in terms of surface area to mass ratio.

Q Is that a generally accepted principle for testing in this area?

A That's a well-known principle of pharmacology, yes. Yes, that's—that's a principle which dates from very early toxicity studies that therefore you must not compare, for example, the dose of the same amount per unit of body weight in a mouse as in a human to look for comparable effects. You need about twenty to thirty times as much in a rat as you do in a human to get comparable drug effects, but the other reason for using much larger doses in rodents is that the rodent studies look deliberately to see the complete range of effects

from very small to the largest you can possibly give and you can't do that in humans for ethical reasons, so that the range of doses you examine in rats tends to go to often hundreds of times what you would give in a human for the combination of the two reasons and the problem is whether the differences between the studies in humans and in rodents are due solely to the difference in requirements of different species or whether they also reflect differences in the objectives.

Q Is the use of rodents as a testing method a commonly accepted way of studying things in the pharmacological field?

A I would say it is almost mandatory. It's almost always the first step. If you're dealing with a drug which has therapeutic uses, you then have to use other species as well in order to study the generality of the effects that you find for licensing of a new drug for a therapeutic use, for example, you have to do it in species other than rodents, preferably in primates, that is in monkeys or chimpanzees and then in the later stage of drug testing, of course, it has to be done in humans, but you begin almost invariably in small animals in order to scan the whole spectrum of effects, focus down on those that you are most interested in for therapeutic or other purposes and then see how defined the quantitative relationships and then see how those change in other species.

Q Has there been any change in the science since 1981?

A Of the methods of studying?

Q Of the methods of studying and—

A No, the methods of studying, I think, are still essentially the same.

Q And the committee's views on cellular toxicity?

A No, I would say that has not changed in the sense that the uncertainty that was expressed by the committee in the 1981 meeting was still expressed by the committee in the 19 -- well, the committee that was charged with preparing the current report that's in progress by the World Health Organization. In the sense that it was still confirmed that there are with high doses of -- or high concentrations of cannabis increased frequencies of breaks in the nuclear material, but there is still not agreement or not certainty as to what the functional implications of that are for humans with respect to, for

example, inflammatory cells or the cells that may give rise to cancer.

Q Okay. I should just insert now that with respect to the 1995 meeting you performed a similar role for the Addiction Research Foundation and the World Health Organization as you did in 1981, did you?

A Not quite identical, because the 1993 to current committee is entirely a World Health Organization committee, not a joint ARFWHO committee, so that my role was not as an ARF member but as a member of the ad hoc committee by the World Health Organization. My role has been to chair that meeting and also to chair the editorial committee that is preparing the report.

Q And are you following the same method of developing a consensus on the document as you did for the 1981 meeting?

A It's rather wider in the sense that the consensus was reached by the committee. Then the committee's draft report was circulated to I believe something like eighty experts in various countries in different agencies or different scientific disciplines for their reactions. The reactions were fed back to the committee for discussion and response. The response was in many cases to make modifications to the report that the committee felt were improvements, clarifications, the reviewers had suggested. In some cases the committee did not agree with the comments made by the reviewers and gave the reasons for their disagreement when they resubmitted the report to the WHO and it is now in a stage of final review by the outside experts and will presumably be published sometime later this year.

Q The next heading in the 1981 committee report is the immune system. What were the findings of the—

A I'm sorry, you're regarding carcinogenicity in the 1981 report as already having been dealt with or—

Q I thought that we had, but please continue, we'll go back to carcinogenicity.

A Right. The only thing that has differed since the '81 report is the question of the case reports that have been published of cancers of the upper airways or upper digestive tract meaning the pharynx and esophagus in people who were heavy smokers of cannabis, and the committee—the current WHO committee reviewed those as well and concluded that while they're only scattered

case reports, they are—they provide grounds for concern in that while most of the subjects in whom these cancers occurred were smokers of both cannabis and tobacco, there were a very small number of cases in which they were smokers of cannabis only and not of tobacco and had not been smokers of tobacco, plus the fact that a number of these occurred in quite young people, people in their late teens or early twenties at an age at which one does not ordinarily see such cancers due to tobacco alone and therefore the committee felt there was legitimate reason for concern of the—either the cannabis alone or the combination of cannabis plus tobacco posed—may pose a greater risk of carcinogenicity than that attributable to tobacco alone.

Q Thank you.

THE COURT: Are these—these are recent cases?

A Yes. These were reports in the late '80's and early '90's.

THE COURT: But prior to that?

A There had been none before.

THE COURT: In the '60's and the '70's?

A No, there were no—

THE COURT: No cases.

A -- reports prior to that, at least none that were described in proper detail as these were.

THE COURT: If there were a connection, between the heavy use of cannabis would not one expect to have seen in some cases through the '60's and '70's as well in teenagers?

A No, for the reasons that I explained earlier. For any production of cancer of this type, given the fact that it's almost certainly the smoke constituents, it would probably be comparable to tobacco in the sense that one would need years of exposure, of heavy exposure, to produce it and the—in the early years of cannabis use in North America, there was not—the use tended not to be as heavy as one sees in a fraction of the cannabis users now. There—the frequency with which there would be an exposed population of users has probably increased and therefore one expects to start seeing cases like that with increasing frequency if they are in fact due to cannabis.

MR. DOHM:

Q Should I direct your attention to anything in the impairment of the macromolecule synthesis?

A No, we've already covered that.

Q Or possible biomechanical mechanisms?

A Biochemical mechanisms. No, that section on possible biochemical mechanisms has proven to be, I think, a non-productive lead. The newer work on biochemical mechanisms relates much more to the discovery of cannabis receptors or cannabinoid receptors and their possible cellular functions and I think the work described in that section on page 13 of the 1980 -- report of the 1981 meeting has probably not panned out. It shouldn't be given much importance now.

Q The next topic then is the immune system. What were the findings of the 1981 committee on adverse effects related to the immune system?

A The literature that was reviewed by the committee for the 1981 report indicated that cannabis or cannabinoids in fairly high concentrations, in fact quite high concentrations, could suppress the function of immune cells either examined in the test tube or in animals such as rats or mice. What was looked for in the test tube was the ability to activate the immune cells to start producing antibodies or to act as scavengers that would pick up foreign material including bacteria and so on and what was looked for in mice was the ability to impair an immune response to a known antigen that should evoke an immune response. Those effects were again considered suggestive but not conclusive and it was recognized that the possible role of the cannabinoids had to be defined more exactly in quantitative terms, in terms of specific functions of immune cells because it was known that immune cells are of different types and have different functions and that these had to be examined in living animals or humans in much more detail before one could conclude that it was a meaningful effect. So that this was regarded as something for requiring further investigation.

It was suggested that the importance of it might lie in people whose immune function was already impaired by other diseases—

Q I'm sorry, Doctor, I didn't hear the—

A It was suggested by the committee then, the section called biological significance, that it might be of greatest importance, a possible suppression of immune function

might be of greatest importance in individuals whose immune function was already impaired by other disease and that in such people it might be a significant consideration. That was as far as the committee felt able to go with a conclusion at that time.

Q Has there been any development in the science since 1981 on that point?

A Yes, there has been—there have been several developments. Not enough to resolve the doubts completely, but certainly there has been considerably more work defining the effects of the cannabinoids on the individual functions of immune cells of different types, both in the test tube and in the living organism. It's been shown, for example, that the cannabinoids can inhibit the production of what are called cytokines—

Q How do you spell that please?

A C-y-t-o-k-i-n-e-s. These are substances that activate immune cells and cause them to initiate their immune functions, including both what are called humoral, that is antibody functions, production and release of antibodies and their ability to attack foreign organisms such as bacteria or fungal cells or particles of foreign material such as smoke condensates depositing in the lungs and it's been shown that in fairly good studies that this is indeed a significant effect of cannabinoids in high concentrations and that these are mediated in substantial part by the cannabinoids themselves, rather than by smoke because there are cannabinoid receptors—I should perhaps deviate here for a moment to explain that development. It was referred to in court yesterday, but I think that it is important to repeat it, that the newer work has been—demonstrate that the cannabinoids act through specific chemical receptors on the surfaces of various types of cells in the body. There's one type of receptor which is found in brain cells and another type which is found on immune cells in the spleen and other parts of the immune system and the rest of the body, so that actions of this type which are initiated through cannabinoid effects on the receptors in immune cells can be considered true effects of cannabinoids themselves, rather than effects of smoking.

Q What is the effect of this discovery? What have we found the effects on the immune system then to be since 1981?

A Unfortunately, there has not been any great advance in knowledge about effects in humans. The only significant experimental study that has involved animal

consequences has been a demonstration or one demonstration that infection with bacterium known as Legionella that produces what's called Legionnaires disease, a form of pneumonia, that the development of immunity to this by exposure of the animals to the bacteria is impaired by cannabis and that is at least suggestive there would be a comparable effect in humans, because there's nothing qualitatively to distinguish that kind of function in animals from similar functions in humans, but there simply aren't data specific systematic examination of such functions in humans, so we have to say we just don't know.

THE COURT: From a clinical perspective then, patients coming into hospitals and doctors' offices, is there anything to indicate that humans are—

A More susceptible to infection—

THE COURT: -- suffering adverse effects—

A No, this is the sort of thing that one would wish had been gathered, in other words prospective studies of users and non-users, do the non-users have a different susceptibility or frequency of infection compared with non-users and unfortunately, there still isn't such information that I have encountered or that the committee encountered. It's another of the areas where one must remain with concerns rather than with factual information to resolve them one way or another.

MR. DOHM:

Q The next topic appears on page 16, Cannabis as an Allergen. Did the committee have any concerns about possible or probable adverse effects of cannabis as an allergen in 1981?

A No, there was no—oh, in '91 it had some concern that there had been reports of allergic responses as demonstrated by skin tests and by the appearance of antibodies, but there has been no new information that has been published since then that we have encountered or that suggests that a major importance of this aspect of cannabis effects.

Q On page 17 of the summary of the committee report is the topic Effects on Endocrine Function. Did the committee come to any conclusions about possible adverse effects or probable adverse effects on the endocrine function?

A In the '81 again?

Q In 1981.

A Yes. The committee felt that there was again reason for some possible concern that required further study, but there was no definite evidence of significant

impairment of function. What they found was some studies which indicated that in the short term the administration of rather large amounts of cannabis in animal studies and in one or two human studies suggested a decrease in the production of testosterone, a decrease in sperm count in the human subjects in whom the testosterone levels were reduced and in females, an increased possibility of what are called anovulatory cycles, in other words menstrual cycles in which no release of egg cells occurs. These were all short term observations and there was no evidence on which to conclude whether that had any long-term significance or not.

Q Has there been any development in the science since 1981?

A A small amount, not much. There has been demonstration that luteinizing hormone—

Q Would you spell for the sake—

A -- which is a—

Q -- of the person who will have to type the transcript?

A Yes. L-u-t-e-i-n-i-z-i-n-g, or luteotropic, l-u-t-e-o-t-r-o-p-i-c hormone which is the hormone that's released by the pituitary gland that stimulates production of sex hormones in the testes for the male or in the ovary for the female is diminished by cannabis smoking or has been shown to be diminished in some experimental studies with humans that involve smoking over a period of weeks; however, again this led to no long-term conclusion on—or rather no conclusion about long-term significance because there is a suggestion in both humans and animals that tolerance may develop to this effect and that the levels may return to normal, despite continued smoking of cannabis, so that there is less concern now by the committee, the current committee than there was by the previous committee in terms of possible effects on the endocrine system.

Q That should take us then to page 21, Reproduction and Development.

A Yes. The—as far as reproduction is concerned, that—the concern at that point was essentially concern about the possible effects on fertility and given the absence of any conclusive findings on the endocrine system and on the production of sperm and ova, there really has been nothing to suggest any long-term effect on fertility. If there is an effect, it would have to be limited to the period in which the demonstrated reduction of sex hormones and of germ cells, sperm or release of egg

cells, occurred and that was obviously of very short duration, so that that is not considered now to be a significant risk.

The main concern is with respect to reproduction is really the development aspect, the development of the fetus and postnatal development. I've already referred to the in utero development, that is the prenatal development of the fetus before birth as being essentially one of modest but statistically significant retardation of growth, including all parameters of growth, not only weight but also head circumference and crown to rump length. The babies are smaller, and I believe I mentioned earlier that at birth they were excessively irritable, they didn't respond as well to light stimuli and they didn't feed as well, but that these all returned to normal within a matter of six to twelve months after birth.

But the aspects of development that have attracted the greatest interest have been in terms of what is called cognitive development or mental development. This has been studied by the Freed group through repeated observations annually and up to currently up to early school age. The first cohorts of babies that were born and studied in this study have now reached the age of first or second grade in school. They had been examined in Kindergarten and followed through to first, second grade. They're now getting to seven or eight years of age and the only changes that have been found repeatedly have been those relating to verbal learning and memory, ability to carry out some school tasks that require verbal learning and these were not seen in preschoolers because, of course, the tests that one administers to preschoolers don't usually examine such functions, but when they get to school age this becomes part of the battery of testing that one looks for to follow intellectual and educational development with progression through school, and these findings, though they have been not dramatic in degree, have at least been consistent and statistically significant and therefore they constitute at least a ground for some concern that future progress in school requires careful monitoring because of the possibility that effects on these skills may hinder academic development. That's something which the committee considered to be important enough to warrant further examination as the children progressed through school.

Q Which committee—

A I'm referring here to the current WHO committee.

Q You mentioned different tests being applied in different times or different stages of a child's development. Is that in accordance with the usual types of procedure in these kinds of cases?

A Yes, this is in accordance with what is conventionally done in child psychology because the types of tests that you can apply at different ages depend on functions which mature to different degrees at different ages, so that you don't test a baby for verbal skills but you test a five-year-old for verbal skills and therefore, tests of this kind become meaningful when the child is old enough to

be normally expected to have developed those skills to a point where they can be quantitatively tested.

