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 28

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

R. WALLS Court recorder

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1997 JANUARY 28

(PROCEEDINGS RESUMED PURSUANT TO ADJOURNMENT)

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

JOHN PAUL MORGAN, recalled, testifies as follows:

THE COURT: You may have a seat, sir.

A Thank you.

THE COURT: You are still under oath. You understand

that?

A I do.

THE COURT: There once was a manuscript right up here.

THE CLERK: Was it an exhibit, Your Honour?

THE COURT: I think so. Did it have a number?

A VOICE: I think it’s that one that right there.

All right? That’s it.

THE CLERK: Exhibit 27, Your Honour.

EXAMINATION IN CHIEF BY MR. CONROY continuing:

Q We were up to chapter six, we were about to

start—

A Yes.

Q -- on that one. And that is headed "Marihuana’s Persistence in the Body".

A Correct.

Q And the myth that's set out for that chapter is, "Marihuana's active ingredient, T.H.C., is trapped in body fat. Because T.H.C. is released from fat cells slowly subtle psychoactive effects may last for days or weeks following use. T.H.C.'s long persistence in the body damages organs that are high in fat content, the brain in particular." And again you've set out a number of quotations that form the basis for that myth, and then the findings as a result of your research and review. Would you tell us about that.

A This is a very important myth. It's akin to some others that I've referred to as medi-toxicity, in that it covers a great variety of issues. Earlier on we talked about marihuana being much more potent so therefore it's generally more dangerous. Later on I think for sure we'll talk about marihuana being a gateway drug, and this affects a variety of things. Even if marihuana itself is not so dangerous it leads to other drugs.

So the persistence in body fat has been a

very, very consistent statement and of course like many myths, it has some basis in truth. The dilemma regarding our interpretation of it is that it doesn't seem to have any basis for toxicity. The fact that the drug persists so long in body fat is not associated with any evidence that that persistence causes harm.

Now let me try to approach it in a logical,

and I hope again, brief sequence.

Delta 9 T.H.C. is indeed highly lipid soluble,

a term used by many people not all of whom understand it. That is, most drugs that enter the brain are more lipid soluble than water soluble. Because if you put them in a beaker that's half toluate and half water, most of it will go into the toluate, very little will go into the water when you shake it up. That's a characteristic of basically all psychoactive drugs. It's a characteristic of Valium, it's a characteristic of methaqualone, it's a characteristic of phenobarbital. Those drugs are more soluble in lipid, in non-polar solvents than in water.

Now this means in general that such drugs are

more likely to enter the body's cells, because the envelope of cells in mammals behaves as if it were a lipid envelope. So for drugs to enter the cell the drug behaves as if it's going into solution in the outer membrane of the cell.

Okay, having said all of that, Delta 9 T.H.C.

has a half-life in human serum of five to six days. That means that once it starts its decline it takes five to six days for it to decline from one concentration to half of that concentration. If you begin measuring at one nanogram per ml. it will take five days for it to fall to a half nanogram per ml. This is important. In fact, the reason it's most

important is that's the reason why marihuana contributes almost all of the positives in urine testing programs in the workplace, because marihuana can be detected in the urine for up to a week or longer in a regular smoker. In fact, it can be detected for five to seven days in a one-time smoker, because the drug stays in the body fat for a lengthy period of time. It does not leave fat cells the way many other compounds do.

Now, the critical issue here is that at

that one nanogram per ml. level which persists for five days there is no evidence that it's having any effect. The analogy I like to use with my students is that we know that it takes three hundred milligrams of aspirin to relieve pain. Now if you take one milligram of aspirin, one-three hundredth, does that mean it relieves a little bit of pain or it will cause a little bit of stomach upset? Well, the answer is no. Drugs have to reach a critical concentration to bind to enough receptors to provoke an effect. And although the literature is a little unclear at what level marihuana's effects go away, it is quite clear that below—I'll take a conservative statement, below two nanograms per ml.—and it may be seven, it may be twenty—the drug has no effect. So, it persists in the fat for a lengthy period of time, comes out very slowly, is metabolized very slowly and appears in the urine, but it has no effect.

Now, I should be able to prove that,

that in the last twenty years many, many studies have looked to see if marihuana users have a persistent effect beyond the two to three hours that acute effects generally persist. People get high, they remain high for two to three to maybe four hours. You can detect some evidence. Either they say they feel high or you can detect some evidence in testing, that they're—that they remain under the influence of marihuana.

Now there have been a couple of celebrated

studies showing that a subtle—a subtle effect was picked up eight hours or ten hours, or even in the one airplane simulator study a claim that effect persisted for twenty-four hours. Now we've dealt with all of those and I think the evidence is they were incorrect. For instance, the airplane simulator study, a group of papers which have been commented on in this case and others. Dr. Yesavage, Y-e-s-a-v-a-g-e, claimed that he could detect subtle effects in an airplane simulator in men twenty-four hours after they had smoked. But then a number of us criticized the study for methodology, and when he tried to repeat it the way we suggested he do it, he couldn't find an effect. That publication achieved very little notoriety, that imposing some controls they could no longer find the twenty-four hour effect.

There’s a similar driving study by Dr. Moskowitz,

who’s referred to in previous testimony in this case, that he says he found a subtle effect for eight to ten hours. This is back in 1977, maybe 1981. No one has repeated that study.

And the weight of evidence, including some recent

studies are that marihuana has an effect in humans for two to four hours after smoking. The persistence of the drug in body fat, which is like the persistence of Valium and Thorazine, drugs which you can also detect for days after individuals stop using it, is not associated with effect in the sort of claim that we see here as in myth two or the myth statement two by a popular writer named Peggy Mann. "Cannabinoids accumulate in the fatty cells and the three-pound brain is one-third fat. Therefore, in the brain of the chronic pot smoker millions of axons are continually surrounded by T.H.C." Sounds pretty frightening. And then Robert Dupont, a former head of NIDA in the United States says, "Even people using marihuana only once each month are continually exposing their brains, lungs, liver and other vital tissues to the poisonous effects of T.H.C." Well—

Q When you were reading there you were reading

from—

A My—

Q Myth two or—

A -- myth six, "Marihuana’s persistence in the body".

Q Under myth six.

A And these are the quotes that we have used to head up. The brain is not one-third fat. The brain is about fifteen percent fat. Ms. Mann got that

one-third by leaving out an important ingredient of the brain, the most important ingredient of the brain, which is water. The brain is probably about—brain is about sixty-five percent water, maybe a little bit more. Which is important of course, because that’s one of the reasons that water soluble chemicals don’t get into the brain very well. So the brain is only about one-third fat.

Marihuana does not preferentially go to the brain.

Take a smoked dose of marihuana and calculate how much enters the brain, it’s about one percent, maybe even a little bit less. And it doesn’t accumulate in the brain. It leaves the brain fairly quickly. It accumulates in fat cells where there are no receptors, where the drug is not changed, where it sits there for a while and then comes out at these low concentrations.

Maybe I've made all my points. What do you think?

Q So how it connects to the receptors is of interest. If you have a receptor such as in the brain you get the psychoactive effect. If you don't have a receptor there's no effect. Is that fair?

A That's generally correct. And you get an effect where there are receptors if you are above a critical concentration. Receptors behave that way, that maybe you can bind to one or two percent or three percent, but until you achieve a significant occupation, thirty, forty, fifty percent of receptors, you get no effect. So a tiny bit of drug has no effect. We carry around in our body residues of chemicals that we've been exposed to, and some of them persist in the body for years. People my age from North America have residues of D.D.T. in our bodies. Young people don't have residues of D.D.T. in bodies. Even though we were quite concerned about that, there is no evidence that it has any effect. There is no evidence that the tiny amount of lead, antimony, cesium that we carry in our bodies has any effect. It's there, you can measure it, but it doesn't matter.

Q So to summarize then, the fact that these—the T.H.C. lingers in the fat cells, there's no indication of any harm or damage coming from that?

A Or any effect whatsoever. The fat cells contain no receptors. Fat cells don't metabolize the drug to excrete it, as others would. They sit in the fat cells for a long time. It does not matter—I should point out that in some testimony that I know came from Dr. Kalant, he talked about this long persistence and maybe the production of active metabolites. Now I myself have referred to active metabolites when the drug is taken by mouth, but the same thing pertains. The concentration of the active metabolite, the one that we know of, drops so low that it too has no effect after two to three hours. So the issue of active metabolites, although one of great interest, is still an issue of a brief-lived effect and then the persistence of the drug fragments for hours or days after the effect is gone.

Q All right. So if—in effect there's no harm caused to the brain or other bodily organs by the T.H.C. but it does remain there for a long period of time and that's why you can be drug-tested weeks perhaps later and it'll show—

A Yeah.

Q -- T.H.C. in your system.

A There would be no drug testing industry if it were not for marihuana use.

Q Okay. Chapter seven, "Marihuana and the Immune System". The myth set out there is, "Marihuana use impairs the immune system. Marihuana users are at increased risk of infection, including from H.I.V. AIDS patients are particularly vulnerable to marihuana's immune effects because their immune systems are already suppressed."

A It was—

Q There's the myth.

A It was a reasonable concern that since people with AIDS have begun to use marihuana and since the United States government has approved marinol[phonetic] for the purpose of increasing appetite in patients with AIDS, the question of marihuana's having an anti-immune effect came to prominence again.

It came to prominence initially in the early

1970's and I'll tell you how that occurred. But I'd like to start my comments here on the immune system by saying there has never been a single study of any sort in humans to show that marihuana has an anti-immune effect. There has never been a single study of any sort to show that marihuana has an anti-immune effect in humans. There are no studies, epidemiological, case survey, clinical, of any sort to show that marihuana users have an increased prevalence of infection of any kind.

Despite that fact, this myth has stayed

alive since 1971. And in fact I see very little evidence of its final dissipation and disappearance, which should occur.

In the early 1970's a prominent physician and

marihuana researcher named Gabriel Nauhaus talked about the anti-immune effect of—of marihuana. He's written a couple of books in which he revealed his thinking about this and those have been very useful to us, because he thought when he studied the immune effect that he would find out that marihuana smokers had hyperactive immune systems. He knew that T.H.C. persisted in the body for a long time so he thought we would be making antigens against T.H.C. and we would be shown to be hyper-immune, and that was why he began his studies.

He then found out in the study—in the first

study he did that there appeared to be some immune suppression in marihuana users and he published that in 1971. Actually he published it four times between 1971 and 1974 and it established forevermore the idea, put it on the research agenda.

If I may, I'll tell you what he did. He recruited

dental and medical students from Columbia College of Physicians and Surgeons in the upper west side in Manhattan and he asked them about their drug use. He divided them into marihuana users and non-marihuana users and he then took blood from them and harvested their white blood cells. Common research technique. And then you put the white blood cells in a culture medium, maintain them by giving them nutrients, and then you can study them in certain ways.

One of the most common ways to study white

blood cells, which are part of our immune response, is to expose them to certain chemicals which are known to provoke an immune response, chemicals called mitogens. There's a chemical derived from poke weed which works very well as a mitogen. There's another chemical called phytohemagglutinin which always provokes white cells, human white cells to spark up. Now when I say "spark up", what do I mean? They get bigger, they take up more chemicals from the surrounding media, their appearance actually changes and they secrete immune modulators. This process is called lymphocyte transformation.

Okay, so Nauhaus published a paper in which

he said lymphocyte transformation in chronic marihuana smokers is impaired, their lymphocytes don't take up as many chemicals from the surrounding medium and it is clear that in chronic marihuana smokers there is an anti-immune effect. Well, it in a certain sense was a revolutionary study because it's changed the way everyone thinks about the drug. The only problem was that he was completely, utterly, absolutely, irrevocably incorrect. No one in five to six to seven attempts has ever been able to repeat that study. No one. Everyone has tried. I mean everyone interested in immune transformation has tried. What they—what they cannot find is that the cells of marihuana smokers are any different than the cells of non-marihuana smokers in their ability to transform. And in fact in 1979 Dr. Nauhaus published very quietly a little note that he couldn't repeat the study, that he had tried to show it again in a group of chronic marihuana smokers whom he hospitalized and exposed to more marihuana and then did the transformation test again.