THE COURT: Have we done similar tests on children of mothers who smoke tobacco?

A Yes. This study examined mothers who smoked -- children of mothers who smoked tobacco, those who smoked cannabis and those who didn't smoke.

THE COURT: How—are you able to summarize how those three groups compared to one another?

A According to the latest Freed reports which were last year in one of the major toxicology journals, the children of mothers who had smoked cannabis showed a significant impairment compared with the other groups.

THE COURT: It's—are we still on—it's five to eleven. I thought if we were going to move to a new topic, we could perhaps take the morning break.

MR. DOHM: I have just one more question on this topic, if I may.

THE COURT: All right.

MR. DOHM:

Q Did the 1995 committee make any recommendations specifically or express any—or would you expect them to express any specific concerns about this variation shown in the latest Freed report?

A Yes. The committee did in its draft report did say this was an area which required follow-up, that its—the degree of its significance to the development of those children would need to be assessed by monitoring of them through subsequent stages of their school careers and it did indicate that this was one of the areas which warranted continued study.

MR. DOHM: That's all, Your Honour.

THE COURT: We're going to take the morning break at this time then. We'll return in fifteen minutes.

(WITNESS ASIDE)

(PROCEEDINGS ADJOURNED)

(PROCEEDINGS RECONVENED)

DR. HAROLD KALANT, recalled, testifies as follows:

MR. CONROY: Just indicating for the record, Your Honour, that Mr. Caine isn't present in the courtroom because he's taking Dr. Morgan back to—out to the airport.

THE COURT: All right.

MR. CONROY: He'll be back as soon as he can. It's not that he's not interested.

EXAMINATION IN CHIEF BY MR. DOHM continuing:

Q Doctor, we're still dealing with the committee report from 1981. I would ask you to direct your attention to page 23 at the bottom of the page, topic 7, affects on nervous system function.

A And you would like me to summarize?

Q Would you please tell us what the committee found with respect to the effects of cannabis on—the acute effects on intellectual functions?

A The—there is a fairly extensive description on pages 23 through to bottom of page 26. I'll attempt to summarize those very briefly. Cannabis at that time was recognized to have, in terms of its acute effects, to have a characteristic time pattern in which the initial effects for the first fifteen minutes or so were mainly excitatory, that's the period in which the heart rate increased, the period in which people are animated, talkative. In experimental animals there's increased movement and increased physical activity and then there's a gradual transition over into a period of sedation in which people become quiet, relaxed and animals become less actively moving and in the first phase, the excitatory effect is additive with that of amphetamine or cocaine while in the quiet phase that follows, the effect is additive with that of alcohol, barbiturates or tranquillizers.

The predominant effects that have been studied have been those during the quiet phase which lasts for much longer part of the total experience and those effects have been most importantly impairment of short-term memory, that is memory for what has happened within the immediate past, while long-term memory is not affected. In other words, the person has no problem remembering things that have happened months or years ago, but may have problems remembering what happened a few minutes ago.

The mental processes are generally slowed. Reaction times are slowed. There is some difficulty making associations between thoughts. At high doses, people have greater difficulty expressing their thoughts or thinking through them in a clear, sequential manner. At very high doses, as I mentioned earlier, there are experiences such as what is called depersonalization, that is the feeling that the person is outside his or her own body and is looking from a distance at it, which can be associated with other feelings that cause it to be classified as a hallucinogen in some text books. The acute emotional effects that are related to that depend on the person's experience and previous state, because those feelings can give rise to panic or they can give rise to feelings of pleasure as part of the total experience of the drug effect and one tends to find panic mainly in relatively inexperienced users or people who are suddenly using a much higher dose than they have on previous occasions.

The problems with short term memory are also demonstrable in both humans and animals in terms of the consequences of that for learning. Learning a new task is impaired by the cannabis effect, and if it's a task which is learned over repeated practise sessions, it takes longer to acquire the learning under the influence of cannabis. This is not unique or distinctive for cannabis. This is also true for alcohol and for other sedative drugs.

There have been other effects. One of those which the committee was concerned about in the 1981 meeting was what was called at that time in some of the literature was called an amotivational syndrome, that is a state in which the person is lethargic, uninterested in systematic, routine work, not terribly motivated in terms of income-generating work and spends more time preoccupied with obtaining and using the drug and/or other drugs. The committee concluded that there was no evidence to support the existence of what was called a specific amotivational syndrome, in other words motivation does appear to be affected. There was evidence at that time, clinical observations that many heavy users were indeed lethargic and had difficulty rousing themselves either to school or to employment and generally performed poorly, had poor work records, had a higher likelihood of being unemployed and had a higher drop-out rate from school, but this was not considered to be a specific amotivation syndrome as such and it was considered to be simply a sign of continuing intoxication and it was recommended that the term amotivational syndrome should be dropped from the literature and didn't warrant any further attention, but that attention should be given to studying how long does—do the effects last if use is stopped. Are they reversible and so on.

Q Would one expect to find similar reactions from people heavily involved with other psychoactive drugs such as alcohol or cocaine or anything you might name?

A No. The pattern of effects with cocaine or amphetamine or stimulant drugs is quite different. With alcohol, yes, chronic alcohol consumption does tend also to reduce motivation, but it was felt to be more marked with cannabis because the duration of the drug and of its actions is longer than that of alcohol that would produce a comparable degree of acute maximum effect.

In other words, alcohol is cleared from the body more rapidly than cannabis; therefore, someone who uses cannabis daily can be to some extent under the influence of cannabis virtually all the time, whereas a person who drinks alcohol daily to produce the same acute effect tends to be sobering up for enough of the next day to be able to work and to maintain a job, so that the effect of chronic alcohol use on employment and performance is perhaps not as marked as that of cannabis for comparable degrees.

Q Where were you going when I interrupted you there?

A Well, I was going to say simply what the changes or—

Q Okay.

A -- what the new information has been since that time. The—one of the things has been a better definition of the type of memory impairment. There have been continued studies which by and large have not added greatly to knowledge, other than to clarify the types of impairment that are most marked. For example, one recent study in 1994 showed that there was more impairment in humans smoking cannabis when they performed a task that required monitoring of the time than in a task which was not time-dependent because their time sense does appear to be impaired and also on this particular task it could have—the results could have implied impaired learning because the task involved was one in which there was a fixed interval. They had to carry out a certain test performance in order to earn a monetary reward and they on a fixed interval, they—that is they would have to wait for so many minutes before the next response would produce a reward.

At the same time there was what was called an escalating interval in which the interval increased progressively from, for example, five minutes to ten minutes to twenty minutes and those—well, the same subjects performed both under the influence of cannabis and without cannabis and on the days when they were doing it without cannabis they learned not to waste their efforts pressing when the interval increased because they wouldn't gain anything by it, but under cannabis they didn't adjust to the increasing intervals and that was interpreted as possibly being either impaired time sense or inability to learn as quickly that it just didn't pay them to keep pressing the lever when they weren't getting any reward for it.

Other than that, there really has not been any drastic change. There's been simply a sharpening, a better definition of the kinds of function that are impaired.

There have been some studies in animals which were mentioned in the 1981 committee meeting report and have subsequently been confirmed by other studies later indicating that long-term exposure may lead to a permanent impairment of learning in very heavy users. That had been suggested clinically by the observation that people who came to treatment because they were concerned about their own use of cannabis and its effects on school performance or on work performance and so on around family relations or on

troubles with the law, when they stopped using cannabis usually recovered their memory, usually recovered their verbal skills, their time perception and so on, but those with very heaviest history of long-term use sometimes did not and the question was whether that represented a permanent effect on the brain, whether that was indicative of brain damage or not.

So there have been various studies done since to try to clarify that. Some of the animal studies which our own group carried out and I mentioned in referring to the curriculum vitae which we reviewed yesterday worked by Dr. Fehr and Dr. Stiglick when they were graduate students at my laboratory, that involved the ability of animals to learn a rewarded maze solving task while they were under the influence of cannabis and then a month, three months, six months or even a year after the cannabis had been stopped, and it was found that the animals on the highest doses which were equivalent to regular daily human use of at least one or two cigarettes a day of cannabis did not recover their learning ability six months or even in one instance a year after they had the chronic administration.

Now, I emphasize that a rat's life expectancy, laboratory rat's life expectancy is roughly two years. They were treated for three months with the drug, which means the equivalent in human terms of several years of heavy daily use. The failure to recover for six months would represent a quarter of their life span or for a year would represent a half of their life span, so we're talking here about the animal equivalent, an animal model of long-term use and very long-term post-use observation in the rat.

On the other hand, the suspicion that this might be due to organic damage to the brain has generally not been borne out. Evidence looking for specific brain damage, cell loss, specific atrophy of particular parts of the brain such as the hippocampus, that's h-i-p-p-o-c-a-m-p-u-s, that's part of the brain which has been very much implicated in mechanisms of learning and memory. Pathological studies have not shown any cell loss in those parts of the brain, but they have not ruled out more subtle changes, because learning ability involves what are called synaptic connections between nerve cells. Learning and memory require the passage of nerve impulses from one nerve cell to another and there is some evidence which Dr. Fehr got during her work which has not been published but because it requires replication and larger numbers of animals to suggest that the numbers of connections of synapsis or connections between the nerve cells are diminished by the long-term cannabis treatment that she used in the learning and memory studies and that type of subtle brain functional change has not been ruled out by the studies done to date and that remain one of the current committee's concerns.

Q Is it fair to describe synapsis being something like an intercellular pathway or messageway for electric impulses or—

A Yes.

Q -- nervous impulses?

A Yes, that's exactly what synapse means. A synapse is a contact of the fibre coming out of one nerve cell that carries impulses to a receiving fibre or part of the cell body of the cell to which that information is being transmitted. It is in the great majority of cases a chemical transmission. The nerve impulse causes release of a chemical at the synapse that then passes over to the next cell and activates a receptor on the second cell in the chain and those synapses are recognized by electron—in electron microscopy as tiny protuberances from the fibres that can be actually stained and counted and the synaptic density, that is the number of synapses that an individual nerve cell makes has been shown to be markedly decreased by high—chronic high dose treatment with alcohol and Dr. Fehr's work suggests that the same would happen or does happen with chronic high dose administration of cannabis, of THC.

Q Does the work indicate whether that is an effect of smoke or of the THC?

A No, that's because of THC because this was not administered as smoke, as the pure chemical.

THE COURT: How was it administered?

A I'm sorry?

THE COURT: How was it administered?

A It was administered by injection, the—

THE COURT: Have there been any tests regarding—

A Yes, we also—

THE COURT: -- inhalation?

A -- tested giving it by mouth, but to give it in smoke is simply not feasible in the rat because there isn't a good method of delivering the smoke in a way that you can quantify so that you know what dose you're giving. It would be ideal to be able to do that so as to simulate what happens in humans a little more accurately, but in terms of the fate of the THC in the body, that is not changed by the administration, by the route of administration. The time of onset is different, the speed with which the drug reaches its peak concentration is much more similar by injection and by smoking than it is by administration by stomach tube which is a lot slower, but the trouble with injection is that it takes a special solvent to dissolve it because it's not readily dissolvable in water-based media and you don't want to inject a suspension of droplets intravenously because that will produce microembolism so you have to use solvents that one would prefer to stay away from but in—if you inject

intraperitoneally, you're not worried about droplets and therefore that gives you a reasonably fast absorption without the risks that intravenous injection would give.

MR. DOHM:

> Q Does that then take us to page 25, acute effects on driving skills and driving performance?
>
> A Yes. Yes, because the—the same effects on attention, arousal, alertness and so on that had been described in the general performance tests are of obvious relevance to driving and people interested in driving accidents have done a lot of work not only with cannabis but with alcohol, with benzodiazepine tranquillizers, with cocaine, with a variety of other drugs, to see what their effects are on level of alertness, on attention span, on the ability to monitor, pay attention to several different sources of information at the same time, this is referred to as divided attention tasks, on speed of reaction, on accuracy of judgment of distances and speeds, on risk-taking and on motor control, accuracy of response, ability to steer, to move a control lever or a drive wheel accurately to keep on target, and these have shown unequivocally, I mean all observers agree that cannabis in socially-used doses in humans impairs most of these functions in a manner comparable to that of alcohol and additive with that of alcohol.

The question has been while cannabis can do this in the laboratory in impaired driving skills, does it in fact be—get used before driving and therefore produce real life accidents or real life risk on the highway and the committee in 1981 had only one on-road study to go on and that was the study by Dr. Harry Klonoff in Vancouver, who had actually studied driving on a test course and in city traffic and for city traffic he used dual control cars so that the observer who was rating the performance could make corrective adjustments if necessary suddenly if the driver had a lapse that put the—that created too great a risk of an accident.

What they concluded was that cannabis on the whole in the whole group of subjects, impaired driving performance, not invariably. There were some subjects whose performance improved compared to their own drug-free state but the proportion who showed impairment was quite clearly larger and significantly so and the degree of impairment in those who were impaired was comparable. They selected two doses to produce effects that were comparable to those of low and a moderately high blood alcohol level, that is forty milligrams per cent and eighty milligrams per cent of alcohol and the effects of cannabis were on the whole comparable to those of the corresponding alcohol levels and were of similar duration in terms of the duration of the driving test.

That has since been followed up. Let me add further to the 1981 report. Other types of evidence, statistical evidence, to implicate cannabis in the actual

production of driving accidents was by and large lacking. There were one or two studies which reported finding cannabinoids present in the urine of drivers who had been stopped for impaired driving or had had accidents, but the great majority of those also had alcohol present in amounts which by themselves would impair driving ability and it was not possible to judge from those findings whether cannabis was contributing significantly or not and the committee recognized that and said that there was a need for further studies of its contribution to actual driving performance in real life situations.

If I may then pass to the—

Q Can I just ask a question for clarification of these. In those studies that found the presence of both cannabinoids and alcohol in the urine tests, was there any way of measuring the level of THC active in the human body at the appropriate time?

A No. That was another problem with those studies because the cannabinoid metabolites, that is the substances to which THC or tetrahydrocannabinol is converted in the body and which are no longer pharmacologically active are excreted in the urine for hours or days and in some cases possibly weeks after the last known use of cannabis so there's not a good correlation between positive test results in those instances and the blood levels of the active material at the relevant time.

Q Does your earlier evidence about the additive effect of cannabinoids and alcohol have any relationship to the evidence that you have just given?

A No, because those were done after the actual administration of known amounts of the cannabis and/or alcohol with testing during the period of a few hours immediately following that, so that both were known to be present in active concentrations during that time. That's why experimental studies are much easier to interpret than statistical studies gathered from post hoc or after the fact information from highway drivers or accident victims.

Q Okay. Thank you. Please continue.

A Well, I was going to say that that problem has been to some extent remedied in the years since the 1981 committee report because there have been now a number of studies in which people have been stopped not because of—they didn't—weren't involved in fatal accidents with chemical analyses many hours later, but were stopped at the time for impaired driving or for minor accidents occurring at the time and blood samples had been taken so that the levels of THC had been measured in serum rather than metabolites in urine. That also does not give a perfect correlation, but it's a much better correlation because one knows that as long as there is

measurable levels of THC present in the plasma comparable to those which have been measured experimentally during periods of drug activity, that one can confidently assume that the drug is producing its usual effects. The question then has been have there been people with such levels who did not have alcohol present and were driving badly or involved in accidents, and several studies have confirmed that that is the case, that in those who were alcohol negative the most common drug found in correlation with impaired driving or minor accidents and the absence of alcohol has been cannabis. The question remains then is that more than random chance, and one can answer that only by extrapolations which are not wholly satisfactory.

You would like to know, for example, has positive—or have positive blood levels of THC been found with a higher frequency in people involved in poor driving or accidents than they would be in a random sample of the population if you simply stop people on the sidewalk and got comparable blood samples? And such samples have not yet—or such studies have not been done; therefore, all you can do is to say how—what does the evidence indicate the probable frequency of current use of cannabis would be in the general population and the only way you can do that is to look at current statistics for daily users or let's say weekly users on the assumption that a blood level might last for more than a day if someone were a heavy user, given the fact that the percentage of users who are daily users or better than weekly users in the population is only a few per cent currently. The chances that if you stopped someone on any day of the month and took a sample would probably give you a purely chance occurrence of significant levels in perhaps a couple of per cent of the population; therefore, if you find as has been found in these accident series, as many as ten or fifteen per cent with significant levels, it suggests, it doesn't prove but it suggests that the cannabis was contributing to the production of the accident and was not just a random finding that you would have found in non-involved passersby.

Q Now, you have explained the difficulty of proving that very hazard. Do similar difficulties exist in demonstrating the effect on psychomotor operations and cognitive functions in people who are impaired at impaired type levels of THC?

A No. No, no. As I explained, where you are measuring the effects during a known period after the administration of a known amount of cannabis for a time during which one knows what the time course of concentration in the blood or in the brain may be, then that worry does not apply. One knows that the drug, period of drug action is associated with a period of measurable impairment of those functions. All the driving simulator studies, for example, or flying simulator studies are done in that manner.

Q The—has the science advanced since 1981 on this point?

A Yes, there have been better tests developed, more refined and more exacting tests of attention and of visual or functional visual attention, the use of perimetry for noting how small or how large a part of a total possible visual field the person actually pays attention to is a recent development and things of this kind have been used in the study, not only of cannabis but of alcohol and other drugs and have served to fairly consistency lower—fairly consistently lower the concentration that has been found to produce measurable impairment, in other words, the more sensitive the test, the easier it is to show a significant impairment at a given blood level.

Q And that applies to which types of drugs again?

A That applies to all of these drugs. The same reasoning applies to the consistently lower levels of drug that are found to impair flying skills compared to driving skills, because flying is a more demanding task. One has to monitor more sources of information currently and make more rapid critical responses and the driving studies have shown impairment of cannabis—or rather the flying studies have shown impairment by cannabis more readily than driving studies have, that is lower doses and for longer periods of time.

Q Did the 1995 World Health Organization address this issue of impaired operation of vehicles?

A Yes, it did.

Q And was their conclusion any different from that of the 1981 committee report?

A Only in the sense that it was more emphatic, that there was clear demonstration of impairment of a wide range of driving or flying-related skills and that there was now at least better suggestive evidence of actual implication in real life accidents, as shown by the kind of epidemiological study that I referred to, that is in non-fatal cases and measurement of levels in plasma rather than in urine.

Q Have you had an opportunity to read in any detail or to—the Robbe report on a driving study done in the Netherlands recently?

A No. I saw—I had a chance to look at it briefly but not to read it in detail. The only things that I looked—had a chance to look for specifically were the doses that were used in that study and the rationale for the selection of

those doses and the authors gave as the reason for selecting one hundred, two hundred and three hundred micrograms per kilo, the fact that these produced blood levels which corresponded to what regular experienced users considered to be mild, moderate or good psychic effects. The three hundred was the one that corresponded to the effects which they attempted to produce in themselves by choice. So that it would be comparable to alcohol levels that might correspond to two or three drinks and I think that from my very brief reading, I gather that that was the reason for them comparing the doses of cannabis that they did to the doses of alcohol that they did.

Q Have you dealt in your earlier evidence on the effects of the dose in studying the motor capacities of an individual? In other words, what I'm trying to say is have you described for us the differences in results that come from different doses of THC when a person is trying to perform complex motor skills?

A I haven't—I don't believe I did—

Q Would you deal with that now please?

A Certainly. It's—I think all that needs to be said is that all investigators who have examined various doses have found that the effects, as one would expect, are dose-related. This is not unique for cannabis. This is a general principle of pharmacology. All measurable drug effects other than allergic or host-related reactions rather than drug-related reactions, all of those characteristic drug effects are dose-dependent. When you study the action of a drug, you always do a dose response curve. You give a range of doses and measure the effects of each of the doses so that you can say how much effect is predictable from how much of the drug and that is true, just as true of cannabis as it is of alcohol or morphine or digitalis or any other drug.

Q Page 26 of the committee report in the last paragraph under this heading, there's a quote,

> "These findings provide a striking demonstration of the ability of even small doses of marihuana to impair driving ability."

What were the small doses referred to in that quote compared to what you have seen from the Robbe report? Are you able to make that comparison?

A They were—they were fairly comparable. My recollection is that they used one or two cannabis cigarettes. I don't remember the concentration. That

may be -- no, I don't believe—it was described in Dr. Klonoff's paper in the complete volume, but I don't believe it was mentioned in the executive summary. No, it isn't. But it would have been comparable to the doses that were used in the Robbe study because they also used, as I recall, either different potencies or different numbers of cigarettes to achieve the three different levels of concentration in the plasma.

Q The next topic is on page—starts on page 26, but I direct your attention to page 27 and Mr. Conroy is directing my attention to the clock. It would appear to be the noon hour, Your Honour.

THE COURT: All right. We will adjourn 'til 1:30 then.

MR. CONROY: Thank you.

(WITNESS ASIDE)

(PROCEEDINGS ADJOURNED)

(PROCEEDINGS RECONVENED)

MR. DOHM: Calling Regina versus Caine, Your Honour.

THE COURT: Thank you.

DR. HAROLD KALANT, recalled, testifies as follows:

EXAMINATION IN CHIEF BY MR DOHM continuing:

Q I'd like now, Dr. Kalant, to direct your attention to the report of the committee of 1981, page 33. There's a heading there Psychiatric Consequences. I would like to ask you whether or not the committee came to any conclusions about the potential psychiatric consequences of cannabis use.

A In the 1981 report, no, it did not come to a very clear conclusion. They recognized that the things which had been described as cannabis psychosis in the older

literature dating from the 19th century on, were questionable, that the case descriptions were not very good and that there was a tendency to take any case that in a psychiatric hospital in which the patient was known to have used cannabis to attribute the illness to cannabis and this was not a very valid procedure.

Q Scapegoating is a word that comes to mind.

A Yes, basically scapegoating.

Q All right.

A And they concluded that this was something which required further investigation, but they were not in a position to offer any clear judgment about it other than that the term cannabis psychosis they felt was probably not a meaningful term.

Q Was there any change in the conclusions of the World Health Organization committee from 1995, was there any difference—

A Yes.

Q -- in those?

A Yes. That—the picture appears to have changed quite substantially since this earlier report because there have been a number of studies looking specifically including particularly a Swedish study which followed schizophrenic patients from their medical records over a period of time to see first of all whether those who used cannabis had a different picture from those who didn't, whether the cannabis use preceded the onset of the psychosis or vice versa or whether they were concurrent and they came to the conclusion, as several other investigators have, including Dr. Negrete (phonetic) of the Montreal General Hospital Psychiatric Unit in one of his published studies that while there is good evidence that schizophrenic patients have a higher probability of using cannabis which may represent a symptomatic part of their schizophrenic behaviour, that there is on the other hand also clear evidence for a substantial group of schizophrenic patients, that the cannabis use preceded the onset of the overt clinical symptoms and that in some of them this happened on more than one occasion, in other words they began to smoke cannabis after a variable period of use. They went into a period of frank clinically diagnosable schizophrenia, they were treated, recovered, left hospital and then had a repetition of the same sequence.

The committee discussed this at some length, concluded that it's not possible to say with absolute certainty that the cannabis use caused the schizophrenia, but that the fact that in a substantial number of cases it—cannabis use preceded the overt clinical appearance, that it had to be considered a very real possibility, a significant possible risk that cannabis could precipitate the

appearance of schizophrenia in someone who had a schizophrenic predisposition.

Q We have heard of a study done with some members of the Swedish military, I believe it was. Is this that same study?

A It grows out of the same study. The trouble was that in the study of the conscripts, they had no way really of being certain of the relationship between cannabis use and schizophrenia other than that those who smoked cannabis when they were conscripted and were followed up subsequently, a higher proportion developed schizophrenia than among those who weren't smoking cannabis, but the study that I'm referring to then took actual cases with significant clinical histories that permitted sequential following of events in the individual cases and therefore was able to come to a conclusion with more certainty and in—diagnosed individual cases of what the sequence of drug use versus onset of schizophrenia had been.

Q So do I understand then that there was some significant difference in the methodology of the two studies?

A Yes.

Q And when was that later report approximately, do you recall?

A It was about 1993. I can check that to be certain.

Q That would have been between then the time of the Hall report and the time of the World Health Organization—

A Yes.

Q -- committee—

A Yes, that's correct.

Q -- or would it have preceded the Hall report?

A I believe that was—no, I think that was mentioned in the Hall report.

Q Thank you.

A I'll just double check that, but I believe it was. I'm afraid it'll be more difficult to find here because there isn't a heading on psychiatric—

Q It's not—I don't see it as being especially—the timing as being especially important in any event, Doctor.

A But I'm fairly sure I can find it here in my—in the draft of the—

Q Let's not worry about that, Doctor.

A Okay.

Q We can go on to the next topic, I think. The next topic is a quick return to the heading epidemiology which is found on pages 34 and 35 of the committee report and there are two items that you and I discussed over the break. The first has to do with what types of studies are going to be needed to show various things. Can you give us your view of the committee's report on that aspect, please? I'm directing your attention, please, to page 35, the paragraph that begins with, "For this reason ..."

A Oh, yes. The point here was simply that statistics, the shortcoming in statistics at the time that this report was prepared was first of all that the shortcomings were that first of all there was grossly unequal quantity and quality of statistical information from different countries. Some countries had a large amount of information over a relatively long time and others had very little, except spotty, occasional, random checks.

Secondly, the relationship to—of cannabis use to some of the potential adverse effects was not carefully documented. There wasn't such information, and given the type of statistical information that was necessary to establish the link, for example, between alcohol consumption and cirrhosis or between cigarette smoking, tobacco cigarette smoking and lung cancer, it was recognized that two things were needed; that what are called prospective studies, that is studies in which you take a large group of people including both users and non-users and follow them up over a long period of time so that you can see what difference there is in terms of the health consequences among the users compared to the non-users. That would be necessary, and secondly, that very large scale retrospective studies, that is taking current data and saying among users—or among people with a particular problem, how many had used cannabis and how many had not and so on, large scale studies of that type, supplemented by the long-term prospective studies would permit resolution of many of the questions that existed about the connection or alleged connection between cannabis use and some of the health problems.

Q Is there any need in doing those studies to have some knowledge of the general rates of use in the population?

A Well, yes. Yes, you want to know the rates of use in the population because that, in part, will determine how large an—how large a population must be observed in order to get meaningful figures. For example, if you have a population in which .5 per cent of the people use a particular substance and another population in which 50 per cent use, you stand a much better chance of discovering connection between use and health consequences in the population of which 50 per cent are users than you do in the one in which .5 per cent are users.

Q I'd like to go back to our discussion earlier on the neurological system and I would like you to explain to us the difference and the significance in the state of scientific knowledge between functional and structural effects of a substance such as cannabis.

A I'm sorry, this is in relation to which system?

Q The nervous system.

A Nervous system.

Q Excuse me.

A Yes. The point I was making earlier, and I think it bears repetition, is that what one is interested in is for most population purposes is the effect on function. Effect on structure represents often extreme cases. In other words, if you look for signs of cell loss in the brain, we know from the alcohol model that we're there looking only at extreme cases. Overt brain damage produced by alcohol, loss of cells in specific parts of the brain is seen in severe alcoholics usually after many years of very heavy drinking and often presents difficulty in sorting out the influence of the alcohol, the influence of head injury while drunk, the influence of nutritional deficiencies. Those are cases which have practical importance in hospitals that treat such cases, but from a public health point of view, one is more interested in consequences that alter function, that alter performance.

Q Is it necessary, as a matter of pharmacology or medical science, for there to be structural changes in order for there to follow functional changes?