It's also now clear what was wrong. He did

not carefully assess the tobacco use in his medical and dental students and workers. And the suppression of immune response in those marihuana smokers was almost certainly because they were—most of them were heavy tobacco smokers as well. There's been some argument in the literature and he's denied the contention I just made, but there's plenty of evidence that that's what was wrong, because—guess what? Tobacco smokers have

impaired lymphocyte transformation. Always. I don't—it's not entirely clear what that means, but they do. Tobacco smoking impairs—tobacco smoking impairs lymphocyte transformation. Marihuana smoking does not.

Q And I see from page 3 apparently Valium, Librium, caffeine, aspirin and alcohol all affect the lymphocyte transformation—

A Right.

Q -- at least in—again in a laboratory with large doses that—

A Very—that's very critical and important. When Nauhaus did the lymphocyte transformation in people whom he said were marihuana smokers versus non-marihuana smokers and saw this impairment, that also provoked people then to take human lymphocytes, put them in a culture and expose them to large doses of T.H.C. and other chemicals.

Now one of the things that you can expose

them to is marihuana and tobacco smoke. And that very much impairs. In both instances the smoke will impair. Or condensates of smoke. Now if you expose normal human lymphocytes to high concentrations of T.H.C. you do see some change, some diminished transformation. And actually not always. In a couple of experiments people reported an increased transformation. But you do see with high concentrations—I'm talking about very high concentrations. I'm talking about ten to a thousand times the human psychoactive dose of T.H.C. placed in this dish, in this culture medium.

Now what you noted is what we've pointed out,

is that a variety of chemicals taken up by the lymphocyte will impair its transformation, including alcohol, Valium, aspirin, non-active cannabinoids such as cannabidiol or olivitol[phonetic], a

cannabinoid-like chemical that's used to synthesize T.H.C. So a variety of chemicals dumped into this medium will interfere with this lymphocyte transformation, and it's not clear what value the test has since so many things will interfere.

So for years we've continued to deal with

this problem, and in the 1983 Fehr and Kalant book from the W.H.O. Addiction Research Foundation text, which has been referred to here I know—

Q Was that '81 or '83?

A Well, the meeting was in '81, the publication was in 1983. Nauhaus's studies are discussed in there to a great degree, despite the fact that no one had ever been able to confirm the correctness of them. But they were discussed. They—they continue to be discussed.

Now let me quickly switch to the fact that

finding a change in a cell dish, in a petri dish, in a culture in a laboratory has some meaning, often helps us understand the mechanisms by which drugs work. But it has almost no meaning in terms of human response to drugs. Because human response is a mixture of dose, a mixture of effect in one cell affecting another cell which affects a cascade of cells, inhibition of inhibition is stimulation in some cells but—in the intact organism. So isolated cells in a culture dish, the impact tells you almost nothing about human toxicity. Almost nothing.

So, what do we have now? We have then—are

there ever studies ever showing increased infections in smokers of marihuana? The answer is no. There are none. There are none.

If you give animals very, very large doses of

T.H.C. there have been some reports of increased susceptibility to infection. Dr. Kabral, who works at the University of Virginia, has now published two papers, one in which he exposed rats and the other in which he exposed guinea pigs to very, very large doses of T.H.C. by injection. Not by smoking. And then he—in those animals he actually painted on herpes virus. He didn't just expose them, he actually applied it. They were all female animals and so he applied the herpes virus to the vaginas of the female rodents and guinea pigs. And he saw a slight increased take; that is more of the guinea pigs and more of the rats exposed to T.H.C. got the herpes infection.

Well, to do that in rats it took a thousand times

the human psychoactive dose. To do that in guinea pigs a little better, it only took forty times the human psychoactive dose applied many times over days.

So Kabral's studies are often cited. Here's

some animal evidence of an impaired immune response. But it too is unclear what it means in terms of humans.

So I guess I'll—I'll close by saying once

again, there is no evidence of any sort that marihuana causes impaired immune responses in humans.

No, I won't close. I'll say two minutes more.

When the AIDS epidemic started it turned out that

> the early demographic epidemiologic studies of men with H.I.V. positivity, those were—most of the people studied in the United States and Canada were men in the early days—it turned out that those men had an increased prevalence of drug use. They were prone to have taken inhalants, they were prone to have smoked marihuana, they were prone to have taken cocaine at a little bit higher rate than the people around them to whom they were compared. So there was some speculation, well the drugs may have contributed to their likelihood of getting AIDS.

Well turns out, what that almost certainly

> meant was an evidence of a kind of lifestyle in which individuals had more sexual activity and therefore more—were more likely to be exposed to the virus. Since that time there have been two very important major studies to look and see if marihuana use in an AIDS population had anything to do with an increased likelihood of contracting AIDS and an increased likelihood of conversion from H.I.V. positivity to full-blown AIDS, and the answer is no. Marihuana makes no contribution to worsening of H.I.V. in AIDS patients. So that's one of the reasons why the Food and Drug Administration in 1991 when a petition by the Unimed Corporation to add the label of H.I.V.-related wasting, the American Food and Drug Administration said yes and said not a word in the documentation about likelihood of immune dysfunction, even though the drug was to be given to people with serious immune dysfunction.

Marihuana does not cause immune dysfunction. It's

> a myth.

Q That appears to be the conclusion arrived at by the conference, the World Health Organization and Canada's Addiction Research Foundation, that '81 conference that—

A I think we even lifted a quote from them—

Q -- published the book in '83.

A -- didn't we?

Q The bottom of page 3. "There's no conclusive evidence that cannabis predisposes men to immune dysfunction."

A That came from the World Health Organization document edited by Fehr and Kalant.

Q And then you note that marinol was then approved, and as you may have mentioned is then used or been made available with the approval of the U.S. Food and Drug Administration.

Now you mention on page 4 again the

business of the smoke, the effect of the smoke. And you state there that, "While T.H.C. has no impact on immune function, the smoke is shown to alter what's called alveolar macrophages."

A Yeah.

Q Which as I understand it is something in the large airways of the lungs, is that right?

A Yeah. The macrophages are large mononuclear cells in the lung—actually they exist other places, but in the lung is where they're most important. They are phagocytic cells. That is they take up particles, they can actually engulf viruses and bacteria and particular matter. The macrophages are important in our immune function. They also secrete chemicals which modulate and increase immune response. They have many functions.

Dr. Tashkin, whom I mentioned yesterday,

and other scientists have looked at the alveolar macrophages in smokers, and there are some abnormalities of the alveolar macrophages in smokers, tobacco smokers and marihuana smokers. Now if you expose the macrophage to high concentrations of T.H.C.—there's a series of studies done by a man named Huber—you see no impact, even at quite high doses. So that the transformation or the disruption of the alveolar macrophage, if people worry may be an

anti-immune effect, occurs with smoke. It does not occur with T.H.C. It would occur probably with any smoke, but the only things that are smoked with much regularity in this culture are tobacco and marihuana.

Q And as I understand it though these abnormalities were found in long-term, heavy marihuana smokers, were less pronounced than in tobacco smokers and again it's a dose-dependency type of situation.

A We have made that point many, many times in the lung issue, that in addition to some pre-cancerous changes in lung cells, in addition to some changes in inflammation and now some changes in the macrophage which was referred to the first time, all of these seem to be a function of the dose of smoke. And occasional marihuana smokers do not have alveolar macrophage abnormalities, but heavy smokers, the group that Tashkin has followed for eight to ten years, do. And again, that—that may mean something, it's just not clear at the moment that it has any anti-immune effect.

It’s one of the issues raised, changes in the alveolar macrophage, but there’s no evidence of this being due to T.H.C. It may be due to smoke and it may contribute to pulmonary problems.

Q So we could summarize then just by going back to what you’ve stated as fact at the beginning of the chapter, no evidence marihuana users more susceptible to viral, bacterial, parasitic or fungal infections and no evidence that marihuana lowers users’ resistance to sexually-transmitted diseases.

A Right.

Q Fair enough?

A Fair enough.

Q And that really summarizes it, does it? Okay. Let’s move then to number eight.

A Number eight should be—

Q Which we’ve—

A -- very brief because we’ve talked about so

many—

Q -- talked quite a bit about this already, yes. Marihuana smoke’s impact on the lungs.

A Yeah.

Q The myth is marihuana is more damaging to the lungs than tobacco, marihuana smokers are at a high risk of developing lung cancer, bronchitis and emphysema. We’ve dealt with it to some extent, but if you just want to—

A I would like to mention a couple things, particularly the—the cancer potential and the argument around the—the level of carcinogens that are in the different smokes.

In the early seventies some people studied

marihuana smoke and published data claiming that marihuana smoke was dirtier than tobacco smoke. Dr. Kalant was among those who said there’s more tar, there’s more particulate matter.

Now it’s not clear to me why those studies were

probably incorrect, but I believe they were. Scientists make mistakes and come up with wrong answers often. Fortunately there is a corrective, and in the last ten years there have been many, many studies, Huber among others, indicating that—I think the truth of what I said yesterday, which is that marihuana smoke and tobacco smoke are essentially identical. There may be a few more—a little bit more particulate matter in very high T.H.C. marihuana smoke, there may be more carcinogens in tobacco smoke, but basically they’re about the same, with the exception of the two active chemicals, cannabinoids in marihuana smoke, nicotine in tobacco smoke. So that over the years it has I think become quite clear, particularly through Tashkin’s studies and some others, that the issue of lung damage is a matter of dose exposure to smoke. And I’ve made the point that heavy marihuana smokers have increased respiratory symptoms, they have more cough, more phlegm, more episodes of bronchitis. But that’s only in very heavy smokers. And most importantly, they do not develop the changes in airway function which we call chronic obstructive pulmonary disease, or emphysema. Something different about the two smoking experiences, and I think it almost certainly has to do with the exposure to smoke. It’s the dose that makes the poison. The same for smoke as for individual chemicals.

Now early on, in the early 1970’s there was

some evidence of pre-cancerous change in the bronchial cells of marihuana smokers. Those data were problematic in that almost all of the smokers from whom Dr. Tenant gathered those cells were also tobacco smokers. But—but Tashkin’s work again has helped us resolve the dilemma, and I believe the following to be true. That marihuana smokers have some pre-cancerous changes in the cells of the lining of the lung, in the bronchia-alveolar washings or lavage, as it’s often stated. So that people who smoke marihuana, particularly heavily, are at a risk of developing lung cancer. At this moment there are no convincing epidemiological linked case controlled studies showing that marihuana smokers develop lung cancer. And I’m actually becoming a little bit optimistic. I’m hopeful that marihuana smokers will not develop lung cancer, because again, they don’t inhale as much smoke. You know, tomorrow I could be proved wrong, but at the moment I—I have high hopes, I’m optimistic that they don’t inhale enough smoke to get lung cancer. But they do have the changes in their cells that tobacco smokers do that presage the development of cancer.

Now what I have to talk about now are

the level of carcinogens in marihuana smoke. Dr. Kalant and the A.R.F.W.H.O. document that I’ll refer to, both refer to a seventy percent greater concentration of benzopyrene in marihuana smoke than tobacco smoke. This has become one of the world’s—one of the mythmakers’ and claims makers’ favourite quotes. Our second quote here says, "Benzopyrene, a known cancer-causing chemical, produced in the burning process is seventy percent more abundant in marihuana smoke than in tobacco smoke."

Well, there were some early studies done

in the 1970's by a scientist who could analyze the chemical constituents of smoke. His name was Novotne[phonetic], and he published a paper which says there's higher levels of benzopyrene in marihuana smoke than in tobacco smoke. And that was—if I recall, that was probably thirty-six parts per billion versus twenty-nine parts per billion. I think that was his first publication.

Now some other people published similar

data, but in later years we've referred to at least further analyses which indicate that the benzopyrene levels in tobacco and marihuana are essentially the same, as one would expect, both combusted vegetable materials. They could be different, but they appear to be about the same.

Now this argument has taken on very big

significance because there's recent scientific evidence that benzopyrene is very important in the production of human lung cancer in tobacco smokers. It's complicated evidence, but it looks as if benzopyrene may cause mutational changes in lung cell d.n.a. and benzopyrene may be very important in some lung cancers. So again this has heated up the claim that benzopyrene is higher in marihuana smoke and therefore it's more dangerous.