A No. No, functional changes can occur from altered cellular function without actual microscopically demonstrable cellular injury. Most cases of intoxication, for example, in which there is altered function, are not accompanied by any overt or demonstrable cell injury.

Q Is there anything else that you had wanted to add to that?

A Simply that this explains why the more recent studies have found alterations of performance, things such as memory, abstract reasoning, learning, at levels of intake of cannabis that in earlier studies were not described as producing any change, which was thought to depend upon cell damage, brain injury. For example, there's a test called the trail-making test, which is taken as a sign of organic change in the brain and that has not shown alteration from which earlier investigators concluded that there was no evidence of brain injury by long-term cannabis use. But with more sensitive functional tests, it is possible to show differences which can outlast by a considerable time the actual period of cannabis use.

Q Okay. Now, in the course of the evidence in this case we have heard something about a course taken by one Western country, the Netherlands, with respect to the way that they deal with cannabis use. Are you familiar through the literature, through your work or as a member of either the Addiction Research Foundation World Health Organization committee or the World Health Organization committee with the—with whether or not there are statistical records of what rates of use may have been prior to the change in the Netherlands approach, both in the Netherlands and in other European countries? That's a two-part question. Firstly, are you familiar and if so how and then we'll get on to the balance.

A Yes, I have had occasion to read publications on that topic in preparation for the current World Health Organization committee report, and there are two points that I feel competent to make, that I think are worthy of consideration. One is that the committee did not have any available data on levels of use prior to the adoption by the Dutch authorities of their policy of non-punished sale in coffee houses, so that it would be unwarranted to attribute the relatively low rate of use in the Netherlands to that policy, since there's nothing to compare it with. In other words, there were no previous figures so that one can say—that would enable one to say whether that policy had caused a change in the level of use.

However, the second point is that since that policy has been in effect, there have been statistical or epidemiological studies of limited scope in the Netherlands, as well as in other European countries, North America and Australia, and one of the published studies indicated that the level of use in the Netherlands during the period in which this policy has been in effect indeed had not increased dramatically. It had increased, but not dramatically, but at the same time in other European countries they had decreased quite sharply, so that the difference between the Netherlands and the other European countries was that at a time when the rate of use was falling in other countries, it actually rose somewhat in the Netherlands.

Q Now, you said that one study showed that. Was that study one of many or was it a study that was inconsistent with other studies? Can you tell us that?

A No, it's the only one I know of.

Q Okay. And I ask you that because I take it from your earlier evidence that rates of use can be important from a health point of view when one wants to assess and measure risks, is that a correct statement?

A Yes. The assessment of risk depends upon—or requires knowledge of two things: what percentage of -- or what level of use is necessary to produce a particular health problem; and secondly, how many people in the population of potential users do use and use at such levels. So that one needs to know not only the mechanistic connection between use and a particular consequence, but also how many people put themselves at risk of such a consequence by using the necessary levels.

THE COURT: What were the years over which that study was done?

A That study was from the mid-1980's up to 1992.

MR. DOHM:

Q From what you told us yesterday, Doctor, and from looking at your curriculum vitae, it is obvious that you have spent a considerable amount of time in your professional career studying the effects of alcohol.

A That's correct.

Q And is there a particular disease or health problem that flows from alcohol use that you look for in coming to your conclusions on the prevalence of alcohol as a health factor?

A There are a number of diseases which correlate in a population with the level of alcohol use, but the one that has been most widely used is the morbidity and mortality rate from alcoholic cirrhosis of the liver.

Q Do you, as a result of your training and your experience, have knowledge of the rates of cirrhosis and morbidity due to cirrhosis of the liver from in the United States from 1900 to the present?

A Yes, there was a study by Klatscan (phonetic) which presented those data and which we have used in one of our own publications to illustrate the influence of other factors on the prevalence of cirrhosis and on the death rate from cirrhosis.

Q What I'm going to ask you to do would be to just give us the trends and identify any years that show a beginning and end of trends.

A From the beginning of the century up to about 1916, there was a slow, gradual decrease in the death rate from cirrhosis. Then in 1916 it dropped very abruptly. This was the time at which the American government and various state governments used—took advantage of existing interstate commerce regulations.

Q All I want you to do is to just give us the—

A The times.

Q -- times and—

A All right.

Q -- the results that you could see as a scientist without trying to tie them to any cause.

A All right. There was a sharp decrease beginning in 1916. It was maintained until 1932, and then after 1932 the death rate began to rise again fairly steadily, right up to about the late 1980's.

Q You said there was a sharp decrease maintained from 1916 to 1932?

A Yes, the decrease, and I'm sorry I'm not explaining that very well, there was a sharp decrease. The low level which was achieved at about two years after 1916 was then maintained 'til 1932 and after that, the level began to increase again.

Q And did—was there any change after 1945?

A I can cite Ontario figures for that.

Q All right.

A In the Province of Ontario, similar records of death rate from cirrhosis have been kept for an equivalent time, almost as long as the American ones, and the cirrhosis death rate dropped very sharply in 1932, 1930 to '32. It fell until 1939 or '40, then began to rise gradually and then post-war, from 1945 until about the late 1980's rose much more rapidly for a period of about twenty years and then levelled off, and in the last few years has begun gradually to drop.

Q I'd like to direct your attention now to what we have been describing as the Hall report. It's Exhibit 5 and it's found in Volume 1 at tab 3. Do you have that before you?

A I have a copy of the Hall report.

Q A copy of the Hall report?

A Yes.

Q Okay. Would you direct your attention please to the executive summary, IX.

A Oh, in the report itself, is this what you're referring to, executive summary?

THE COURT: Yes.

MR. DOHM:

Q Yes, page—

A There's no Roman Numeral on the front of that.

Q It has a heading on the copy that I have that's printed large for folks like me, so I'll know where to go.

A Executive summary. Yes.

Q Do you have it?

A Yes. This is the page which begins, "The following is a summary of a major adverse health and psychological effects ..."

Q That's right. Let's just wait for my friend to find his copy.

Now, I don't wish to take you through each of these separately, but I do wish to deal with the headings and I would appreciate it if you would indicate to the Court whether the—any parts of the headings are in—are areas with which you disagree. And the first heading is Acute Effects and that lists a number of five effects. Is there anything in there that you would consider is not consistent with the current state of scientific knowledge on the effects of cannabis?

A No, I would have no quarrel with any of the items listed here.

Q The next heading is Chronic Effects and it is divided into two categories, the first being major probable adverse effects and the second being major possible adverse effects. I would ask you to direct your attention firstly to that category listed as major probable adverse effects. Is there anything listed under that category which in your professional opinion is not consistent with the current state of scientific knowledge?

A No, I would agree with all of those.

Q The next heading is the major possible adverse effects of chronic heavy cannabis use which remain to be confirmed by further research and before you answer the question that I've already asked three times, can you tell the Court whether or not the Hall report defined the terms major possible adverse effects of chronic heavy cannabis use?

A No, I don't believe it did define probable and possible. I think it left those to the commonly understood English meaning of those words. It did define, I believe, adverse effect in much the same manner that the WHO ARF report did and it did have an extensive discussion of what types of knowledge one needed to attribute causality so that that would be a significant factor in relation to what you would call an adverse effect unless you could show that there was some sort of causal link, you could not really call something an adverse effect of cannabis.

THE COURT: Ah, well. Then how does one read that remaining clause in that statement, "The following are the major possible adverse effects which remain to be confirmed?"

A Yes. Well, I think that is exactly what they're saying, that one cannot be certain that they are, in fact, adverse effects. Possible not in the sense of this can cause it, but it is possible that it causes it. In other words, information is needed.

THE COURT: What elevates an event to the level of possibility from a scientific perspective? I mean, let me give you an example. If I were to set out a hypothesis, moderate consumption of cannabis slows the aging process, that might trigger some legislature, considering the average age of our legislators. Some research is done and indeed, there is some association in the sense that moderate users seem to go grey slower, go bald slower, subjectively appear to others to be younger. Another report comes out and criticizes that in terms of methodology, is there any causal connection, and maybe they just happen to have less stressful lifestyles in general, so the first study is criticized in the sense that can you draw any logical inferences from it. Is that now on the table as a possible effect, because we've raised it as a hypothesis, we've tested and we haven't been able to confirm it one way or the other, is it now something—a possible effect that remains to be confirmed simply because we put it in the form of a hypothesis?

A Yes, I see what you mean.

THE COURT: Or is there something in the research that I've gone through or—

A No.

THE COURT: -- by the word possible means something more than that?

A No. I think what is meant in the present context, judging by the examples which are given her or the items which are listed here is that there is enough evidence to make one feel that it conceivably may be. It cannot be dismissed and therefore further mechanistic studies are needed in order to clarify whether these really are consequences of the cannabis use or not. In other words, if someone says the moon is made of green cheese and the rest of the world says no, it isn't and the astronauts have not found any green cheese on the moon, that would not be listed here as a possible scientific fact. There has to be some credibility of the association, plus enough evidence of the linkage to warrant more careful analysis and I think the important thing is what the possible means, in other words, is it referring to an adverse effect which may occur. Is it a possible effect or is it a possible effect of cannabis use? Is the possibility relevant to the causal link?

THE COURT: All right. Thank you.

MR. DOHM:

Q The major possible adverse effects of chronic heavy cannabis use are listed and you can see them. Does that—is there anything in the statements that are contained in that paragraph and the possible adverse effects that is not consistent with the current state of scientific knowledge?

A The only one that I think there is not a very compelling reason to continue entertaining as a real possibility is the leukemia among the offspring of women who used cannabis during pregnancy. If I may come back, Your Honour, to the question you asked, I think the next item, decline in occupational performance, may help clarify what I was trying to get across. There is ample observation, there are many observations that chronic heavy, and by that I mean daily and often multiple daily use, users who at that level do show impaired school performance, impaired work performance. What makes this a possible adverse effect is the possibility that the cannabis use is the cause rather than a coincidental phenomenon. The phenomenon is real, but the question that is being asked is the possibility that—of the causal link between the use and the observed behaviour.

THE COURT: And your position would be that there are at least some studies that give some credence to—

A Enough to—

THE COURT: -- the causal link—

A Yes.

THE COURT: -- as opposed to the mere association of those two variables.

A Yes, enough to make it—to warrant a different type of investigation which can establish change by prospective observation.

MR. DOHM:

Q Would it be fair to describe that as being the identification of an area where there is enough evidence to justify further investigation?

A Yes.

Q Nobody is suggesting that these things are proved.

A That's right. I was just going to say, the things that are considered probable also warrant further investigation, but the possibles are ones in which there is enough uncertainty about a causal link but enough real concern about the event itself to warrant a look, a closer research to establish whether there is a causal link or not.

MR. DOHM: Does that answer your question satisfactorily? It might not be the answer that—it might not be as simple an answer as you were looking for, but—

THE COURT: No. I'm certainly getting closer to understanding it. I know how one might react to those words in the common sense terms, but I just was uncertain as to how scientists were using them and I think I have a clearer picture of that now.

MR. DOHM:

Q Doctor, did the 1994 World Health Organization committee on possible adverse—or adverse effects of cannabis, what was the correct name of that committee, please?

A It's the Committee on Health Implications of Cannabis Use.

Q And can I just address that as the 1995 committee, is that all right? Did the 1995 committee deal with the possible adverse effect of an increased risk of leukemia among offspring exposed while in utero?

A It was discussed at the meeting and considered not sufficiently convincing to warrant being mentioned as a seriously-entertained risk in the report.

Q Overleaf, at page X, there are a list of high risk groups. Is there—are the listings of high risk groups contained on that page which include and in fact are described generally as adolescents, women of child-bearing age—no, let's do it one at a time so it's a little more fair, I think. Certain adolescents are identified in the Hall report as being at a high risk group. And two categories are identified there. Is that still consistent with the state of scientific and medical knowledge?

A Yes. I think that's a valid statement.

Q Okay. Are there any other groups of adolescents that have been added in there since the Hall report?

A No, I don't think so.

Q The next high risk group identified is women of child-bearing age; that describes two groups. Now, does the Hall report there still accurately reflect the state of scientific and medical knowledge?

A The 1995 committee agreed with the first one of the risks that may be consequent upon the mother smoking cannabis during pregnancy or during critical periods of

fetal development. There was not much support for the idea that smoking of cannabis at the time of conception might pose a significant risk.

Q The third category is persons with pre-existing diseases and there are four subcategories of that. Does that list of categories still reflect the state of scientific and medical knowledge?

A They reflect the present state of medical and scientific concern, I wouldn't say knowledge, because as I pointed out, the first one, individuals with cardiovascular diseases such as coronary artery disease, there's really been no significant body of evidence presented that such individuals are, in fact, at risk. It's a theoretical or a hypothetical risk.

Q Does that remain then a valid hypothesis?

A Mm hmm.

Q And the others?

A The others, I think, are consistent with current thought.

Q During the course of preparing for this case, I asked you—one of us asked you fairly recently if you would have available to you statistics relating to the current potency rates of cannabis and you obtained rates of potency for the Ontario region, is that right?

A Yes. That's correct. These were from the Ontario Laboratory of Health Canada, the Health Protection Branch sample control office.

Q The numbers that you have produced come from 1975 through to 1996, is that right?

A That's correct.

Q And the document that you have given to me, was that produced by you or was it produced by Health Canada?

A That was produced by Health Canada.

Q There are some handwritten numbers on the bottom of the document for the years 1995 and 1996.

A Yes, that's my handwriting, because I have another document from Health Canada which gave figures separately for 1995 and '96, which included not only marihuana which is what the first sheet deals with, but also some information on cannabis resin and cannabis resin liquid, hash oil, and I took the marihuana data and added them to the list of the other marihuana data to make a single composite.

Q That second document you're referring to is similar to the one I'm holding up now?

A That's correct.

Q The only way that the first document has changed is that you have—

A Transcribed.

Q -- transported the information from it from part of the second document to the first?

A That's correct.

Q For convenience sake.

MR. DOHM: I'm going to tender the two documents jointly as the next exhibit, please.

THE COURT: All right.

THE CLERK: 29, Your Honour.

THE COURT: 29.

MR. CONROY: 29?

THE COURT: Still?

THE CLERK: The last one was 28 from yesterday.

THE COURT: What was 28?

THE CLERK: 28 was one typed copy of curriculum of Dr. Harold Kalant, total of forty pages dated 27th January.

THE COURT: All right.

THE CLERK: 29.

EXHIBIT 29 - TABLES OF MARIHUANA POTENCY

MR. DOHM:

Q I'm going to ask you to just quickly deal with these, Doctor. Firstly, I'd ask you to direct your attention to the year 1975, and the average THC sampled for 1975 in Ontario appears to have been .5 per cent. Do you have a comment on that?

A Yes. That's a bit on the low side, because the samples which I obtained from the R.C.M.P. for purposes of extraction to use in our own experiments had a content about 1 per cent. The samples varied from a little below to a little above 1 per cent, so the .5 per cent seems a bit too low and particularly since the high is shown only as 0.6 per cent, I suspect that that must have been a small number of samples that were not typical of what was generally available.

Q One might perhaps make a similar comment about the average recorded for 1976?

A Yes, which was even lower. But perhaps significantly, the higher point is much more in keeping with the range that we found, which was 1.2 per cent.

Q Are the balance of the figures then commensurate with your experience?

A Yes. The remaining figures are all fairly consistent with what I or my colleagues at the ARF or in other research labs have found and with the samples that NIDA was distributing for experimental use in the United States. They're all for the next few years, they're all around or above 1 per cent.

Q Up to about 1983, they stayed around 1 per cent then, I would take it?

A Yes.

Q I take your evidence to be. And there's been a fairly steady growth with some anomalies in—since then?

A Yes. It's—one shouldn't be surprised by some anomalous values, occasional fluctuations, because this is after all seized material and it depends to a considerable extent on where that material came from, how the material was handled after seizure, how long it was between seizure and analysis and so on, so that

some variation is not unexpected, but the overall trend has been upwards.

Q Anybody interested in playing with these numbers could develop some pretty strong multipliers by comparing, for example, the 1975 averages with the 1996 high, but that would be distorted?

A That would be distorted.

Q Simply, to look at it from about 1980 to currently, we have on the average an increase in potency of how many times?

A About four and a half times.

THE COURT: How many samples are being tested?

A The number of samples is shown in the 1995/6 and 1996/7 two hundred and twenty-one samples of marihuana, forty-three samples of hashish resin, ten of hashish oil and then a hundred and thirty-three, thirty and eleven respectively.

MR. DOHM:

Q You have had an opportunity to read Exhibit 27, being the manuscript that Dr. Morgan presented during the course of his evidence?

A Oh, yes. Yes, I have.

Q Is there anything in the Zimmer and Morgan manuscript which would cause you to rethink your understanding of the combined scientific opinions expressed in the 1991 committee report, the Hall report or the 1995 report?

A I can't think of anything that would cause me to change my interpretation. There were understandably differences of interpretation between Dr. Morgan's handling of the data and my interpretation, but I don't think that that in itself requires any change of assessment of the overall body of evidence summarized in the three reports.

Q Were you present yesterday when Dr. Morgan described cannabis as being—I'll paraphrase here, but the most studied drug in the universe in the last twenty years?

A Yes, I heard that statement.

Q Do you agree with that?

A No, I don't.

Q Why do you not agree with that?

A I—as part of my response to the Hall report, I was asked to write a critique for the British Journal of Addiction and my overall response was that it was a very good report, but that it was disappointing how little really fundamental new research there had been in the long interval since the previous WHO report, and in preparation or in justification of that statement, I counted all the publications listed in the cumulative index medicus, which is an index that lists virtually all of the publications in the world in the clinical and scientific literature—

Q Would that include editorials and opinions?

A Yes, it includes editorials and opinions, but principally it includes published studies and the total of all of them, that is studies and opinions and comments and so on, I graft in—on page 763 of this collection of commentaries on the Hall report there's—

Q Do we have a copy of this for the Court? I do believe that Mr. Conroy has a copy.

MR. CONROY: We diligently searched overnight for that.

MR. DOHM:

Q You're going to direct us, Doctor, to—

A Page 763.

THE COURT: Do you wish this filed as an exhibit?

MR. DOHM: We should. He's going to give evidence about it, Your Honour, please.

THE CLERK: That's 30 then, Your Honour.

THE COURT: Exhibit 30.

EXHIBIT 30 - CRITIQUE OF HALL REPORT

MR. DOHM:

Q And what do you have to tell us about what is now Exhibit 30 at page 763, Doctor?

A The graph, Figure 1, illustrates the number of publications dealing with cannabis each year from 1971 up to 1994, which was the time of the Hall report and the thing to pay attention to is the dramatic increase in the number of publications from a hundred and fifty in 1971 to between three hundred and fifty and four hundred in 1976 and then the sharp drop again after 1977 back down to a level of—that has drifted up and down between a hundred and a hundred and fifty a year, and the slight apparent increase from 1990 onwards is in part fictitious because in that year the index medicus adopted a different system of cross indexing, so that the same paper might be listed twice under two different indexing terms, so that the slightly increased figures after 1990 are probably not valid. The publication rate has, in fact, remained at or below a hundred and fifty a year.

Now, in contrast there are many more than that in one month for alcohol. Alcohol has far more publications each year than cannabis does and there are more on cocaine than there are on cannabis. There are more on opioids than on cannabis. I would say overall, cannabis has been one of the least well-studied, except for that short period between 1971 and '77 when government funding provided a strong stimulus to research on cannabis and when that disappeared, the level of publication fell back sharply. You can see the abrupt drop from '77 to '78.

Q So does that have any relationship to the current degree of—the current status of scientific and medical knowledge in the area in your view?

A Yes, I think it is directly related. I think that the reason that many questions remain unanswered is that there has not been the degree of stimulus to and support for research on these questions that would be necessary to answer many of the questions.

Q I'm getting to the point where I'm going to be summarizing Dr. Kalant's evidence, so I haven't been paying any attention to that device behind me and I'll leave that to others. The—Doctor, there are main areas of concern that appear from your evidence to be common to the medical scientific community when it comes to the adverse consequences of cannabis.

A Yes.

Q Can you briefly just list those and summarize them?

A Yes. In terms of acute effects, I would say there's the obvious concern with the effects of cannabis intoxication and its effects on psychomotor performance with the practical implications for driving and for flying or operating complex machinery of any kind. There is also the potential—there is still concern with the reasons for and the potential risks of the precipitation by cannabis of psychotic breakdown in people with either a known or a predisposition to schizophrenia. There is concern in terms of the effects on the intellectual and social maturation and adjustment of teenagers because of the demonstrated effects on memory, learning, motivation for purposeful activity. There is concern in the chronic—with respect to chronic administration about the degree of damage to the pulmonary system and the interaction between cannabis and tobacco in relation to such damage. There's concern with the possible production of—or at least of malignancy, principally of the upper airways and the upper digestive tract. There is concern also about the possible effects on the immune system which as I indicated in my testimony are not conclusive but are persistent enough to be worrisome. There is also, I should point out, by the same token, a potential therapeutic interest in that because with the demonstration that it depends upon an action through specific receptors, there is the possibility of developing a derivative of THC which will have more selective and more potent action on the immune system to permit its medical use as an immunosuppressant in transplants, for example, or in autoimmune diseases, and conversely there's also the possibility that one may be able to stimulate the immune response in some way that might be valuable in AIDS, so this is an area which certainly warrants continued investigation. And there is—there has been—I didn't mention in my testimony but there has been concern about the duration of the acute effects and the possibility of hangover effects. A number of papers have demonstrated persistence of disturbed function on driving or flying simulator tests for periods as long as twenty-four hours after a single use and that raises the question as to whether there can be a risk factor not only during the acute action of the drug, but also in the period one would normally—when one would normally expect it to have disappeared.

There is also continuing concern about—for defining the magnitude and duration and the nature of the interactions between cannabis and alcohol, cocaine and other drugs which are frequently taken together with cannabis by a certain fraction of users. Again, there are studies showing for example with cocaine the simultaneous administration of cannabis and cocaine increases the speed of onset of the cocaine action and the intensity of the high produced by the cocaine and that also is something which requires further careful examination and I think finally, I would mention the long-term effects,

the question of reversibility of mental effects after a user stops and the question related probably to that of whether or not there are significant residual developmental effects in the offspring of women who have smoked cannabis during pregnancy.

Q Have you listed those in any particular order of priority or are you just simply listing them—

A No, I'm afraid I've listed them largely as they occurred to me.

Q Very well. This action with cocaine, is that the additive action that you have described before or is that some other action?

A No. This is a potentiating effect which has been attributed by the investigators to an effect of cannabis that increases the ease of absorption of cocaine through the nose when it's sniffed, so that the effect is not an addition of a cannabis effect to the independent effect of cocaine, but rather facilitation by cannabis of the onset and therefore the speed of effect and the intensity of the maximum effect from the cocaine.

Q I'd like you to bear in mind your history of studying this substance, your involvement in it, your professional qualifications, both as a scientist and as a doctor. I'm going to ask you whether in your opinion it is supportable medically and scientifically to state that there is minimal or no risks to an individual or to society from cannabis use.

A No, I would not agree with that statement.

Q It is not supportable then?

A It's not supportable.

THE COURT: It's a kind of a double-barrelled statement. Can we deal with individuals. Minimal or no risks to individuals? Is that supportable in your perspective?

A I believe that my answer applies equally to the individual and to society, because of the—the demonstrated effects that have already been well-documented in the view of the 1995 committee. For example, if one takes the effects on driving automobiles and on the possible effects as a contributor to vehicle accidents, that represents a risk both to the individual who is using the drug and driving and a risk to society because of the potential involvement of other people in

accidents who were not using it but are bearing part of the cost of the accident.

MR. DOHM:

Q Again, emphasizing I have been asking for your professional opinion, your expert opinion throughout and emphasizing that as an expert witness you were brought here for the purpose of assisting the Court and not for the purpose of taking one side of a debate or another, I would like you to tell the Court whether or not you are able to give an estimate, not numerically, but an estimate of the position of the greater part of the medical and scientific community on the issue of harm or risk to the individuals or to society from cannabis use.

A I'm not sure that I understand the question clearly.

Q Is—we have seen that there are divisions of opinion in the area of risk attributable to cannabis use. Is there in your professional opinion a preponderance of opinion that would take the position that cannabis use is likely to cause minimal or no harm to individuals?

A The only way I can attempt to answer that is by contrasting the opinions, for example, there was an editorial in Lancet, another editorial in the B.M.J., British Medical Journal which took somewhat different stands and both of them were very much different from the official expressions of concern by agencies such as the American Medical Association, the Canadian Medical Association or World Health Organization and so on. The one in the British Medical Journal simply said that it's highly unlikely that drug use will ever be stamped out and that legislative change may be advisable in order to minimize the social harms produced by it, but in no sense did it claim that there was no—no harm produced by the drug.

On the other hand, the editorial in Lancet began by saying the smoking of cannabis even long-term, is not harmful to health. That was unsupported by any references at all and I was quite astounded because such an opinion first of all represents the opinion of the individual writing the editorial and not of the journal, and secondly, one expects a medical or scientific journal to have evidence to back up a statement of that kind in the form of references to published work that justifies the statement, so I find myself unable to give any credence to that, simply because it represents one person's opinion, an anonymous person's opinion, without any reason being given to support the view.

On the other hand, the medical associations of most of the Western countries, the World Health Organization, have all indicated that there is ground for

concern. They do not agree on the magnitude of the problem, that is they don't attempt to say whether it is grave or is moderately serious or a potentially serious problem, but they all indicate that it is something which requires medical concern and that it should not be ignored as a source of threat to health.

MR. DOHM: I would—

THE COURT: Can—

MR. DOHM: Sorry.

THE COURT: Are you going to go back to your original question?

MR. DOHM: I was going to actually ask for a brief adjournment at this stage, Your Honour, because I'm nearly done. I had not planned to go back to that original question. I'm content with his answer.

THE COURT: I'd like to—

MR. DOHM: Sure.

THE COURT: -- ask the question perhaps in different words. There are clearly divisions of opinion as to whether or not there are risks of harm attributable to cannabis use.

A Yes.

THE COURT: In the medical scientific community as a whole, does the weight of opinion fall on one side or the other of those two opposing opinions?

A I would have—I would have to say that the weight of opinion is on the side that there is a significant health hazard.

THE COURT: Thank you.

MR. DOHM: With that, being about—just a few minutes before three, is that when you normally break?

THE COURT: Yes.

MR. DOHM: Could we take the afternoon adjournment now, please?

THE COURT: Yes, we could.

MR. DOHM: Thank you.

THE COURT: Fifteen minutes. Thank you.

(WITNESS ASIDE)

(PROCEEDINGS ADJOURNED)

(PROCEEDINGS RECONVENED)

DR. HAROLD KALANT, recalled, testifies as follows:

MR. DOHM: Your Honour, recalling Regina versus Caine. I've concluded my examination in chief of Dr. Kalant.

THE COURT: Thank you.

CROSS EXAMINATION BY MR. CONROY:

Q Dr. Kalant, you started your studies into cannabis particularly, I think you said, beginning in about early '70's, 1970, '71, is that correct?

A That's correct.

Q And it was at about that time that you published the book I think some reference was made to that you and your wife together co-authored, Drugs, Society and Personal Choice?

A That's correct.

Q As I understand it, that book was published by the Addiction Research Foundation and essentially was in and around the time of the LeDain Commission, when it was going on, and during that whole period to further stimulate discussion and so on in relation to the—not just cannabis but non-medical use of drugs—

A That's correct.

Q -- overall, isn't that—

A Yes. This book arose because the preliminary report of the LeDain Commission had been released. They asked for and I think deserved a good public discussion and

the government of Ontario, the Minister of Health of Ontario asked in what way the government could facilitate or could contribute to the discussion that the LeDain Commission had asked for and this was our contribution to that response.