First of all, it's not higher. It appears to be

about the same. And I also picked up what I cited in here, a very interesting study in Japan in which Japanese police gathered a lot of pipes, a lot of marihuana pipes and scraped out the residue and could find no benzopyrene. Now I don't know what that means, I don't know if that means that Japanese marihuana is different than western marihuana, but the issue of how much carcinogenic material is in marihuana smoke versus tobacco smoke is an unimportant statement because of the dose. The tobacco smoker inhales multiple times more carcinogens, more tar, more chemicals, more particulate matter, more hydrocarbon than the marihuana smoker, and that's the critical issue. It has to be the critical issue.

Once again in early studies, and Dr. Kalant refers

to these, the marihuana smoker deposits more tar per cigarette than the cannabis smoker, and that's because of this—I'm sorry. The marihuana smoker deposits more tar and particulate matter per cigarette than the tobacco smoker. And that's because of the traditional way that marihuana is smoked, the deep inhalation and the lengthy breath-holding. Those cause the deposition of more tar and particulate matter per cigarette.

There are two answers to that, two responses

to that. The first is it's still forty cigarettes a day versus one, in most people. And actually less than one in most people. And you can stop doing that. It's out quite clear that that manoeuvre, which has become traditional for reasons that no one understands, doesn't really increase the amount of T.H.C. very much. And so in a publication I recommended to marihuana smokers that

they stop doing that, as a harm-reduction manoeuvre. Smoke it like a tobacco cigarette if you're going to smoke it. There's no reason to hold it into your lungs and increase the deposition of material. It might be more dangerous.

But the bottom line is that chronic

high-dose marihuana smoking produces some of the same changes as tobacco, more inflammation, more irritation, more changes in the alveolar macrophages. And smoking of marihuana and tobacco together may be additive. However, marihuana smokers will not get emphysema. Tashkin's data indicate that very strongly. Marihuana smokers will not get emphysema. And that's critical. Marihuana smokers may get cancer. It hasn't happened yet and I'm optimistic that it won't.

Q Can we draw anything from countries like Morocco or Jamaica again, places like this, or even just bearing in mind the reported number of users of marihuana throughout the sixties and seventies, we know that in Canada anyway there's some forty thousand deaths a year from tobacco causes of one kind or another, and it's a major contributor in terms of cancer, heart disease, lung disease, these sorts of things.

A Right.

Q Can we say that that's—it's always this

dose-related factor? If we take these populations who are consuming large amounts of tobacco and some who are consuming large amounts of marihuana we don't seem to have the same consequences occurring as a result of the marihuana smokers. Is that—is that what you're saying in terms of the dose thing—

MR. DOHM: Your Honour—

MR. CONROY:

Q -- you attribute the dose—

MR. DOHM: Excuse me, Mr. Conroy. Your Honour, Mr. Conroy

is not under oath and I'm not going to be able to cross examine him on that statement. I would request that he ask the witness a question.

MR. CONROY:

Q All right. Are there large numbers of hospital recorded medical problems from marihuana use documented, bearing in mind the large numbers of users or reported users throughout history?

A No, it’s clear that there are not. And one can draw some inferences from the fact that there’s been heavy smoking of marihuana in North America since—since the early 1960’s. There’s been heavy smoking of marihuana in Jamaica for a long time and there is no evidence that there’s more pulmonary disease in marihuana smokers in Jamaica. But critics of—of what I said have a point, which is that it has taken us a long time to learn about the pulmonary hazards even of tobacco.

It’s important to put this in perspective,

that if you look at the overall range of tobacco smokers only one to two percent of them will get lung cancer. Now that’s an enormously high percentage, but of course it’s not the majority. Now very heavy smokers with a family history may get a higher prevalence, but overall in the smoking population it’s still a relatively rare event to get cancer. No one should take my remarks as approval of cigarette smoking. However, that means that if there were a

slight increase, even looking at the lower doses of marihuana smoke, it would be hard for us to find it.

But, having said all of that, having done

the proper scientific demurring, there is no evidence that marihuana smoking is associated with significant pulmonary disease in this culture. And one might wonder, as some people speculated in the sixties, it would be emerging now, we would see it now. And we have not. And I hope it’s because of the low dose of smoke, and I think it may well be so.

Q So the conclusion that you have arrived at after surveying the literature and so on is again as set out under the heading "Fact" on the face page of chapter eight.

A Yeah. I believe that moderate smoking of marihuana appears to pose minimal damage—danger to the lungs.

Q And then you set out the dose factor again.

A Poisons are always related to dose. I said it before and I don’t mind saying it again.

Q And when you say no documented cases of lung cancer—

A Yeah. This is—

Q -- you’re talking—

A This is a problem of course, because—I'm actually surprised that there have not been case reports of lung cancer in heavy marihuana smokers. There actually have been only one or two in the entire medical literature. And I would think that with the interest and surveillance that we would have found some lung cancer cases in smokers of marihuana who were occasional tobacco smokers—I mean I'm sure that people are looking. And we're not seeing any. And that's another reason why optimism is raised. However, there could be an increased prevalence of cancer in marihuana smokers and we have not detected it yet because we haven't done the right kinds of studies. Case reports and even clusters of case reports are helpful, but what you have to do is—and anytime there is a disease of low prevalence in a group of people you have to do a difficult kind of study which is a case control study in which you have to gather a large group of people who have lung cancer and you look back in their histories to find out the prevalence of certain behaviours they've engaged in, such as smoking marihuana, smoking tobacco, being urban dwellers versus rural dwellers, etcetera. Then you have to have a large group of people who resemble them in every way, age, gender, urbanity, family history, weight, diet, every way that you can measure, except that these people do not have lung cancer. Then you look back in their history and see what's the prevalence of marihuana use. Is the prevalence of marihuana use in the lung cancer group nine percent and the prevalence of marihuana use in the non-lung cancer group one percent? Then you've got some proof. However, you may find out that the prevalence is exactly the same and that marihuana is not a contributor to lung cancer. That's the kind of study that needs to be done when you have a toxicity of low prevalence.

Q We've talked about the smoke effect both from tobacco and marihuana and we talked a bit yesterday about the oral—taking it orally and the other effects that taking it orally have. Do you know—have there been any studies or do you know as a physician what the impact of using vaporizers, water pipes, these sorts of devices are? Does it make any difference or—

A We don't have much information. Sadly, water pipes don't help. It was believed for a long time that they did, by cooling the smoke that you might precipitate out some of the tars and hydrocarbons in the water and in the line, but it's not true. In fact what water—what pipe smoking does is decrease the amount of T.H.C. and deliver the same amount of hydrocarbons. It was a sad finding, only found last year.

So at the moment there have been a couple of

studies with aerosol nebulizers of T.H.C. looking—looked at for therapeutic things, and they showed that at least at that concentration in alcohol T.H.C. was very irritating.

And there is an attempt now in the United States

government laboratories to produce a heat vaporizer which will put the T.H.C. into pure—the T.H.C. into a vapour smoke that can be inhaled, and that might be useful.

There are other approaches that can be taken

to diminish the problem of smoking. At the moment those are not currently available. In fact, there's even a patented enema preparation under examination in the United States, for medical purposes.

Q When you said the United States laboratories, you're talking about government laboratories?

A Yeah, the attempt to vaporize is in a government laboratory. There may be—nowadays, now that—first of all, now that medical marihuana is legal in California, that might provoke lots of research to look for other ways to deliver T.H.C. I look forward to that. That's likely to happen.

Q The only other point in terms of the lung factor that I noticed, just so that we have this clear, is there's an indication in your chapter that says tobacco smokers, it affects the small airway abnormalities or you see small airway abnormalities, marihuana it's large airway abnormalities. Significance of that?

A Well, the significance is unclear except that one must have an impact on the small airways, the alveolar endings of the lung and those that exist in the periphery. For reasons that are not clear, tobacco smoke is—has a very deleterious effect on the periphery, marihuana smoke does not. And again, that may have to do with the volume of smoke once again. But yeah, that difference has been seen, that early on in Dr. Tashkin's studies he showed that marihuana had some impact on the large airways. In fact, as some—some of you may know, marihuana is a bronchia-dilator. People have hoped it might be useful in the treatment of asthma, even smoked. There isn't much evidence that that's so. So it does change the large airways, it makes them bigger, it gives them a larger diameter, a larger lumen. But tobacco smoke doesn't do that. Tobacco smoke harms the small airways and makes them dysfunctional.

Q There were some studies which suggested that—I think we have here before us or that are in the materials, that suggested its use as a

bronchia-dilator. Have those now been—

A Well, no—

Q -- discredited, or—

A -- it is a bronchia-dilator. The question is can that effect be exploited therapeutically. At the moment there's no evidence that it can. If you inhale the smoke you have then the mixed dilemma that you get bronchia-dilation but you also get irritation, which is not useful in asthmatic patients. It's entirely possible that T.H.C. delivered in another way, under the tongue, even swallowed despite the inefficiencies of swallowing, other ways, T.H.C. might end up therapeutic in asthma. But at the moment there's very little research going on.

Q All right. Let's move on then to chapter nine, "Marihuana, Sex Hormones and Reproduction". The myth set out here is, "Marihuana interferes with male and female sex hormones. In both men and women it can cause infertility. In adolescents marihuana retards sexual development and may produce feminine characteristics in males and masculine characteristics in females."

A Again, in the early 1970's there was the claim that marihuana decreased testosterone in male smokers and the speculation that that would both interfere with fertility in adult males and that interference with testosterone would decrease sexual development in adolescent males.

Then later on, almost as an extrapolation

of that, there was a claim that marihuana had adverse sexual hormone effects in females, that is by decreasing a particular brain hormone called luteinizing hormone it would interfere with female sexual maturation in adolescents and it would interfere with pregnancy.

In essence, every attempt to find out

whether marihuana has important effects on sex hormones, sexual maturation and fertility has led to the consistent answer it does not. That doesn't mean that people have stopped worrying and people have stopped looking and people have stopped making claims such as smoking a single a marihuana cigarette suppresses production of the female hormone essential

for the implantation of a fertilized egg in the uterus. If you give large doses of T.H.C. to monkeys and rodents you do see some immediate decrease in luteinizing hormones, some decrease in prolactin. Interestingly enough if you keep on dosing them with T.H.C. the effect goes away, they become tolerant to it. And that might be one of the things going on.

But in humans it's very difficult to show

that marihuana has any impact on any pituitary hormones or sex hormones from the—from the ovaries or the testes. In most studies that have been carefully done, and I particularly refer to one here by a scientist named Bloch, in a group of chronic marihuana users saw no change in hormones. And when people tried to follow up Dr. Kaladni's claim that testosterone was decreased, almost all laboratory studies showed either there was no decrease or if there was a decrease it was very evanescent, did not go below normal levels and had no obvious functional significance.

So there's much that one can say. There's an

enormous number of animal studies looking at the adminstration of T.H.C. either by smoke or by injection to rodents and to primates. There are all the speculations about changes in males and female adolescents, interfering with their sexual development, masculinizing females, feminizing males. And I guess actually I should mention that one of the reasons this area became so approached was because of some early reports, I think in the early 1970's, describing three cases of male breast enlargement in marihuana users. And this is commonly stated, smoking marihuana will cause males to develop breasts and will feminize them. It's not true. In fact, the military, the United States military did a large study and looked at individuals who developed gynechromastia, something that occurs in males usually for unknown reasons, they develop some breast tissue, and there was no evidence of marihuana making any contribution to gynechromastia in young men. But that's led us into this long, long dance about marihuana's impact on sex hormones. And the bottom line is there appears most likely to be no effect. If there is an effect it's evanescent, minor and of unknown functional significance. In fact, I recently told my students that I think the T.H.C. receptor in brain cells may down-modulate the secretion of some hormones very briefly. That may be one of its effects. So if we looked at cells in the pituitary and hypothalamus, the cannabinalant receptor in an endomyte may be one of the ways that that system is regulated. So it is possible that given large doses of T.H.C. one might see some slight changes in sex hormone secretion, although it's been very difficult to do that. It could be true. However, there is no evidence that it has any functional significance in sexual maturation, sexual behaviour, fertility in human users of marihuana.