Q And in that book you dealt not only with the question of the impact of various drugs on either individuals or through their use on others, but also on various social policy questions, didn't you?

A Yes.

Q In terms of government's role and the role of the judiciary and so on?

A Yes, the intention was to clarify the elements that go into the policy decision by pointing out all of the different considerations that have to be taken account of in deciding how a society reacts to drug use.

Q And then some ten years later, and we're talking roughly ten years, not exactly necessarily, that's when you were involved with the Addiction Research Foundation World Health Organization 1981 report that's been referred to extensively, Exhibit 5 tab 1?

A That's correct.

Q So we have approximately ten years pass before you were involved in a process which reviewed specifically what the current state of scientific knowledge was in relation to cannabis specifically?

A Right.

Q And then approximately ten years after that, I'm sure you were involved in many things in between, you were then a witness in a case involving a Mr. Hamon in the Province of Quebec?

A That's right.

Q And you testified there in October of 1991, didn't you?

A That's right.

Q And you recall testifying there and mentioning that by that time, 1991, we had accumulated a considerable more knowledge and information about cannabis than you had fifteen to twenty years earlier?

A Yes.

Q And the same, of course, was true by the time you testified in Hamon insofar as the ARFWHO report is concerned. Over that ten years, even though you've said there wasn't as much research as had been done previously, we still had accumulated a --

A There has been some—

Q -- reasonably large—

A Yes.

Q -- amount of additional knowledge that we didn't have before?

A Yes, a reasonable amount. As I said, not as much as has been accumulated with respect to other drugs such as alcohol or cocaine, but still a significant amount.

Q And now you've told us that the—not the ARF but the WHO process has been revisited and that's been ongoing, as I understand it, up to 1995, at least my friend's been referring to it as the 1995 report, but am I right in understanding that that's when most of the work was completed and it's now awaiting the feedback, I think you've said, from other people?

A Yes, that's correct.

Q Now, did I understand it correctly, it was sent out once for feedback from other people—

A Yes.

Q -- came back, a revision was done and it's been sent out again?

A That's correct.

Q Was that done with the '81 report?

A No. The '81 report didn't have as extensive a consultation process.

Q Okay. And it's been referred to as still in publication, it's not available yet for the public to read, I take it?

A That's correct. Unfortunately, it's not available yet.

Q You've referred to it quite extensively in giving your evidence and you do have a copy of it here or a manuscript?

A I have a draft, I guess an early draft.

Q Is there a late draft, a draft that exists prior to it being sent out to the last group of experts?

A Yes, there's a later version than that which incorporates many of the changes that reviewers had suggested or that arose from comments that they had made.

Q And is that available for our purposes here in these proceedings, or is there some restriction on its release?

A I regret that it's not, because until the WHO releases it, it can't be considered an official WHO document.

Q Okay.

A What I have described to you is my account of what happened during the discussions which I chaired.

Q All right. So we can't take it at this point then to be a WHO endorsed as its position then, can we?

A No, I don't think we can.

Q Okay. Now, is that because there's some considerable debate going on as to whether it should be put out as a World Health Organization report amongst the various people who are involved in it?

A I would say no, there's remarkably good agreement among those who are involved in the preparation of it.

Q Yes?

A There has been what is in my view extraneous considerations that come into it that have perhaps contributed to the delay in publication.

Q And those extraneous considerations involve other scientists, doctors, pharmacologists, disagreeing with its contents and feeling that it shouldn't be put out as part of the World Health Organization report, isn't that correct?

A I'm not sure whether it's appropriate for me to paraphrase what I think other people's responses are. Perhaps Your Honour could instruct me on that.

THE COURT: Seems to me you've already done that in terms of presenting to us the general opinions of the people that you've been involved with in preparing the draft, in the drafts.

A And that therefore it's permissible to say—

THE COURT: I think the question—

A Yes.

THE COURT: -- is permissible and certainly the answer in this context.

A Very good. Well, I would say that the main factors that have delayed publication have been concerns on the part of some of the respondents or reviewers who interpreted it as being pro or con legalization. I mean, they did not look at it as a report on health issues, which was its stated purpose, but looked at it as something that might have significant impact in terms of their respective governments' policies and were concerned that the report could be seen as being either overly harsh on cannabis or overly soft on cannabis and favouring legalization and I think it's essentially considerations of this type that delayed the publication.

MR. CONROY:

Q And do we know who those people are and what governments they represent—

A Unfortunately not, Your Honour

Q -- in terms of—

A They were anonymous comments that were forwarded to us.

Q Now, is this in that first circulation or is it in the ongoing one, the one that's going on?

A No, that was in the first circulation, and we replied to it by pointing out that we were concerned exclusively with the assessment of the literature on health consequences and not on any implications that these might have for one or another policy and we don't know

how those replies have been received until we get feedback from the second consultation.

Q So let me see if I understand this correctly then. The group that comes together to do it, are they picked by somebody in particular from the World Health Organization or are they put up by their respective governments—

A No, they weren't.

Q -- or how does it come—

A They're picked by the World Health Organization.

Q And is there somebody in particular who does that or do you as chairperson get to do that or—

A No. No, I was—

Q Selected.

A -- chosen to chair it after the selection was made.

Q And then—and it's how many people roughly?

A There were about fifteen. There were people from—there were several from the United States, there were three from Canada, there were two from Australia, there were two from India, there was one from Hungary. Where else? There was one from the U.K. How many does that add up to, I haven't—

Q I'm sorry, I didn't count them.

A In any case, it's an international group which is chosen by the director and secretariat of the corresponding section of the World Health Organization that is part of the Mental Health programme that includes the group concerned with matters of health problems or public health aspects of drug use.

Q How many from the United States?

A There were either two or three. I believe there were three.

Q And do you know who they are? Can you give us their names?

A Well, Dr. Martin was one, William Martin or Billy Martin, Dr. Hartell, Christine Hartell was another, and I'm trying to think who the third was. Unfortunately, this draft doesn't identify any authors with—associated with particular sections. I'm quite sure there was another, but I'm trying to recall who that was. Sorry, I can't remember who the third was.

Q We could have—

A The three from Canada were all from the Addiction Research Foundation. They were Dr. Reginald Smart, who is an epidemiologist, Dr. Corigal (phonetic), Willian Corigal who is a behavioural pharmacologist, and myself.

Q I see. Dr. Smart testified in the Hamon case too, didn't he?

A I'm sorry?

Q Dr. Smart, it's Dr. Reginald Smart?

A Reginald Smart is an epidemiologist.

Q He was a witness in the Hamon case in Quebec too, wasn't he?

A Yes, he was. Yes.

Q Were there any from Jamaica?

A No.

Q Were there any from Costa Rica?

A No. There are—really, it's very difficult to identify scientists from either Jamaica or Costa Rica whose work would be—would fit the mandate of the group. Discussion was certainly made of the results of those studies, but there were no scientists from those countries on the committee.

Q Germany? Germany?

A No, there wasn't from Germany.

Q Italy?

A From Finland, no.

Q Italy? Greece?

A There was on the first one, not on the second.

Q South Africa?

A No.

Q Malaysia?

A No.

Q Okay. Now, a number of these countries that I've mentioned, you know from your experience that there's quite widespread cultural—there has been quite widespread use of cannabis as a cultural --

A Yes.

Q -- factor, isn't that right?

A The problem with respect to the composition of the committee is that it's difficult to identify scientists from many of those countries who have a continued interest in problems related to drug use. There is one in Malaysia, for example, who is a very solid, very well-qualified scientist who has participated in other WHO groups that I have been on, but he was—I don't know why he was not selected for this committee.

Q When you went—you referred to the current report a number of times and you mentioned a number of studies but not always by year or by date. Are you able—and I'll go to the specific ones in my notes, are you able to give us that kind of information from the report, if we—so that we can know exactly which studies were referred to and considered and so on?

A I think probably I can. I can just check whether the bibliography listed is listed here. Certainly some of them are, yes.

Q Now, do you know if there's any possible way that we could obtain some kind of release of the document for purposes of this case, given the extent to which you have been involved in the process and its ultimate conclusions and the extent that you rely upon?

A The only way I can think of is by direct communication with the director of the corresponding division with a request that a copy be released in its present form for

the Court's purposes. I don't know what the response would be, but I can't see any problem with—I mean any reason not to try it.

Q Okay. And where would we have to communicate? Is that New York or is it—

A No, that would be Geneva.

Q In Geneva. Okay.

THE COURT: I'm sorry, Jamaica?

A Geneva.

MR. CONROY: Geneva.

THE COURT: Geneva.

A Switzerland. It's the home base of the World Health Organization.

MR. CONROY:

Q It ties in the United Nations, doesn't it?

A No. The World Health Organization has working relations with the United Nations, but it's not an agency of the United Nations. It's a separate international organization which was set up at the same time.

Q Okay. And who would we have to contact?

A Dr.—probably the best person would be Dr. Mario Argandona, A-r-g-a-n-d-o-n with a squiggle over the n-a.

Q Okay. Would you be able to do that? If it came from you, I imagine it would be much more persuasive in being able to get it released for us, given that you've relied upon it?

A I would have no problem writing to them to request permission to release the report in its present state.

Q Okay. Could we telephone them, do you think?

A Yes. I can't give you the number here, but on return—

Q No, no, maybe—

A -- to Toronto I would certainly be able to give you his telephone and fax number.

Q All right. Thank you. I'd ask you if you could do that, maybe you could give it to Mr. Dohm and either try to arrange it and then have it produced to Mr. Dohm or alternatively, if that doesn't work, to have him pass the information on to me and I'll see what I can do.

A Certainly, I'll be happy to do that.

Q Thank you. The information you produced today from Ontario, I think you said came from Health and Welfare Canada, correct?

A Yes, the Health Protection Branch Regional Laboratory.

Q And I assume that they have the same sort of data for each province, do they?

A I would think so. Not so much by province as by region, because it depends—this comes from the laboratory for Ontario. There's a laboratory for Quebec, there's a laboratory for the Atlantic region, there's a laboratory for the Prairies, there's a laboratory here in—I believe in New Westminster or somewhere near Vancouver.

Q They call it the Pacific region, I guess.

A Yes.

Q Okay. So we ought to be able to obtain the same kinds of statistics in terms of THC levels and—for the whole country?

A I would think so.

Q And as I understand it, these basically represent police seizures of cannabis which are then sent to the lab, they're analyzed perhaps for court purposes or others, but they keep the statistics and the—

A That's correct.

Q Yes. Okay. There's no indication where the cannabis came from—

A No.

Q -- whether it came from the Pacific region or from the Atlantic region?

A Oh, no, no. It would come from the region served by that laboratory. In other words it would—

Q It would be seized there?

A Be seized there.

Q But we have no way of knowing whether the cannabis itself came from out of the country—

A No.

Q -- or in the country or anything like that?

A That's quite true.

Q Because, you see, Dr. Morgan told us about a project in Mississippi, I forget the name of it, but it was a—

A VOICE: (Indiscernible).

MR. CONROY: Sorry?

A VOICE: Potency monitoring project.

MR. CONROY:

Q A potency monitoring project. You probably read about that in his manuscript.

A Yes.

Q Are you familiar with that project?

A No. I know that it exists but I'm not familiar with where its samples come from or who refers samples to it.

Q Well, my understanding is that they grow the marihuana for—

A Oh, well that's different.

Q Are you familiar with a different project in Mississippi?

A Yes, there has been for some years.

Q Well, maybe I'm mixing the two up.

A I believe so, yes.

Q Okay. Well, are you familiar with the one that grows for the U.S. government?

A Yes, there has been a group at the University of Mississippi at their experimental farm, I believe, that has grown marihuana of different varieties over quite a few years and have prepared it for NIDA and other U.S. government agencies and one of their concerns has been how the growth conditions affect the potency in terms of the THC content.

Q And my understanding of Dr. Morgan's evidence, both in relation to—as I understood it, the Mississippi project had three different aspects to it but it was essentially one group at the university. Now, maybe I misunderstood him, is that your understanding?

A Well, if you were—I mean, in relation to other aspects that you may have discussed with him, I really don't know whether—who was involved in the various aspects. I know only—I'm acquainted only with the programme that grew marihuana and tested the potencies of different strains and different growing conditions and so on.

Q I'm trying to remember the name of the person that was—

A I believe Coy Waller was one of them.

Q And there was another name, but I'll—I'll dig it up and have it for you tomorrow. Okay. I want to then go through the evidence that you've given, first to be sure that I understand exactly what you've told us so that I can then compare it to the other evidence that we've heard and see just where we possibly differ. First of all, just to touch on your curriculum vitae, you've done approximately on my count, and I may have miscounted, about—I think you told us this, twelve journal publications relating to cannabis, is that right?

A Twelve experimental studies related to cannabis.

Q A great many you have done on alcohol?

A Yes.

Q And your involvement in relation to tobacco isn't quite as clear, but I take it you've had a—

A No, I've done—I've not worked on tobacco.

Q Not much on tobacco at all. Okay.

A Sorry, I'll make one correction to that.

Q All right.

A In the study in which we measured the tar content of cannabis smoke—

Q Oh, yes.

A -- we also measured the tar content of tobacco smoke.

Q That was the first study, I think you told us, to do that.

A Yes.

Q And at that point as I recall it, you determined that there seemed to be a lot more tar in the smoke from marihuana than in the smoke from tobacco?

A That's correct.

Q There have been subsequent studies to that, haven't there?

A Yes, there have.

Q And would you agree that the studies now seem to say that the elements in the two types of smoke appear to be substantially the same?

A Qualitatively?

Q Yes.

A Yes.

Q Okay.

A Yes, by and large they are very similar. There was an early claim that there was significantly more carcinogenic material in the—

Q In the marihuana.

A -- in the marihuana smoke, but I'm not acquainted with any recent study that makes the same claim.