Q Again, at least when—if it's a problem such as this gynechromastia, could we expect to—if there were any truth to that would we—could we expect to see results like that developing in these populations such as Jamaica again, and Africa or—or even the studies by Mr. Tashkin at U.C.L.A., long-term studies like that? Would—would we—

A Always a problem with the number of people who are being followed. If gynecomastia occurred only one in a thousand smokers you wouldn't obviously notice it. But the case control study of the sort done in the military which you look at people with gynecomastia and see if they've used marihuana, compared to a control group, is pretty good, convincing evidence.

Now fertility problems, you know, in

cultures where marihuana is used very, very extensively particularly by lower class, uneducated, illiterate people such as in India, such as in Egypt, to some degree in Jamaica—I want to be very careful because there are middle class users of marihuana in Jamaica, not so much in India and Egypt—there has been no evidence of diminished fertility. If anything, as you know, we are concerned about the high fertility among poor people in India and Egypt and the rest of the world, particularly in those cultures where cannabis is commonly used. There—there is no indirect evidence that marihuana interferes with fertility. Although again, the Partnership for a Drug-Free America ran a lot of ads in the United States showing a young couple sitting in their physician's office and he's saying, "I'm sorry, you'll not be able to have a baby," and then the text goes rolling under that it was their marihuana use when they were young people. This is a means of frightening people in trying to promote a war on marihuana. But not a shred of evidence. Not a shred.

Q So in this area we're talking about fertility, sex hormone levels—

A And sexual maturation of young people.

Q -- and sexual development.

A Yeah.

Q And in each case your investigation of it indicates there's no evidence to support—

A Yeah, and actually even all of the claims have no evidence that it matters in humans. The claims are only that they see slight changes in hormone levels. I think in most of the instances they're wrong, but even if they did, it doesn't seem to have functional long-term significance.

Q Robert Kaladni you mentioned.

A Yeah.

Q I think you were in court when Dr. Connolly was being examined. That's the name that—one of the names that was referred to.

A Dr. Kaladni became very well known in the United States as an investigator of sexual behaviour and hormone function. In fact, he was an associate of Masters and Johnson who became well known as writers and describers of—of sexual behaviour in humans. But he was a laboratory worker and could measure hormones. In fact, one of the first studies that he became well known for was measuring testosterone in male homosexuals. And he made the mistake of publishing in the late 1960's that male homosexuals had lower levels of testosterone. He was wrong about that. It's not true. So then he soon there followed with his claim that smoking marihuana lowered testosterone. And it really—well, it just provoked enormous fears and twenty-six years later we're still dealing with it.

He just had a group of marihuana smokers

and he compared their testosterone levels to

non-marihuana smokers and said it was lower. Then he put people in a laboratory and had them smoke lots of marihuana and although they at the end of the laboratory time had the same testosterone levels, he said there was a slight decline and then it came back to normal. So he's the genesis and put testosterone and other hormone measures on the research agenda by his publications in 1971 and '72, and they attracted enormous attention. Just they appear not to have been correct.

Q All right. Let's move on then to chapter ten, "Marihuana Use During Pregnancy". And again the myth set out in your manuscript is stated to be, "Marihuana use by pregnant women damages the fetus. Marihuana causes birth defects and later developmental problems. The health and well-being of the next generation is threatened by marihuana's use by pregnant women." Again, what are your findings there?

A Well, again there's a pattern of—of claims and reports here. Again, to some degree these reports are tied to what we just talked about, that if marihuana provokes changes in sex hormones that it might have an impact on the developing fetus. And I guess most of this research started with the claims of chromosomal abberations in marihuana users. The idea being—most of you will recall that people were very worried about L.S.D. producing chromosomal aberrations and maybe birth defects. And what happened in early such studies is that one can make preparations of human cells and tease out the number of chromosomes and the integrity of those chromosomes. And most everybody was able to find an impact of one drug or another on chromosomal integrity in the early studies of the sixties and seventies.

And those claims have not held up, that marihuana produces

- or L.S.D. in fact produces chromosomal abnormalities. So the early idea was that birth defects and harm to pregnancy would occur because of this impact of marihuana on the chromosomes, and particularly the chromosomes in female ova or male sperm cells. That's all gone by the wayside. There's nobody at the moment that I know of, except an occasional DARE officer who tells people that marihuana will harm their chromosomes.

However, the idea now is that somehow

exposing the fetus to marihuana smoke, particularly if the pregnant—if the woman carrying the fetus is a marihuana smoker, that somehow that exposure to the marihuana smoke or T.H.C. is causing fetal harm. This is a very complicated issue because there are at least three kinds of—three or four kinds of fetal harm that have been proposed.

Let me quickly go through—there were some

early studies—and again, Dr. Kalant in the 1983 document referred to the fact that there were some early studies indicating that children born to women who smoked marihuana had low birth weight, low head circumference, low gestational age, meant that they were small for the number of weeks they'd been in the uterus, and other minor abnormalities that might be attributable to marihuana. Well, after many, many years of study and after attempts to look at these issues very carefully it is our belief and we do cite one paper by Linn who studied twelve thousand newborns regarding their marihuana exposure, that there is no impact of marihuana use on the obvious physical integrity of the newborn or the fetus.

You know, to give the devil her due,

Nancy Day, who publishes many papers out of Pittsburgh, recently published a study in which she looked very carefully at marihuana use during the three trimesters in a large group of—of poor urban women. And she looked at all the things that are supposed to have been affected by marihuana and she found no impact on head circumference, no impact on birth weight, no impact on age of gestation, no impact on usual abnormalities and the Apgar score, the score of infant robustness when it's born, how well does it cry, how well does it move. However, she published that depending upon exposure in I believe the second trimester, that the marihuana exposed infants were shorter than the non-marihuana exposed infants. Shorter. I don't know what that means. It also turned out it was two-tenths of an inch shorter. Other people have not found that. However, it was published with great fanfare. It was not published with quite so much fanfare that women who use marihuana in the third trimester had heavier infants, which is generally thought to be a good thing.

So, that's the story of the epidemiological

surveys of children born to marihuana users. If you're not careful with your controls you can claim that there's low birth weight. But it turns out if you control for nutrition, the birth of that woman's—the weight of that woman's previous pregnancy, etcetera, etcetera, that there is no evidence of an impact on birth weight, head circumference, birth length, etcetera. In fact, there are a few studies in Jamaica which show improved statistics in marihuana smokers. The marihuana-smoking women seemed to be a little healthier and their children seemed to be a little healthier in terms of size, etcetera.

But occasionally there'll be a finding such

as Nancy Day's finding of two-tenths of an inch shorter in the infants, and then that will be published as evidence of marihuana harm. Well, those—those findings are inconsistent, they're not repeated from study to study, they do not seem to correlate with the amount of marihuana used so it's unclear how they can be a pharmacological or toxicological effect of marihuana. And they tend to be quite minor and of no functional significance. So that's why we've arrived at our conclusion that there is no evidence of physical harm to the developing infant, to the developing fetus because of mother's exposure to marihuana.

And I hasten to say, as is responsible and

important to say, pregnant women should not smoke marihuana. Of course they should not smoke marihuana. Just as they should not do many things. On the other hand, we don't live with them and control what they do. And in fact, even imposing laws on them does not control what they do. The prevalence of marihuana exposure during pregnancy in surveys in the United States is probably up to thirty percent, which becomes important in another discussion we're going to have.

So I don't think pregnant women should smoke

marihuana. I don't think they should live with people who blow marihuana smoke into their faces. On the other hand, there is no evidence that marihuana harms the developing fetus in terms of birth weight, head circumference, etcetera, etcetera, etcetera. Okay?

Q The—my recollection is is that we've had some reference to studies to do with the low birth weight and that it didn't last for long, that after a year or so or maybe less, I can't remember exactly, that the birth weight came back. This study by Nancy Day on the shorter babies, did—did that change?

A Don't know.

Q They stayed short forever?

A Don't know of her follow-up data so I can't say. I actually believe that there is no diminished birth weight

in—in women who are marihuana smokers. There's a possibility that if they are women who would give weight to low—birth to low weight children because of their nutrition, their history, their size, their exposure to other chemicals—incidentally, there is a fair amount of evidence that exposure to tobacco smoke and alcohol have impacts on the fetus. Again I'm not sure how important they are, but they have an impact greater than marihuana. So these things complicate. You know, women who smoke marihuana are very often tobacco smokers and it's difficult to tease those effects out.

But I don't believe there is any impact

of marihuana, even at fairly high doses of use, on the developing fetus. But I don't believe that women should smoke marihuana while they're pregnant and I counsel all who ask me, not to do so.

Q So once again the—the basic conclusion is set out under the heading "Fact" on the first page of that chapter.

A Yeah. Now what we deal with now are more subtle effects. If marihuana does not affect birth weight and size and obvious health at the time of parturition, is there some more subtle effect. And one of the things referred to in the testimony of—which you supplied that Dr. Kalant gave, was this claim that an unusual cancer, a

non-lymphoblastic lymphoma or non-lymphoblastic leukemia—hold on a second—non-lymphoblastic leukemia occurs at a higher rate in marihuana smokers. This—it's astonishing how much publicity this report gave—how much publicity this report provoked. Hundreds of newspapers in the United States as well as government spokesmen and anti-marihuana spokesmen and supporters of prohibition said, "Look, here is this unusual, rare cancer that occurs at a higher rate in marihuana-smoking women. This is of obvious critical importance. It may indicate the impact of marihuana smoking and/or T.H.C. on the developing infant's cells. It's obviously of critical importance." I actually have a publication about to appear in a journal called the Forensic Drug Abuse Advisor, in which I analyze the study, the Robison study which claimed to show the increased prevalence of acute non-lymphoblastic leukemia.

Such studies are done, again, in the

case control method that I described before in which you look at the illness, in this case the acute

non-lymphoblastic leukemia, a rare tumour in children, an important tumour although interestingly enough they're most often cured of nowadays. So you look at those children and then you look back into the mother's history and

see what she was exposed to. Was her—was she a worker in a factory where there were chemicals? Did she use marihuana or cocaine? What was her nutrition like? Did she live in a particular place? All the things, all the demographic and dietary and health things that might have had an impact. And then you take a group of women who again, as I said before, resemble the pregnant woman—and they were pregnant women. They were women who gave birth at about the same time. They resembled the—the experimental women, the women with—whose babies had leukemia in every way except they did not have leukemia. So then you add up, you know, were they too big? Were they too small? Were they all black? Were they all from a small indigenous people sect? Did they all smoke marihuana? Etcetera, etcetera. So you add those up and look for differences.

Well, Dr. Robison and his group discovered

that there was a difference in the prevalence of marihuana use. That is, he published a paper which said the risk of marihuana exposure was tenfold in women who had infants who developed acute

non-lymphoblastic leukemia. Tenfold. And what that means is that the prevalence of marihuana use in the mothers of leukemic children was five percent. The prevalence of marihuana use in the mothers who did not have children with leukemia was one-half of five percent, .5 percent.

Now—

THE COURT: Sorry?

A Hmm? One-tenth, five percent versus .05 percent.

Tenfold.

THE COURT: All right.

A Okay? Did I have that right?

MR. CONROY:

Q Just review that again.

A Okay.

Q Five—

A We—we—we measured the prevalence of marihuana use in the mothers who had leukemic children. Their prevalence of marihuana use was five percent. That meant five out of a hundred mothers had used marihuana during pregnancy. Five out of a hundred mothers whose children had leukemia. Okay. Now, we looked at our control group and found out that the

percentage of them who used marihuana was .5 percent. In fact, I'll tell you exactly what it was. It was one out of two hundred control mothers. So we then publish a paper that says marihuana has a tenfold risk of causing leukemia.

Now I bet everybody here knows where I'm

going. How did we find out the prevalence of marihuana use in those women? We called on the telephone and asked them. We called on the telephone and asked them.

Now, those mothers who had children with

leukemia had been affiliated with this hospital program. They then received a phone call and said, "We'd like to interview you about a variety of things during your pregnancy," and included in that list of questions would be, "Did you ever use illegal drugs during pregnancy, such as marihuana or cocaine?" Five percent of those women told what was almost certainly the truth, that they had used marihuana. Now those women of course had been actively involved in the care of their children. They, despite the guilt that might come down on them, would tell the truth to some degree, about their drug use. So we got a five percent prevalence. Five of them said, "Yes, I used marihuana."