Q All right. Okay. And as I recall it there was also something about that time that was significant and that was the method of use by the marihuana smoker, who would take it in deeply into the lungs and perhaps hold the breath and in the result you get more particulates, I think you call them—

A Yes.

Q -- in the lungs that you wouldn't get from the way people normally smoke cigarettes?

A That's correct. Plus the fact that very many cigarettes are filtered while cannabis cigarettes are not.

Q Right. And contrasting that with the tobacco smoker, we know that the usual method of ingestion, as you say, is perhaps a filtered cigarette, but that the consumption is usually somewhere a pack a day or maybe more for most tobacco smokers—

A Yes.

Q -- is that fair?

A A pack a day is twenty.

Q In terms of rates of use of tobacco smokers, is that a fair thing to say, a pack a day?

A Yes, to the best of my knowledge that is so. There—the number of people who smoke small numbers of cigarettes are—as a proportion of all smokers is really quite small.

Q All right. I mean, we've got some that smoke two and up a day, don't we, and we could call those the chronic heavy users, could we?

A Two packs a day?

Q Yes.

A They are the very—I mean, they are very heavy smokers.

Q Okay. And could we call the pack a day people moderate tobacco smokers?

A It's the definition of moderate is flexible, I suppose, but they would be fairly typical smokers.

Q Fairly typical. Or—okay. And less than a pack people obviously would be the light—

A Would be—

Q -- smokers?

A -- lighter smokers, yes.

Q Now, if we try to do that with marihuana, I know you've said this is difficult to do, but it's important to know what we're talking about, isn't it, in terms of rates of use and amount of use. You've told us that's pretty fundamental to one's opinions about the health consequences, correct?

A Yes.

Q So when you do use those terms, heavy user or chronic heavy user, am I right that you're talking about a fairly small stream group of people in the marihuana consuming population?

A Yes. Probably somewhere in the neighbourhood of five per cent or a little more of cannabis users.

Q Now, when you say that, five per cent, is there a document that exists that tells us that, that that's the latest estimate or something like that?

A There are a number of documents that can be consulted for that purpose. The surveys that are carried out periodically on extent of use, one of the unfortunate things is that the earlier surveys tended to be—not to include separate categories for the high end of the scale and in some of the early surveys done, I regret to say, even by the Addiction Research Foundation, some of the things that were called heavy use or the upper end of the question scale were more than once a week, which is not particularly helpful. What one wants to know is once a day or more than once a day. Those are now asked and one can consult the surveys of such groups as the ARF or in the States, the high school survey by Johnson et al, which I believe now—well, I know the foundation ones do, but I believe the Johnson ones also have more useful breakdown at the higher end of use.

Q And can you tell us how they break it down? I mean, do they—do they—let's take Johnson in the U.S., do they break it down in terms of low, moderate and heavy

in terms of how many cigarettes per day or per week or per month?

A No. No, the way they're usually done is to say do you smoke less than once a month.

Q Yes.

A Up to one a week, more than once a week, daily, and the—and at the upper end, the tendency now, I believe, is to ask more than once a day as the upper category.

Q Okay.

A They don't go beyond that, to the best of my knowledge.

Q So could we say more than once a day obviously or clearly falls into the chronic heavy use category?

A Yes, I would agree.

Q Up to once a day from what, once a week is in our middle sort of category?

A I would think once a day also is probably fair to include in heavy use, because of the duration of the drug within the body and on the other hand, if you're once a week or less, there's undoubtedly time for the drug residues to be cleared between exposures, so that I think that's the critical differentiating factor.

Q All right. So it's either at the top of the middle category or the bottom of the heavy category, would that be a fair way to put it?

A Which is at the top?

Q If the heavy category definitely includes more than once a day and we've got a middle category—

A I would perhaps—I would say once a day or more is generally considered heavy.

Q Okay.

A And once a week or less would be considered in the intermediate and then once a month or less would be considered in the very light or occasional use.

Q Okay. So once a month or less is what you would—is the rate of use that you would refer to as occasional use?

A Occasional, yes.

Q And—because you don't believe that marihuana is ordinarily harmful to healthy adults in terms of their occasional use, do you?

A No, I don't believe there's any evidence to indicate harm other than harm that may result from acute intoxication such as the effects on driving—

Q Yes.

A -- or whatever on a single occasion.

Q And just to be clear about that, because I've read a number of different things, be clear about your understanding or your evidence on it, the person smokes the—usually smokes the marihuana cigarette, and these acute effects are what occur usually within the first thirty minutes to an hour?

A No, I would say within—up to a few hours, depending on the amount smoked.

Q But they reach an effect—

A Up to—

Q -- after that first thirty minutes, don't they?

A Yes, they reach a peak usually.

Q And then they start to decline?

A That's right.

Q And they can last an estimated two to four hours, although they've had some cases that have been longer, is that right?

A Yes, there have been, as I described earlier, some studies which report effects lasting for as much as twenty-four hours.

Q And those are quite rare, aren't they?

A I'm not really in a position to say how rare they are, other than that that would imply either someone with unusually slow metabolic clearance or else a very large dose.

Q All right. When you talk about the metabolic clearance, are you including the metabolites in the fatty tissues and so on that I understand can remain there for long periods of time?

A No, I'm referring to the elimination of the tetrahydrocannabinol.

Q The THC itself from the brain and the bloodstream?

A And the circulation.

Q Okay. All right. Now, my friend started off by asking you about classifying cannabis. I take it you'd agree with me that the usual medical definition for a narcotic is the opiates or the opiate derivatives or drugs having that type of an effect, right?

A It depends on what period or what year you're talking about, because the usage has changed.

Q Okay.

A It used to be when I was a medical student, for example, which is going back some years now—

Q Right.

A -- the meaning of narcotic was generally opioid analgesics. On the other hand, in the basic scientific literature the term narcotic was used for any drug which depressed cell functions such as cell excitability or cell membrane transport of essential materials, and alcohol was referred to in that sense as a narcotic, and that was as recently as the 1950's, so there has been an attempt to clarify that in recent years and for medical purposes, to avoid the term narcotic, and to say instead opioid analgesics, which includes morphine, heroin, codeine, the things that are used for the relief of pain or for some other treatment of some other symptoms, and are either derived from opium or are related to it chemically and functionally.

Q Okay.

A And narcotic now tends to be used mainly for legal purposes. It's—the word narcotic no longer is favoured for use in medicine.

Q Back in nineteen—I think it was '22 or '23 when they included cannabis in the schedule pursuant to the Narcotic Control Act, in those days narcotic meant an opiate derivative type of drug, didn't it?

A Not exclusively, no. The term narcotic was used rather loosely to mean any drug that could produce what was variously called intoxication or stupefaction or depression. It was used really loosely enough that at that time cannabis and cocaine and drugs that were really pharmacologically and chemically quite unrelated to opiates were also classed as narcotics.

Q And I thought you said that that—at the time when you were in medical school, that the term narcotic was used for the opiates.

A Yes, which was --

Q So it changed and became that, did it?

A It became that, yes.

Q And then it changed again to become a looser definition.

A Well, now—I would say now the term narcotic is used almost exclusively for legal purposes and it means, I take it, whatever is classed under the law as a narcotic.

Q Because marihuana certainly didn't meet that definition of a narcotic that existed for that period of time when you were in medical school?

A It was certainly not similar to the opioids now.

Q All right. Okay.

A On the other hand, I suppose in terms of its basic biological science use, it did fit to the same extent that alcohol and barbiturates did.

Q Right. And what you have described it as here is essentially a mild sedative?

A I would say yes, that's the best description of it. It's a hypnotic sedative drug which has—with the difference

that in its very early part of the onset of its action, it has some excitatory effect as well, which soon passes over into the sedative phase.

Q You then took us through the definition of adverse effect in the ARFWHO report and in that discussion you made reference to how as an example one might be driving a car and there could be an adverse effect.

A An effect which would have adverse consequences if one were driving a car.

Q Right.

A Might not have any adverse consequences if one were simply sitting quietly smoking and doing nothing else that was threatening.

Q And if one was very angry, for example, that has an effect on our body system, doesn't it?

A Yes, it does.

Q And if you're very angry and you drive a car, that could have some adverse consequences for others too, couldn't it?

A Yes, I agree.

Q But if you were very angry and stayed at home and sat and tried to calm down and not drive or complex machinery or do anything like that, then there isn't likely to be any adverse effect, isn't that correct?

A Well, there still could be, because one of the adverse effects of anger is an increase in adrenalin release, an increase in heart rate, a rise in blood pressure, and it's conceivable that someone who is at home mulling over some offence or whatever and was very angry about it might have an adverse effect due to the high blood pressure.

Q Right.

A But again, it would depend on the situation and on the person.

Q But there are lots of things, aren't there, that aren't prohibited that may have, if we consume them or they affect us in a certain way, that if we go out and drive a car or get involved with complex machinery, that we

could cause an adverse effect in terms of others, wouldn't you agree?

A I'm not sure that I understand the question. You say other—

Q Emotional states—

A -- substances or—

Q Either. Emotional states that affect the adrenalin and—

A Oh, yes.

Q -- so on, or other substances that we might consume.

A Yes. Yes, I—

Q Fair enough? Okay. Now, you talked a bit about rates of use, and I wanted to just put to you some information that's already before the Court that we received that relates to this business of different types of policy approaches and how rates of use have been affected. You mentioned Holland in the later part of your evidence, and it's my understanding, correct me if I'm wrong, while we've heard that we may not know exactly what the rates of use were prior to 1976 when they adopted this new or different approach, am I right in understanding though that—and I think you did say this, that rates appeared to go up after they started the different approach.

A That's correct.

Q And then levelled off, I think you said?

A I'm—I can't really—I'm not in a position to say that they levelled off. All I can say is that in the period of observation that was described in the Dutch report to which I'm referring, they had gone up from the mid-'80's to 1992 and that at the same time other literature indicates that the rates had fallen substantially in other European countries.

Q And did you compare those rates to the U.S. rates of use—

A Yes.

Q -- during the period of time?

A All—in virtually all of the European countries, the rates are less than in the U.S.

Q Because the U.S. rates are very high—

A Yes.

Q -- compared, aren't they?

A Yes.

Q And so while the rates of use in Holland may have gone up after the new approach, they don't come close to the United States' rates of use, do they?

A I believe that's correct.

Q And have you become familiar with other, maybe specific states in the United States where they have different approaches to cannabis possession? Some states, for example, have a traffic ticket type of approach and others don't. Have you looked at that in terms of how those—how rates of use have been affected, depending upon those different approaches in those different states?

A No, I have not. Some of my colleagues at the foundation have and I have—that's not been an area that I'm specifically responsible for. All I know from discussions with them is that the figures have not been very clear. In other words, that the—it has been difficult to interpret changes that over time and different states which altered their laws because there were no studies before, and so that again there's that same problem as in the Dutch studies, that there was no before and after comparison to permit an assessment of the results.

Q You see, the information we have in Exhibit 22, Your Honour, which was letters that were written by citizens to government of Canada representatives seeking information back, in one of them they question and answer cannabis was provided and the question was, and this is towards the back of that set of documents, I think it's about three pages from the back, the particular one I'm looking at, it said,

"Has cannabis use increased since decriminalization?"

And the response was

"Three U.S. states have surveyed the extent of marihuana use following decriminalization. In the four years since Oregon eliminated criminal penalties for simple possession, 1974 to 1977, the number of adults who claim to have ever used marihuana has increased six per cent and the number who claim to be current users has increased one per cent. The usage trends previous to the reformed legislation are unknown."

You didn't know that maybe before, but that's consistent with what you've been saying in terms of not knowing the earlier rates. You're nodding. I just have to make sure you say yes or no because—

A I didn't hear you very clearly—

Q Oh, I'm sorry.

A -- the last—

Q Let me read it to you again. The question that was posed was,

"Has cannabis use increased since decriminalization?"

And the response was,

""Three U.S. states have surveyed the extent of marihuana use following decriminalization. In the four years since Oregon eliminated criminal penalties for simple possession, 1974 to 1977, the number of adults who claim to have ever used marihuana has increased six per cent and the number who claim to be current users has increased one per cent. The usage trends previous to the reformed legislation are unknown."

A Mm hmm.

Q I'll read the rest of it to you as well.

"California compared usage at five months before and at seventeen months after decriminalization. A seven per cent increase was found in the number of adults who reported having ever used marihuana. Those considering themselves current users rose five per cent, although their frequency of use decreased.

A main survey of high school adult users found that at two years after decriminalization forty-eight per cent of the adult users had decreased their usage, thirteen per cent reported an increase and thirty-nine per cent little or no change. Twenty-six per cent of high school users claimed a decrease in use, while thirty-eight per cent reported an increase and thirty-six per cent little or no change.

American data indicate that increases in marihuana use are most rapid among states which maintain relatively severe penalties, i.e., substantial fines or imprisonment for possession of small amounts,"

and a number of references are given.

Did you know that information?

A No. That's not information that I have studied in any depth.

Q Okay.

A I knew in general terms but I knew also the problems that were raised in discussion of these findings by Dr. Eric Single, who pointed out the difficulties in that generally where states have changed their regulations, there has not been, except possibly in those instances, any appreciable previous record to compare with. I would say, for example, seven months before is not a significant time and the other thing he pointed out is that the meaning of decriminalization had not been specified, that what was—that practises that were changed were different in the different states, therefore it was hard to make much out of the impact of what was called decriminalization.

Q We also had some evidence from Professor Boyd, I think it was, and I'll dig up the specifics overnight, but it's my understanding, my memory that in Canada our rates of use in the '60's and '70's was quite high, that then we had a trend of—it went up for a period and then the trend was down for a long period of time and that it's only been recently, in 1991, I think it was, '92, that we've had a slight increase and primarily in adolescents, I think.

A That's correct.

Q So that we know that the rates of use today starting in '91, 1992, while they have gone up slightly, they're still very much lower than what they were back in the '60's and '70's.