Now we called a control group by telephone,

women we didn't know, women who'd never been to the hospital, said, "We're going to ask you some questions about your recent pregnancy. Did you use marihuana?" One out of two hundred women said yes. So immediately the question is raised, was this incorrect reporting.

Now, the next question, as a student of

pharmacology, is what is the prevalence of marihuana use during pregnancy? It's fifteen to thirty percent in most surveys in the United States. So both groups of women under-reported because of the embarrassment of telling somebody over the telephone, "I smoked marihuana and maybe harmed my child." But the mothers whose children were leukemic were much more honest than the women contacted randomly by phone. The study is useless. That's my most charitable comment. It is useless. It has been harmful because of all the publicity it's generated.

Q And that's the Robison study?

A That's the Robison study, the acute leukemia caused by marihuana.

Q I notice in this chapter that you refer to a Canadian study, Ottawa Pre-Natal Prospective Study. I wondered if we should just touch on that.

A Oh, we may have to do more than just touch on it. Since 1978 Dr. Peter Freed—I guess he's at Carlton—Carlton is in Ontario, is that right?

Q Right.

A Yes, I'm pretty sure Dr. Freed is at Carlton. Since 1978 he's been funded by the United States National Institute on Drug Abuse to follow the potential harm to the developing infant, developing on into infanthood, even late infanthood, elementary schoolhood, on into adolescence. He's been funded by the United States government. And what he did was in the—what do we—what do we call it? Ottawa—

Q Pre-Natal Prospective Study, page 5.

A The Ottawa Pre-Natal Prospective Study. What Dr. Freed did was to recruit a group of women who gave birth in and around Ottawa at various hospitals—I think various hospitals—and he gave a questionnaire and had careful interviews to find out the degree of use of alcohol, tobacco and marihuana in these mothers. Since 1978 Dr. Freed has published ten to twelve different papers about these children and it is our belief that taken as a whole Dr. Freed's studies show essentially no impact of marihuana.

Now I don't think Dr. Freed would agree with

me, so let me take a moment—if you look at the bottom of 10-5 you'll see what we think Dr. Freed's published by reading his papers. He found that age one—incidentally, at birth there was—he maybe had one test that said that the marihuana exposed children were different than non-marihuana exposed children, but it was inconsequential. He's not said very much about that. But then at age one he found that marihuana exposed infants scored higher on one set of cognitive tests, that is the marihuana exposed kids were smarter. But at age three the children of moderate marihuana users again had higher scores on a test of psychomotor ability. So in Dr. Freed's first two findings, at age one and at age three, marihuana exposed children were not harmed but actually seemed to be a little bit better, a little bit healthier.

Now at age four, the children of women who

were very heavy marihuana smokers, nineteen joints a week during pregnancy, scored lower on one sub-scale of one cognitive test. Imagine that, you know, you have a cognitive test that can be given to a four-year old. It may have—and I'm not a child psychologist, but it may have twenty scales. In fact some I.Q. tests have twenty-five, thirty scales. So they found one abnormal scale, one abnormal sub-scale score in the marihuana exposed infants. And by and large such a finding—you know, one out of twenty-five tests is abnormal on the basis of chance alone. Actually one out of twenty if

you use a .05 level of significance. So you find one test out of twenty-five, it's generally dismissable.

But okay, at age four he found this impact

apparently of marihuana use on these children. But at age five and six this difference was no longer present. He said maybe it's because they went to school and the effect went away. Maybe so.

Then measures of attentional behaviour added

at age six years old and a lower score on one

computer-based test of vigilance among the children of heavy marihuana users. Then eleven new psychological and cognitive tests given to six to nine year olds revealed no statistically significant difference between the children of marihuana users and non-users. So we're now to these group of children who are approximately nine years old, we've had these occasionally abnormal tests as they've developed, but now at age nine we find absolutely nothing.

Also, at the time of age six to nine mothers

were asked about behaviour. And the mothers of the marihuana using kids said that their children were a little bit more often behavioural problems than the non-marihuana exposed children, but once—there were a number of compounding variables having to do with educational levels of the mother and family income, etcetera, etcetera, this difference in behaviour disappeared.

Now—so Dr. Freed has now spent you know

a hundred million dollars of American taxpayers' money following these children—and I'm envious because he got all that money to do research, quite envious—and basically though what he's given us, up to age nine was no real difference in these children due to marihuana exposure.

However, here's a quote we took from Dr. Freed's

recent paper. "Instruments that provide a general description of cognitive abilities may not be capable of identifying nuances in neuro-behaviour that may discriminate between the marihuana exposed and

non-marihuana exposed children. Tests that examine specific characteristics that may underlie cognitive performance may be more appropriate and more successful."

Now in research grant language that's, "Please

give me more money because I have some other tests that I'm going to conduct."

I'm not—I'm being a little flippant here,

and I hope you'll forgive me if it's too much so. But I'm not being insulting to Dr. Freed. This is what researchers do. "I've got some new ideas, I've got some new tests, maybe I can try those."

Now, there's a problem in cognitive behavioural

tests. If five years after you started the testing you come up with a new test and a new index and a new batch of post-designed studies, there are some research psychologists who will say, "No, no, no, you can't do that. You can't change the playing field five years after you started examining the children, particularly if you haven't found anything heretofore, because then it looks to us like maybe you're trying to find a way to find something." That's what he did. He's now generated something called executive function. Now this is a new series of tests—it's been proposed by other psychologists that it's a way to look at development in children, helping them—helping you see their development of flexibility, their ability to assess input that's going on, their ability to make decisions. I'm being vague about this because I—I don't understand it in any more than a vague way. But it's a test of executive function.

Now I saw the handwriting on the wall in

1995 at a Washington conference with his funders, Dr. Freed stood up and said, "I think there's a possibility that tests of executive function may identify differences in the marihuana exposed children." Although he's down now to fewer than thirty marihuana exposed children because they've dropped out, mothers have moved away, he can't follow them so regularly. But I knew what was about to happen, that sure enough he's found some differences in tests of executive function. And this has now been jumped on by American media saying marihuana damages the cognitive ability of the fetus twelve years later.

This is very interesting because of course

drug exposure, if it has an impact, seems to have an impact early. And with passage of time drug-caused impact disappeared. I learned that from Peter Freed who has shown quite clearly that the adverse tobacco effects, which are pretty high, at least easily measurable, have dissipated over the years. But however, we now have a new test out, executive function in marihuana exposed infants, which supposedly shows some harm.

I think Dr. Freed is seriously wrong here. And

I think he has manipulated the research process to such a degree that I find him untrustworthy now. I've not said that about anybody else we've talked about, I don't think, in terms of the production of studies regarding marihuana effect. I don't think Dr. Freed is to be trusted any longer. Excuse me, the fact that he's a Canadian had nothing to do with my comments.

Q We say things like that about Americans all the time.

A I do. Actually mostly I have, haven't I?

THE COURT: So why have lunch for us? Can we—

MR. CONROY: I'm going to move on to eleven, so this would be—

THE COURT: All right. We'll take the morning break.

Fifteen minutes, please.

(WITNESS ASIDE)

(PROCEEDINGS ADJOURNED)

(PROCEEDINGS RECONVENED)

JOHN PAUL MORGAN, recalled, testifies as follows:

EXAMINATION IN CHIEF BY MR. CONROY continuing:

Q Just before we go on to the next chapter, there was one study that you mentioned to me that we haven't touched on and that relates to the previous chapter, and it's a study by a person called Bloom.

A Yes. The reason I wanted to mention that was because it too was referred to by Dr. Kalant in the testimony heretofore provided to me.

Dr. Bloom published a paper regarding

pulmonary symptoms and pulmonary function in a group of people around Tuscon, Arizona who were surveyed as part of a measure of pulmonary health and the impact of smoking. And Bloom had a question on his survey about non-tobacco cigarettes. Now I'm in agreement with Bloom that those cigarettes were almost certainly marihuana cigarettes, and there was some argument about why he chose to ask the question "non-tobacco cigarettes". That's not important. In fact, in my chapter I refer to a paper by Sherrill et al—that's my reference number eleven—and that's Sherrill, Bloom and other authors. So there are two publications then of this survey of pulmonary health of volunteers around Tuscon, Arizona. And basically what the Department of Medicine and Pulmonary Function did was to interview large numbers of people at intervals over time and to ask them about questions and to measure pulmonary function. Now Sherrill—then the two papers, one by Bloom and one by Sherrill, they said that the non-tobacco cigarette smokers had some evidence, at least on one test, of airway malfunction of the sort that I've said did not occur in any of Tashkin's prospective patients.

So Sherrill and Bloom are often cited as

showing that the smoking of non-tobacco cigarettes, almost certainly marihuana cigarettes, does cause some pulmonary damage. Now we have discussed the paper briefly, not solely to dismiss it but to point out that there is a very, very small number of non-tobacco cigarette smokers. It is unclear from Sherrill's publication how much tobacco they smoked.

It also has a very curious finding, which

is that the diminished pulmonary function in one test of four given was abnormal only in people with a history of non-tobacco cigarette smoking and not current non-tobacco cigarette smokers. So it's a little unclear how an abnormality of pulmonary function could occur more in past marihuana smokers rather than in current marihuana smokers.

We think these things all together make the

finding not reliable, and Dr. Tashkin agrees with us. Recently had a long discussion with him about the Bloom and Sherrill papers.

I did want to mention it because it was referred

to by Dr. Kalant in that testimony you provided me.

Q The reference you said was footnote eleven, and that's chapter eight.

A Correct.

Q On the lung effects.

A On the lung, right.

Q And Sherrill is S-h-e-r-r-i-l-l, I believe.

A That's correct. That's the survey of lung health in a group of people recruited in and around Tuscon, Arizona.

Q Let's move then to chapter eleven, the "Marihuana and Brain Damage". The myth set out on the face page for that chapter is, "Marihuana kills brain cells. Used over time marihuana permanently alters brain structure and function resulting in memory loss, cognitive impairment, personality deterioration and reduced productivity." What can you tell us about that?

A I can. The idea that marihuana kills brain cells or harms brain cells irreparably has been part of the research agenda and the debate again since the early 1970's. Just as we've been able to identify a particular person associated with immune claims and testosterone claims and other claims, the physician involved with

these claims is a man named Heath in the Department of Psychiatry at Tulane Medical School.

Now before Heath's study there was the

report by Dr. Campbell which I know has already been discussed at length, but I'll say a little bit about it again leading into Dr. Heath. Dr. Campbell was a British physician who in the early 1970's—a British radiologist who in the early 1970's using a technique called pneumoencephalography—and Dr. Connolly mentioned that yesterday—Campbell said that chronic marihuana smokers had shrinkage of their brain tissue. The study set off two kinds of responses. One

is the immediate acceptance of it by those who fear marihuana and who oppose marihuana reform revision, and also it provoked a number of letters and responses from those who said this is a dreadful study and is not to be believed. And that was its fate. Dr. Campbell's findings of brain shrinkage using an imprecise technique called pneumoencephalography have not been confirmed and modern techniques of computerized axial tomography or CAT scanning have not shown any evidence of gross brain function harm in chronic marihuana users. And Campbell's group were people who had been referred to them because of neuropsychiatric illness in a number of spheres and they were all chronic users of a variety of drugs, and he chose to attribute this finding to marihuana.

Now—so Campbell having been set aside and

dismissed by most people—although it's occasionally discussed—comes Dr. Heath. Now Dr. Heath had reviewed the literature showing by and large one could not find changes in the electroencephalogram of marihuana smokers, either acutely or chronically. The electroencephalogram is the technique in which electrodes are applied to the scalp and these monitor brain electrical activity and produce a wave form printout. Most people are familiar with the electroencephalogram. And a number of people had looked at the electroencephalogram, the surface electroencephalogram in marihuana smokers both acutely and chronic users, and could find no changes at all.