A I believe that's correct, yes.

Q Okay. So our rates of use, notwithstanding the increase that we've referred to, is still substantially lower in Canada than it used to be back in the '60's and '70's.

A Yes, I think that's true. That's also true, of course, in the United States. The high school seniors' surveys showed the same trend, an increasing rate during the '70's and then a long period of decline and then an upturn again in the past three years or so.

Q And in Canada, at least, maybe not as easy to judge in the States because of all the different approaches in each different state, but in Canada at least, our law has remained the same throughout this whole period as far as simple possession is concerned?

A That's correct.

Q Yes. So these rates of use appeared to have—there's been this huge increase in the '60's and '70's, but then we had this drop-off, notwithstanding the law, and then this increase more recently—in more recent times, notwithstanding the law.

A Yes. The—I think the surveys from the Michigan group attempted to resolve what the major factors were that contributed to the decline, up to a few years back, and their conclusion was that it rested in large part on the attitudes rather than the question of punishment. The general feeling that it just wasn't a very smart thing to do, that it wasn't particularly good for them.

Q So as a result of education or information being provided to them about the consequences.

A One hopes that's the case, yes.

Q Certainly not from being put in prison, as far as you know?

A No, because most of these were people who had not been put in prison and were not particularly afraid that they would be.

Q Okay. My friend then took you into general toxicity and you said that it—cannabis is not a severely toxic drug, right?

A Yes.

Q You told us that no known deaths in humans are—

A That's correct.

Q We don't have any knowledge of.

A That's right.

Q And that's quite unlike tobacco, isn't it?

A Well, not quite. You see, the—well, no, I'm sorry. You're right. Yes.

Q I mean tobacco—

A It is possible—it is possible to give a poisonous dose of tobacco.

Q Well, I'm told that tobacco kills forty thousand people a year—

A Ah no, but that's not acute.

Q No, no. I wasn't saying acute and I don't know if my friend at that point was talking—was just talking general toxicity.

A But that refers to acute toxicity.

Q All right. So I'm misunderstanding then in terms of—so when you talk general toxicity, you're talking about simply when you take the actual cigarette and can you die from it or after one, two, three or more in one sitting, which could be a long sitting, I suppose.

A That's right.

Q Okay.

A Yeah. I would still have to say you are correct, that tobacco can be more toxic in that sense because it's been calculated that a—if a child gets a cigarette and eats it, a small child can possibly experience fatal poisoning.

Q Right.

A No such claims have been made, to my knowledge, for cannabis, although there have been a couple of published case studies of severe acute intoxication by cannabis in small children, again gaining access to it and eating it and fortunately, they—neither case was a fatal one, but they were in profound coma and depressed respiration.

Q Very young child.

A Very young child.

Q Yes. But if we focus not just on acute factors in terms of immediate consumption, we know in the case of tobacco that as a result of all of the health consequences of tobacco consumption, which again undoubtedly have something to do with rates of use and how often people smoke and so on—

A Yes.

Q -- tobacco kills, doesn't it?

A Yes.

Q And as far as marihuana is concerned, or cannabis, we have no evidence of that, do we?

A This is the problem which I did discuss at some length in direct examination, that we simply do not have the information to enable us to say because while there's reason to suspect or perhaps put it a little bit stronger, there is reason to fear that cannabis may give rise to, for example, bronchial cancer in a manner comparable to the production of lung cancer by tobacco, we don't yet have the long period of observation that's necessary to permit us to state that with any certainty or how many people may be at risk.

Q But in the case of tobacco, we have apparently something like forty thousand people per year dying from tobacco—

A Yes.

Q -- consumption, don't we?

A Yes, that's—

Q And as I understand it, we have somewhat analogous or comparable figures in relation to alcohol, don't we?

A Yes.

Q But we don't have any of that for marihuana, do we?

A No, we don't yet have comparable figures.

Q And we've had reference made to these field studies from Jamaica. We know that they've been smoking

cannabis in Jamaica for a lot longer than thirty years, don't we?

A Yes.

Q We know that people in Africa have been smoking cannabis for a lot longer than thirty years, don't we?

A Yes, we do.

Q We know that they've been doing it in Malaysia as well for longer than that?

A The trouble is that those countries have not had very systematic or good collection of types of public health statistics that you need to assess the impact on health generally in the population.

Q But we know without doing any studies that forty thousand people a year die in Canada from the tobacco complications, don't we?

A Well, no, that was done because of studies.

Q But we now know that that's the obvious cause, correct?

A Yes, that's correct.

Q We don't have a huge group of people dying from a cause that we don't know about in Canada, do we? Numbers like forty thousand dying from some unknown cause that we haven't figured out yet, or do we?

A Well that's not really the question that I would ask. I would say are there people dying from identified causes, the more remote causes of which we are unaware of? In other words, if someone dies of lung disease, do we know how many of those people who die of lung disease contracted that disease because of smoking cannabis and I would say no, we don't.

Q But we do know that we have people in North America who've been smoking cannabis on a daily basis since the '60's, don't we?

A Yes, but we don't know—we have—we don't have studies to say how many of them have died and of what have they died.

Q Right.

A And what has the role of cannabis been.

Q Right. As I understood you in part of your evidence you said that you didn't expect that we would be able to figure that out, partly because the drug is illegal and we can't get users to provide the data, you recall?

A Yes, I think that's correct.

Q And if it was legal, we could get that data and we might be able to figure it out, isn't that right?

A Yes, I think that's probably true.

Q And so when you go to a place like Holland and all of a sudden they change their approach to—in 1976 to one of tolerance to simple possession and use, the first point I'd make is that once that policy changed, it would be more likely that people would admit use, wouldn't you say?

A Among youth, probably so. Among the general population, probably not because it's still not legal.

Q But if there's no penalty for admitting use, wouldn't you agree that people would be more likely to admit use in those circumstances?

A I would expect they would be, yes.

Q That might explain why the rates of use went up, wouldn't it?

A Well, the rates of use that were—that went up were among the—were found in the school surveys and the school students probably all along would be more willing to admit use than the adult population would. That's been—the experience has been in the school surveys in Ontario, for example, that there's generally little or no hesitation on the part of the students to indicate their use. The concerns come mainly in older people who are afraid that the consequences for their careers or whatever may be in jeopardy, so that I don't think the school surveys in the Netherlands would have been—would be as much affected by that as general population surveys which have not been unfortunately.

Q Okay. I didn't understand that before. You're saying that it's the—it was in the school population—

A Yes.

Q -- in the Netherlands that there was the increase, but there was not a comparable increase in the adult populations after the change?

A No, the general population has not been surveyed in the same way as the school populations.

Q So we don't know?

A We don't know.

Q Okay. All right. We've now had, though, this period of twenty years with this liberal policy or approach in the Netherlands and so they ought to have been able to acquire this data in terms of determining the number of people that it might kill and things of that nature over the twenty years, wouldn't you say?

A Well, as I pointed out earlier, one needs not only a suitable length of time, but also suitable numbers of people using.

Q Yes.

A And we're talking about cohorts of students that have started using at different times within that twenty year period, not about very large numbers of people who have used for twenty years, so I would not expect that even the Netherlands would yet be able to provide us with the kind of data that would help to answer some of these questions.

Q So how long more in the Netherlands where they have this policy, do you think it will take for—how many more years do we need in the Netherlands to be able to come up with some of these answers?

A I think we—I would still stand by my previous answer. I think we will need at least another twenty, thirty or possibly more years, because of the numbers who are using are not large, as was pointed out, compared to the United States, the numbers are not large and therefore they don't generate a large enough population at potential risk to be able to assess the outcomes.

Q But when we talk large versus not large, what figure do you have in your mind in terms of numbers of users in the Netherlands when you say that?

A Well, what I can say is that for the public health statistics relating to tobacco or alcohol, we're talking about hundreds of thousands or millions of people.

Q Right.

A In the case of cannabis, we're talking about very limited surveys which yield data on for at the most tens of thousands of people and for relatively short periods of time.

Q Okay. And so if you have tens of thousands of people smoking marihuana on a regular basis over a twenty year period, you would expect that that then—we'd be able to tell something at the end of the twenty year period, would we, in relation to—

A We would be able to tell something about high frequency complications, not about low frequency, and that's the whole point. I think one has to recognize that when you're talking about an event that occurs to perhaps what, two or three per cent of the population, then in order to have significant conclusions about the risk, you need to have many more than tens of thousands of people of whom only a small fraction have been using for twenty years. If we're talking about a student population growing up over the twenty year period, only one or two groups of them, quite small numbers, will have used for twenty years. So that this is why I keep insisting that you need large scale observations over a long period of time before you have the data that permits you to draw reliable conclusions.

Q And you would require a change in policy, so that you would be able to get the data in order to do the experiments properly and adequately, isn't that right?

A It would probably be easier if the policy were different.

Q Well, there's large restraints on being able to do a lot of the research because of the prohibition, isn't there?

A What sort of research are you referring to?

Q Well, how about the National Institute of Drug Abuse research in the United States? They don't give out money very often to study the positive effects of cannabis use, do they?

A I'm not in a position to say now how much of their money is spent on cannabis, but there was a period in which they did give quite a lot and—

Q But not to study—sorry, I interrupted you.

A And then that diminished after time that I showed in my graph.

Q Right.

A I'm not privy to their total expenditures now on cannabis compared to other drugs, so I just am not in a position to answer that.

Q Have you not observed that in your period of time with the Addiction Research Foundation or with the—in conjunction with your experience with the World Health Organization that the American funding agencies particularly are looking for harms and problems and not for any positive benefits from cannabis and that if you as a researcher come up with some positive result, you're not likely to get funding again in the future? You haven't observed that?

A No. The—certainly in the recent work on mechanisms of action on the receptors, on the possible medicinal applications of cannabis actions to the best of my knowledge, NIDA has funded a substantial part of that, where again, I can't say what fraction has come from NIDA and what fraction has come from drug company developments, but I know that recipients of NIDA funding have contributed to that work, so I can only assume that NIDA has not objected to it.

Q We only have a few minutes left for the day. I want to ask you though if you're familiar with a couple of studies, so that I can ask you some more about them perhaps tomorrow, but you made reference to the Tashkin study.

A Yes.

Q Are you familiar with the complete Tashkin study?

A I'm familiar with many of Tashkin's studies.

Q Have you had his most recent information up to I think it's 1996?

A Well, I have some. I don't know which you're referring to.

Q Okay. Well, I'm getting his actual most recent report, the most up to date report in terms of his longitudinal study overnight and maybe I can provide you with that one.

A Oh. I see what you mean. Of the continuing longitudinal study. No, I don't believe I've seen anything in 1996.

Q Okay. How about the study by a person called Linn to do with this question of birth weight and head circumference and so on in babies, L-i-n-n?

A I can't recall whether that was one of the ones that was—no, that wasn't one of the ones that was used for the—

Q I've got a few that are on this topic, so if you want to keep what you had in front of you just handy. The other name was Dreher, D-r-e-h-e-r.

A When and—what was the study and when was it published?

Q Again, in relation to birth weight and head circumference in babies. Let me give you all of them. There's Dreher, Linn, Tennes, T-e-n-n-e-s, and Day, D-a-y.

A Yes, Tennes—

Q You're familiar with?

A Yes.

Q Okay.

A And there—certainly there has been—there have been differences of findings in some of the studies on birth weights and head size and so on in relation to the prenatal exposure to cannabis, that is correct. There have been differences of finding.

Q You mentioned, Dr. is it Freed?

A Freed.

Q Freed.

A Yes.

Q A number of times and you said that—and correct me if I got this down wrong, but that the cannabis exposed babies were in worse shape than the tobacco exposed babies, did I understand that correctly?

A No, they were at—they were in worse shape than the non-smoking and when correction was made for tobacco consumption by the mothers, as well, they were still in worse—small undersized, compared to the controls, the non-using controls.

Q Because the findings, as I understood them in Freed, were certainly that the tobacco exposed infants—

A Yes, tobacco had—

Q -- were the ones in the worst shape?

A Tobacco had an influence, but when the tobacco contribution was—was taken out, that is corrected for by statistical means, the cannabis still contributed a significant reduction in body size.

Q But the tobacco exposed infants were still—came out as the worst influence, did they not?

A I would have to check back to the Freed studies. I think that may be correct, but I don't remember.

Q Okay. Do you have enough information here to check that overnight or—

A I didn't bring the Freed studies with me, unfortunately. I have them in Toronto. I would have to—I would have to check that when I get back.

Q All right. I'll see what I can find overnight and maybe I'll—

MR. CONROY: Would this be a good time, Your Honour?

THE COURT: I'm sorry, I misunderstood. I thought you mentioned that there were actually three groups that were tested, cannabis only, tobacco only and mothers who smoked neither of those, but now—

A No—

THE COURT: -- you're talking about statistical corrections.

A Yes. I don't believe I said that there were three—perhaps—

THE COURT: Oh, you may not have said it. That's just the way I understood it.

A There were—I mean, most of the cannabis smoking mothers also used—

THE COURT: Tobacco.

A -- used tobacco, and the—the estimation of the cannabis effect was by statistical correction for the contribution of the tobacco. This is a well-recognized procedure in examining drug effects when more than one substance is used. Where there were what I referred to was the Tashkin group, which did have separate groups—

THE COURT: Ah.

A -- of tobacco smokers, marihuana only smokers, tobacco plus marihuana and non-smokers.

MR. CONROY: I had a note that that was in Freed as well, but I think it was your question, Your Honour, that asked that about the three different categories. Anyway, sort that out overnight.

THE COURT: All right. We'll adjourn. You are under cross examination, which is—means that you're not to discuss your evidence with counsel or anyone else—

A Okay.

THE COURT: -- over the evening. Thank you and we'll see you tomorrow 9:30.

(WITNESS ASIDE)

(PROCEEDINGS ADJOURNED TO 1997 JANUARY 31 AT 9:30 A.M.)