Now Dr. Heath conducted a study in which

he first in a single human patient—and I'll not comment very much about that individual—but then in a group of monkeys he planted deep electrodes, deep in the brain and exposed those animals to—these were mostly Rhesus

monkeys. And he exposed them to marihuana smoke and he said he saw important changes in some important areas of the brain where marihuana might work. It showed changes in the septal area, the area which is the division of the brain from right and left. And then in a particular area of the cortex called the hypocampus[phonetic]. The hypocampus is frequently referred to in fact because of Heath's work. So Heath's first publication was, "Yes, I can see brainwave abnormalities." And then Heath, before he had any data, said, "I think with repeated exposure these brainwave abnormalities will become permanent." He said that before he had any data to support it. But then he published some data saying that in the early exposure these changes in the brainwaves, which had reverted to normal an hour after exposure, persisted even during times the animals were no longer being exposed to T.H.C. or marihuana smoke.

Now let me hesitate, digress here for a quick

second or two. That it's impossible to make a monkey smoke cigarettes. Monkeys will not smoke cigarettes. They will not smoke tobacco. They'll not smoke T.H.C. They actually will inject tobacco intravenously if you—I'm sorry, nicotine, if you provide them with a pack and an apparatus in which they can get a reward by pushing a lever. They'll do that for nicotine, but they won't do it for T.H.C.

So to expose monkeys to marihuana smoke you

have to engage in some difficult, problematic techniques. And what Dr. Heath did was to actually blow the smoke through a tube which was inserted down into the—into the monkey's airway. Well, he admitted he had a lot of trouble doing that. Monkeys didn't like it very much, fought against the tube. It was unclear how much smoke they got, how much smoke they managed to get rid of. So his dosage levels were never very, very well understood. But still, along with the animals given T.H.C. intravenously he said the animals exposed to smoke one, had these abnormal brainwave forms which persisted. And then, although he ended up with very few monkeys, he sacrificed those monkeys, he killed the monkeys and looked at their brain tissue. He actually ended up with only one or two monkeys who had been chronically exposed to marihuana smoke. But he published in the early 1970's the claim that exposure to marihuana smoke produced irreversible brain damage in this particular area of the cortex, the hypocampus, in Rhesus monkeys. And this claim persisted for twenty years. And every time you turned around someone was saying, "Well yes, but marihuana kills brain cells, at least in monkeys."

Now as I've already mentioned to you, the

Heath study had many, many problems. It's unclear how much smoke they got in. The monkeys fought against the smoke. It's unclear how much oxygen deprivation they had because of the administration of large amounts of smoke through a catheter. But the studies were talked about over and over again as evidence of brain damage.

Now there exist in the United States now a

National Centre for Toxicological Studies at Jefferson City, Arkansas. It's a large federally funded unit which looks at toxicity, which looks at harm in humans, animals and other preparations by exposure to chemicals. The researchers at the National Centre for Toxicology conducted in the early 1990's a series of studies which showed that Dr. Heath was completely wrong. They repudiated everything that Dr. Heath and others had been saying for twenty years. What they did was to take a group—four groups of monkeys, sixteen each, and the monkeys this time were exposed by face mask. That is, a face mask was placed on them and the marihuana smoke was delivered to the face mask and they had to breathe it in but it wasn't being injected down to their throat. So it's a little closer to the smoking experience. And the amount of oxygen they were given was controlled. They were given adequate oxygen, just that marihuana smoke was puffed in. So you had one group of monkeys who were exposed to the human equivalent of five joints a day and exposed—they exposed them for a year to five joints per day. You had a second group of monkeys who were weekend smokers, they were exposed to the smoke of four to five joints a day only on Saturdays and Sundays and then the rest of the week they—they didn't get smoke. Then he had a group of animals who were exposed to smoke without T.H.C. in it. And then they had a group of monkeys on whom the masks were placed but no smoke was blown in whatsoever. So these animals were followed for this year period of time in which they were delivered either large doses of marihuana or small doses of marihuana or smoke or nothing.

And a large number of studies were done,

neuroendocrine studies, hormone level studies, adaptational studies, learning studies, etcetera, etcetera. Then the smoke experiment was stopped and the monkeys for seven months were allowed to go about monkey life. And their behaviour was measured during those seven months off the drug. And then all of the animals were killed and their brains were examined.

And in a series of probably twelve different

papers Drs. Slicker and Paul and Ali and Scallett, all from Arkansas, published the fact that there was no cellular changes in the marihuana smoking monkeys. None. No changes in the cellular architecture in the hypocampus, no changes in the neurotransmitter concentration, no changes in the synaptic space. No changes whatsoever after a year of smoking.

And I don't know if we took a—we didn't

take a quote from the Arkansas people, although we have in the past which basically says year-long exposure to marihuana smoke produced some acute changes, we found some increased levels in blood cortisone, they found some changes in pulmonary macrophages, they found that during the height of marihuana exposure the monkeys wouldn't work as hard for food as they had before. You know, when they were stoned they wouldn't work so hard for food as they had before. But then at the end of a seven-month non-exposure time there were no abnormalities at all. These were adolescent monkeys, interestingly enough. That was done on purpose because of the fears that adolescent exposure in humans is—is the issue we're most concerned about.

The bottom line is four to five joints a day

for a year in a Rhesus monkey produces no permanent effects of any measurable sort.

Again, it's a very important study. It's received

some attention, although not as much attention as you would like. It's—it's among the category of studies funded by the federal government in which the federal government doesn't talk about the study very much in the United States.

Q I was going to ask you that.

A Yes.

Q Was this—this Arkansas—the National Centre for Toxicology is a federally—

A That's correct.

Q -- government—federal government-funded study.

A And for instance, none of them were invited to the marihuana conference in Washington that I talked about a couple times that caused us to work on this text in 1995. None of the people from Arkansas were there. They continue to be funded. They're hanging on, doing okay.

Q And the Partnership for a Drug-Free America, is that a government—

A No.

Q That's private sector.

A Partnership is completely a private organization in the United States which basically takes money donated to it and advertising time, television, radio, movies, print, and then it goes to advertising agencies and says, "Make us an

anti-drug statement and we'll place it for you." So the largest advertising agencies in New York and elsewhere have made anti-drug ads for the Partnership and then the Partnership places it in the New York Times or the Wall Street Journal or N.B.C. local and national affiliates. Partnership is—well, the Partnership places a million dollars of ad space per day in the United States.

Q When a group is funded like the Arkansas group is it funded through NIDA, National Institute on Drug Abuse?

A Most often, although not necessarily so. The N.I.H., the National Institutes of Health, has other funding mechanisms. But most studies involving drug abuse are funded through NIDA and certainly some of the money that went to Arkansas came through NIDA. But I think some of it came through other branches of the federal government. And of course people doing drug research may get private funding as well, although nobody can do big time drug research without some federal monies because it's so costly.

Q But does NIDA continue to put out the results of Heath's studies or Campbell's studies and ignore the Arkansas study, or do they acknowledge the Arkansas study when they're putting information out to the public?

A They—I cannot tell you a single instance in which—no, I take that back. There was one instance in which NIDA said something about the Arkansas study. Remember I mentioned to you that animals exposed to marihuana at the—during the time of active exposure, who had been trained to push a lever for food would not work so hard for food. NIDA mentioned that in one of their publications, that marihuana caused decreased motivation in monkeys to work for food. But that's the only mention I've ever known of NIDA to make of the Arkansas study. And NIDA has never made any mention of the Robe driving studies, which we're certainly going to come to and discuss, even though that was funded by the federal government.

Q All right. At the end of chapter eleven you indicate that a number of the other effects on memory and cognition and so on we deal with specifically under the following chapters, so let's—and that's under twelve, thirteen and fourteen. So let's move on to those.

THE COURT: Can I just ask one question in relation

to the concept of a drug, any drug killing brain cells or altering brain cell structures or functions permanently.

A Yes, ma'am.

THE COURT: Do we know or have there been studies

in relation to alcohol consumption and the effect on brain cells?

A Yes. Alcohol in high doses, without question, alters brain cells, harms them, and if the dose is high enough it harms them irrevocably. These studies have been done in a similar fashion to this by giving alcohol to animals, primates and rodents. And also of course there have been a significant number of post-mortem studies in humans in which it appears that the brain damage is secondary to large doses of alcohol. Those studies are somewhat problematical because people who take large doses of alcohol also have poor nutrition and also get head injuries. But the general consensus of belief is that alcohol specifically harms brain cells in all species, depending upon the dose.

There have been no post-mortem studies

in human marihuana smokers to indicate brain damage of any sort. Doesn't mean of course it's absolutely impossible that it occurs, but there have been many studies in alcohol consumption indicating that brain damage actually occurs.

THE COURT: What about tobacco?

A None of which I'm aware. There have been many

studies of course, showing tobacco damage to pulmonary and other tissues. There have—I mentioned to you before that Dr. Freed's study shows development problems in tobacco-exposed infants but I'm not aware of any studies showing that tobacco smoke harms brain cells. I don't know of any.

MR. CONROY:

Q Coming back for a moment to the alcohol one, you mentioned high doses. We know or we've been operating on the assumptions that the marihuana smoker smokes far less than the tobacco smoker even though they inhale it more deeply.

A Right.

Q Tobacco smoker smoking forty cigarettes a day or

- or whatever is a common thing. But—so when you talk about high doses of alcohol—

A Yeah.

Q -- what are we talking about?

A Alcohol is consumed—well, let me answer in two parts, if I may. One of the bad breaks the human culture got was that the psychoactive drug which we could easily discover from the fermentation of any carbohydrate source was alcohol. The reason I say it's a bad break is that alcohol is particularly dangerous because it's so

weak. The statement surprises people somewhat, but if I wished to get high on marihuana or L.S.D. it takes a few micrograms. If I want to get high on cocaine it takes a few milligrams. Amphetamine takes a few milligrams. If I want to get high on alcohol it takes a few glasses full. Alcohol is a very weak psychoactive drug, and we therefore have to consume large amounts of it to get the desired brain effect. And in that consumption of large amounts of alcohol we expose the cells of our body to lots of alcohol.

Now I think it's very important to note

that two drinks of alcohol a day in humans is associated with positive health outcomes. Although there is some argument about that, by and large it appears to be true.

Q This is the wine and the beer—

A Yeah, well two drinks of any sort. I mean two drinks of bourbon apparently are the same thing. That is, there's salutary effects on blood lipids and there is now some evidence to indicate that there is increased longevity with two drinks of alcohol a day. But if you go much beyond that you begin to see some deleterious effect, you begin to see some change in the liver which is quite sensitive, you begin to see some change in the stomach lining, you begin to see some change in the peripheral nerve function and at higher doses you begin to see some changes in the brain.

So the dilemma for us is that our

preferred consciousness-alteration agent is weak and requires large doses and is therefore particularly dangerous.

Q So but when we—when you use the reference in the studies to high doses, does that have any specific meaning in terms of amounts of alcohol consumed in the studies?

A Well, if people begin to consume more than four to five to six drinks per day for a month, you can begin to measure some deleterious outcome. It's very mild at that level, but you can do it. And my guess is that five to six drinks a day for years is deleterious to human health.

Q We're talking a four to six glass range?

A Either way you talk about it. A drink is sixteen ounces of beer, twelve ounces of wine, an ounce and a half of distilled spirits. Those are drinks. They each contain about the same amount of absolute alcohol.

Q Okay. Let's go on then to chapter twelve, "Marihuana and Intellectual Functioning". The myth set out there is, "Marihuana impairs memory and cognition. Under the influence of marihuana people are unable to think rationally and intelligently. Chronic marihuana use causes permanent mental impairment."

A In the previous chapter and in our previous discussion I've talked about the idea that marihuana would harm brain cells. Out of those ideas, although not necessarily related to them, there came to be a set of claims regarding marihuana's function—marihuana's impact on intellectual cognitive function. Later on we spend a chapter talking about the claim that marihuana reduces motivation in human users. And then finally the third part of this set is the claim that marihuana produces mental illness in users or at least may make mental illness worse.

The—the intellectual function one

is a particularly interesting one because of course no one doubts that marihuana, like all psychoactive agents, produces acute changes, immediate changes on intellectual function. At high enough doses marihuana stops almost all intellectual function because it puts you to sleep, just the same as high enough doses of alcohol and phenobarbital would. You can't measure much intellectual function when people are asleep.

However, from the earliest days of the

seventies and actually the first important studies in the late 1960's, people have given marihuana acutely, that is immediately to individuals and then assessed their intellectual function in a number of spheres, sometimes their psychomotor function, their ability to perform tasks. But the biggest set of studies were those that looked at acute dosage producing acute alteration in thoughts, ideas and perceptions. Now we can't really measure thoughts, ideas and perceptions but we can give tests to see what marihuana or other drugs does acutely.

And I'm sorry for all that lead-in, but that leads

me to the fact that the test which is almost always found abnormal is a test of acute recall. So marihuana, in the language that everyone knows, has an important impact on short-term memory.

At the doses that it has an impact on short-term

memory, causes a high, causes the heart to beat fast, causes the eyes to get red, it doesn't do much else. I think that's important. At fifty to a hundred micrograms per kilogram, the low dose of marihuana that's effective, you see red eyes, fast heart rate, "I'm high", people tell you, "I'm high," and you see this change, quite predictable change in memory.

I'd like—like to describe that change in memory

for you because it’s important. Under the influence of marihuana individuals can recall what they’ve learned before. You know, if an hour before you got them high you asked them to learn a series of words or you asked them to learn a series of concepts or you said, "I want you to tell me an hour from now when you’re high the story of Uncle Remus or something that Stephen Leacock has written," they can do it quite well. However, if they’re high and you give them something to learn they have a fairly specific defect. And again I’ll tell you what it is, I’ll tell you what it is.

If you give them a list of words when they’re

high, let’s say you give them paired list of words, you give them words that are related, black-white,

coat-sweater, hair-beard, give that to them while they’re high and then give them the cues later on, they do quite well. You say "beard", they’ll say "hair". "Coat", "sweater". They’ll do that. However, if you show them a list of words and then take the list away and later ask them to recall those words, a process that the psychologists call free recall, people can’t do it when they’re high on marihuana. Their free recall is impaired.

Now it’s interesting that their free recall

is not impaired only because they can’t remember exactly the list of words, but they keep remembering things that they weren’t taught. The marihuana high, as everyone who’s been high a few times knows, is associated with the intrusion of memories from someplace else. The intrusion of words that were not part of what I’m supposed to recall at this moment, the intrusion of ideas that are not what I’m discussing with my friend at this moment. That’s what marihuana does. It loosens associations, it promotes what people, if they’re not being critical, refer to as lateral thinking. It causes intrusions of things which the—which the user then thinks, "Well, that’s on the list I’m supposed to recall. You gave me a list—" You actually gave him a list that said "beard" and then he recalls Morgan the pirate. You can actually find those keys, those things that have provoked a memory that intrudes on him.

But the bottom line is that he has

impaired recall of a variety of things, words, concepts, pictures, sounds. They’re all affected.

Now most of the other things we’ve looked at,

such as simple reflex time, questions of calculation, questions of perception in which he doesn’t have to recall things, he’ll do pretty well. In fact, there’s no regular defect that you can identify consistently much beyond the memory recall. There are some other things, some changes in perception, some changes in ability to calculate, but particularly those tend to be related to memory, the ability to recall. So by and large that’s the most important impact on marihuana.

Now what individuals note, of course, is

that they'll be having a conversation under marihuana and then they'll—suddenly the conversation will cease because nobody can remember what exactly was being talked about. And everybody who's had a marihuana experience has had that experience.

Q Is this—

A Can't keep the threads of the conversation going. And then people tend to laugh when they can't remember the conversation and that's one of the reasons why marihuana social events are accompanied by laughter, because people are using it to cover up the fact they can't remember what was being talked about.

Q And what we're talking about is during what you call the acute—

A Yeah.

Q -- phase.

A It's very important to state that this is a

short-lived effect. It occurs two to three hours of smoking, occurs during the high, which is almost always over by four hours. And I've actually seen a number of studies that show that it's pretty well over by two hours. So marihuana has an impact of distractibility. People are turned away from what they're supposed to recall and intrusions occur.

Now, immediately the question is is this a

chronic effect. Does it persist when individuals don't use marihuana? And we spent twenty-five years trying to find out if there is a problem of memory in

long-term users. And that's a long story. Shall I begin it?

Q Probably not, because it's just five after twelve.

A I'm sorry about long stories, but—

MR. CONROY: I don't know what the Court would like to do.

Should we break now and then carry on—

THE COURT: We may as well break now before we—

MR. CONROY: All right.

THE COURT: -- commence this story. For lunch, back

at one thirty.

THE WITNESS: Thank you.

MR. CONROY: Thank you.

(WITNESS ASIDE)

(PROCEEDINGS ADJOURNED)

(PROCEEDINGS RECONVENED)

JOHN PAUL MORGAN, recalled, testifies as follows:

EXAMINATION IN CHIEF BY MR. CONROY continuing:

Q We apparently overlooked a study that we should have referred to when we were dealing in chapter eleven, so I want to just go back to that very quickly, chapter eleven being the marihuana and brain damage. The research done by the Australian researcher Nadia Solowij, I think is the way

you—

A Solowij, that's correct.

Q -- pronounce it. S-o-l-o-w-i-j. Could you just comment on that.

A Yeah. Dr. Solowij's work and opinions has come before scientific notice in two ways. One is that she was the co-author of a lengthy review of the adverse consequences of marihuana published in Australia by Hall, Limmon and Solowij, or Hall, Solowij and Limmon, and it's frequently referred to. It has been in this case and in others. It's an extensive review of the adverse consequences of marihuana.

Now in addition to that—and she has—she

has I think a big impact on the chapters on cognitive influences of marihuana. Dr. Solowij has done two or three studies now in which she believes that she's identified a brain abnormality in chronic users of marihuana. I'll introduce it by saying that her study is done the way most of the studies in the latter days have been done, in which one identifies usually by advertisement a group of

individuals who admit to being chronic, heavy marihuana users. And you recruit them and ask them if they will take part in your studies. And then of course you have to construct a control group. You have to identify a group of people whom you will subject to the same kind of testing. And these are people who are matched again as best one can to the chronic use group in every way except they're not chronic users of marihuana.

Now all such studies, and there have been

probably hundreds of them done in the last decade, are beset with the difficulty is that it's unclear what should be a control group for chronic marihuana users. That is, chronic marihuana users are—first of all, they make up a very small proportion of marihuana users, since most marihuana users are not chronic, heavy users, very small percentage. So then you try to match people for intelligence and education and other drug use, which is often a very big problem because chronic marihuana users, high dose marihuana users are often users of other psychoactive drugs.

Having said all of that, let me tell you what

Dr. Solowij has published. And I suspect that it will come up again. We've talked already about the electroencephalogram in which you attach electrodes to the scalp and measure electrical brain activity. And this is a technique that's been utilized a long time and people look for diseases of the brain using electroencephalogram, look for epilepsy, look for evidence of brain tumour, look for evidence of vascular disease. But there is a very specialized electroencephalographic technique that I must explain to you. I think we can do it quickly.

If you have someone hooked up to an

electroencephalogram and then you give him a stimulus, a tone, have him open his eyes, have him even think of a particular thing—although it's most often done with a tone—you will get a response which has for years been called an evoked potential. In recent literature and in Dr. Solowij's papers it is always called an event-related potential. So that this is not just a routine electroencephalogram, although the individual has all the routine electrodes attached. But you give a particular stimulus and you tell him to pay attention to that stimulus. You often give him directions to anticipate the stimulus. So then the event-related potential not only measures something about the brain's intactness, but it also tells you something about the individual's ability to pay attention.

Her studies have all been done in the

following way. People sit in a room with headphones on. They are told that there's going to be a series of tones that come through the headphones. And then they're told to look for, to anticipate a particular tone. The tone is described to the individual before it occurs. "It will be high-pitched, it will be louder than the other tones and it will occur only in the left ear." So the individual's listening to this random set of tones and then the tone comes and he's to push a button. I hope all—I'll hope that's clear.

What you then see with an event-related

potential is a particular curve, particular wave form. And the most important one, although there is more than one, is called a P-300. If you look through the brain literature you'll see many references to the P-300. And that means that it occurs three hundred milliseconds, .3 seconds after the event. So electroencephalographers have in recent years spent enormous amounts of time looking at the P-300 wave. Measures brain intactness, it measures brain pathways, but it also measures the ability of your subject to pay attention to his directions and to focus his attention. Because actually if he's not focused you'll see a blip but it won't be the normal P-300 blip.

And P-300 wave forms have been said to be abnormal

in individuals under the influence of drugs, said to be abnormal in patients with schizophrenia, said to be abnormal in a group of people. So Dr. Solowij has now published two papers in which she says, with all this introduction, that the P-300 wave is abnormal in chronic marihuana users. It's abnormal. It has less amplitude, it has a slightly unusual wave form, and so she's now published this as evidence—although she has only a few subjects—that there is a chronic brain damage in marihuana users.

Now these are very high dose users. These

are often people who have used for more than fifteen years and often at you know, more than one—one—once a day, for fifteen hundred years—sorry, for fifteen years, more than once a day, hundreds of ingestions of marihuana. There's a small percentage of marihuana users who are chronic heavy users.

Now when I first read her studies I worried as

everybody does and as she does, about the adequacy of the control group. I mean, are these people really the same as the chronic marihuana users? And they have these different tone responses and therefore is there something that was different about them even before, or different about the marihuana users before they became chronic users, etcetera, etcetera.

Now I have in this chapter—in a certain

way I have not treated her with disrespect as a scientist, but I pointed out that there is a similar study by an electrophysiologist named Gloria Patrick who works at Tulane University in Louisiana. Gloria Patrick has now published two studies, the first one in which she agreed with Solowij and said chronic marihuana users have abnormal P-300 e.r.p.'s,

event-related potentials.

Then Professor Patrick did a second study in which

she controlled for age and she made sure that her chronic marihuana users were psychiatrically and medically normal. She excluded from the group

anybody who had a psychiatric diagnosis, anybody who had any kind of medical disease. And having done that, she published a paper in a very important journal called Life Sciences, in which she said there is no difference between chronic marihuana users who are medically and psychiatrically normal and age-matched to a control group in terms of the P-300 e.r.p.

So I believe at this moment, although Dr.

Solowij's publications have gotten lots of attention and identification of a brain abnormality in chronic marihuana users, I believe that her work has been called into question, quite seriously into question. And again, I believe that that little bit of evidence of chronic brain damage in marihuana users recently raised by Solowij and discussed widely in the Hall book which I know has been part of the Court's consideration, that she's wrong. I believe she's wrong.

Q When was the Patrick study done?

A It was done probably three years ago, because it was published two years ago.

Q And so it was after the—

A Yes.

Q -- Solowij—

A Yeah. In fact, if you look at the journal Life Sciences—if you look at my chapter eleven reference thirteen, you will see Nadia Solowij publishing two papers, one called "The Events of Long—The Effects of Long-Term Cannabis Use on Selective Attention and Event-Related Potential Study" that was published in 1991, and then Solowij in Biological Psychiatry presented the results again, very similar results in 1995. And then Patrick "Auditory and Visual P-300

Event-Related Potentials are Not Altered in Medically and Psychiatrically Normal Chronic Marihuana Users" was published in 1995.

And that journal, Life Sciences, contained another

article by Nadia Solowij which I didn't refer to here although I refer to it elsewhere in the document.

So one—and I guess I would also make the

important comment/observation, which I think everyone here knows, that when only one investigator has made a finding it really has to be confirmed by other investigators. If it's in the hands of one scientist or one group of scientists, although it's evidence, it's not acceptable evidence to the scientific community as a reflection of truth at a certain level. So the first group of people to try to confirm

Solowij's findings have said, "No, we can't find it." And no one else has confirmed it yet. So it hangs in abeyance. Okay?

Q Okay, let's go back then to where we were. We were about to start I think number twelve, "Marihuana and Intellectual Functioning".

A Yeah. And I actually had spent a fair amount of time talking about the acute effects and the distractibility and the acute memory effect.

Q That's right, we—

A Yeah.

Q -- weren't just starting, you were about to explain—you said it was a long story, the chronic—

A Well, I can do it without so much length now.

Q Okay, good.

A The first important thing to say is that obviously having identified this acute effect on memory and other potential acute cognitive effects or other cognitive effects, then investigators said, "Well, what we really need to find out is are there chronic effects of marihuana use, particularly chronic effects on cognitive function and intellectual function."

The—one of the reasons this got

placed on the research agenda—no, let me not do that yet. Let me say again as I said about Solowij, that studies of cognitive function rely upon this case control phenomenon that I've described to you before. I described it to you with the study of the mothers and the babies with leukemia. I've described it to you with Dr. Solowij's work. Which means that the way studies are done in this area is to take a group of people who are chronic marihuana users—they say, "I'm a chronic marihuana user." In some cultures, saying you're a chronic marihuana user brings about trouble, in others it doesn't. But, "I am a chronic marihuana user. Yes, I volunteer to be in your studies."

So then the investigator has to construct a

control group. And I realize I've done this about ten times now, hold my hands up: the experimental group, the control group. And we assume they are alike in every way except their chronic marihuana use. And that's a big problem. It's unclear if such control groups are alike. There always is a problem in constructing the control group.

Q I take it this is true no matter what your study is.

A No matter what.

Q It's not peculiar to marihuana.

A Exactly so. And again, what it is is an attempt to study a toxicity that has a fairly low rate of occurrence. You don't really need case control studies if we're looking at something that occurs in sixty percent of users, or even forty percent of users. But if you're speculating in this small group of chronic users some of them may have this slight brain defect, then it's not big and gross, they're not psychotic, they appear to be functional but we're going to find out this carefully studied little thing.

Okay, that's where we begin.

Now the first reports of brain damage in

chronic marihuana users all came from cultures in which there was a history, a cultural history of chronic marihuana use. Remember, that history does not exist in the western world, until now maybe, since there are now people who have used marihuana maybe for twenty or thirty years in this culture. Before the 1960's and 1970's, in the western world there was no cohort of chronic marihuana users. And indeed what began to develop as a cohort in the western world bore very little resemblance to the cohorts in other cultures. Now the most important two I'll comment on

are in Egypt and in India where there is a large group of chronic marihuana users largely confined to the lower classes in terms of educational status,

socio-economic status, education and literacy. That's not what happened in the western world but that's what happened in Egypt, where a psychologist named Sawif published a large number of studies in which he said marihuana users have chronic brain deficits, they have intellectual impairment, they have lower i.q.'s, they have many, many things wrong with them.

Now Sawif's studies were all done on men

in prison. Most of them were there because of cannabis-related offences and he constructed a cannabis-using group and compared them to a control group and said they have cognitive abnormalities, memory abnormalities, calculating abnormalities; they are significantly harmed by their marihuana use. Published these papers in the late sixties and early seventies. Almost every commentator in the western world said these studies are seriously flawed. I can easily express to you why. That the marihuana cohort were less educated, more—more often illiterate, more often rural, and there was very reason to believe they might have scored low on tests whether they'd used cannabis or not.

So Sawif's studies, although they're

commonly still cited, have not been acceptable by western psychologists. But again, Sawif accomplished the function of putting the psychological damage, the brain damage issue on the map and on the research agenda.

Okay. Now we come to the western world.

There actually were a number of studies in India which are the same sort, that they were done in a cohort of lower class, uneducated, economically depressed individuals. And it's unclear if those findings, which are not so consistent as Sawif's, relate to marihuana or relate to something else.

So then we come to the 1970's in which

people in the western world decided we had to find out about the likely intellectual and other harms of chronic marihuana use. The United States government funded three important studies, commonly referred to as field studies. They are studies which all in general looked at a cohort of heavy users of marihuana and tried to construct a control group to see what the differences of heavy use were. The three sites of study were Jamaica, Costa Rica and in Greece.

Interestingly, a group of Canadian

scientists who were not part of the initial group also went to Costa Rica—I think Costa—I'm sorry, no, they went to Jamaica. So there's a separate report by some Canadian scientists very much the same as the studies done by the Americans, it just happens they were not funded by the American research apparatus.

Let me quickly tell you that the three

studies resulted in no findings. There was no evidence of cognitive harm, brain dysfunction, memory loss in any of the three field studies in the 1970's. Although again, small groups of people, the difficulty of constructing a control group, but using standard tests of psychological function, memory, intellect, none of these field sites identified cognitive harms related to marihuana use.

I guess one could say well, we could have

stopped there. But we didn't, of course. Everybody always wants to do more research.

So people began looking at western users

even though they didn't have history of chronic use, they only had a few years' use. And in the paper—I'll come back to the follow-up of the Costa Rican users—on page 12-5 in the middle paragraph I've gone through quickly a large group of studies mostly focused on memory, to see if chronic marihuana users had altered memory functions. And these were done by recruiting young people, most of them college age or thereafter who had already developed heavy marihuana use habits in the late sixties and early

seventies. Almost all of these studies gave a negative result. There were a couple that showed some evidence of impaired memory and I've mentioned that. "Two studies in the 1970's found memory deficits related to chronic high dose marihuana use, but three others found no related marihuana differences. On a variety of other tests no differences." And then I cited the fairly well known study of American Rastafarians who are very, very heavy users of cannabis and who had no obvious abnormality on any psychological test and compared to standard scores. They had normal i.q.'s. They had been smoking enormous amounts of marihuana daily for years. Okay.

So in a certain sense by—in the decade

of the 1980's I think western scientists had lost some of their zeal for studying the possibility of chronic brain damage or brain damage in chronic marihuana users. There were very few studies conducted in the 1980's, very few.

But then in the 1990's there have been two

important studies which have gotten lots of attention. The one by Harrison Pope I'll discuss first because it was done in college students in the Boston area. They were heavy users but actually only of about three years' duration. And most people would not have figured this kind of use, even heavy use to have caused cognitive impairment. Pope brought them into the laboratory, had them do a number of studies, and found some abnormalities compared to a control group.

Now Pope admitted in his paper that he had

difficulty constructing a control group because his controls had less use of other psychoactive drugs, they had lower S.A.T. scores—I'm sorry, they had higher S.A.T. scores than the control—than the marihuana use group. They also had slightly higher i.q.'s. So there's a reason that they might do a little bit better on tests. But he tried to adjust for all of this, pointing out that it's hard to control a control group.

Now Pope published his studies in the Journal

of the American Medical Association showing some abnormalities of a few tests, particularly something called the Wisconsin Card-Sorting Task, which is designed to measure mental flexibility. Basically people are just given a deck of cards and told to sort them. Jacks here, clubs there, rules change sometimes but—in fact I'm not even sure if they're always standard cards of jacks, kings and queens. But they're card-sorting and you look and you—you—we measure how—how many cards you can sort and how many abnormalities. And his chronic marihuana users were able to do less card sorting. For example, in the first but not the second trial of the card-sorting task heavy marihuana users sorted fewer items correctly, 51.3 compared to 53.3 for light users.

I should point out that his control

group were not people who abstained completely, but who were in a light use group. That raised some questions, but he was able to justify what he did.

He also gave a memory test and subjects were

given five chances to recall words from a list of twenty words. At the test completion the average number of words recalled by light users was 15.3 and the average for heavy users was 14.9.

Now I obviously cite these to make clear that

everyone knows these are not very dramatic differences in people who have been heavy marihuana users for three years. And I again cite the fact that he had a great deal of difficulty in constructing a control group and that his marihuana users were—had lower i.q.'s, lower S.A.T. scores and heavier drug use of other sorts. But the paper's now there in the medical literature and says that there is a cognitive impact of chronic marihuana use, and it's received a lot of attention. People will wait to see if he can reproduce those results.

Q So there's only the one at the moment—

A Say what?

Q There's only one that achieves those results?

A I think there's a second. I'll tell you about that, too. An anaesthesiologist, a man who's in the anaesthiology department at Iowa named Robert Bloch, R.I. Bloch, has actually published some fairly similar results. He recruited a group of heavy marihuana users and had them do a series of standard i.q. tests. In fact they're the standard twelfth grade tests in the state of Iowa. And then he had them do some other computer-based testing of a more intricate sort.

Bloch's studies received a lot of attention

because he had their fourth grade test scores. These are all Iowa residents and they were all willing to give him their names and so he was able to try to balance his groups as to their fourth grade intellectual capacity. So then when they were adults, the non-users and the chronic users, or the light users versus the chronic users, he thought he had matched them beforehand for their intellectual abilities. And the study again raises some—some interesting questions. Bloch for instance said that heavy marihuana users who reported seven or more uses per week for an average of 6.5 years scored lower on two sub-scales of the i.q. test—that's the standard twelfth grade test—and one computerized test of memory.

This has raised, although a small group of

people and a very slight effect, it's raised again the possibility that we're discussing here, does chronic marihuana use cause cognitive defect and apparent brain damage.

Now Professor Zimmer and I have criticized

Bloch's study for the following reason. We found out that Bloch published a preliminary version of the study and that preliminary version was published in a United States government publication. And in that version he did not have a division between the heavy use group, people who use seven or more times, and the division of people that use five to six times. And that's actually a bit of a strange division. They all seem to be pretty heavy users, that is, people using five to six times a week versus seven times or more per week for 6.5 years. But he in his first publication, his preliminary results, he collapsed these two groups together and the differences were nowhere near as dramatic. In fact, they had a difference from the control group on only one scale, i.q. scale, and they also were different on their fourth grade scores.

Now Bloch never mentioned this when he

published his final paper, and made the division between the seven times more who had abnormalities on the two i.q. tests and one computerized test of memory. And it is striking to us that the individuals who used five to six times a week had no abnormalities. But he broke out a group of seven or more times a week who had abnormalities. And it raises the question, since he had not brought out that group before when he presented his data the first time, why did he bring it out this time. And one of the reasons he may have done so is to get more dramatic differences. That doesn't mean the differences are not there, but it looks a little bit like post-analysis manipulation.

I would make one other point. Bloch's study

has been criticized because he brought these users into the laboratory, many of whom who had used seven times per week or more for 6.5 years, and he told them not to use marihuana the night before, the day before, and to come into the laboratory clean. But he did no urine testing to insure that was true. So people have raised the possibility that Bloch's findings had to do with requesting daily marihuana users of 6.5 years to abstain and then not doing any testing to see that they had abstained, so that his findings may indeed in fact relate to use on the day of the test.

I'm—you know, I'm worried about the fact

that I'm—I might you know, sound as if I'm looking for reasons to criticize the studies. And at a certain level of course, I am. I'm a critical reader. It's my job to not just report what these people have said, but since there are these—only—only these two studies to show some slight cognitive harm with chronic marihuana use, then I—it's my responsibility to be very critical of the studies. So I have looked very carefully for reasons to worry about these findings, which have not been found by other people and were not found generally in multiple studies in the 1970's.

So I have concluded—Professor Zimmer and

I have concluded our chapter—we've actually talked about a few other studies which have shown some memory deficit supposedly in chronic users. We've not felt them to be very strong studies. So we've concluded our chapter by saying that we think there's no convincing evidence that chronic marihuana use is associated with cognitive deficit. Now if we're wrong, the cognitive deficit appears to be pretty minimal, maybe of no functional importance, and it occurs only in an extremely small group of marihuana users who are very high dose users. In fact, Dr. Bloch's data could be interpreted that individuals who use marihuana daily for 6.5 years have no cognitive deficits five to six times per week, while those who are using seven or more have this slight finding, which may be lost in the analysis—or found in the analysis. Okay?

Q All right. You mention the Schwartz study—

A Well, and as I was—

Q -- I don't know if there's a need to go—

A -- sitting here I realized I wanted to say something about the Schwartz study, too.

Q Just before you—you move on to Schwartz, I notice the reference to Pope and Todd at 12-7 and the criticism there in relation to the groups as between men and women.

A Oh, I'm sorry. Thank you. I didn't mention that. If—there was no distinction in terms of amount of use between the males and females in the groups, which is—when you recruit heavy marihuana users you always get more men than women. Always. Always. And in fact most of the people designing these studies are worried about the fact that they have more men than women in most of the groups. And when they start recruiting they find that their non-using group fills up with women volunteers and their using group is mostly men. So they worry about that balance.

Now what Pope did was to try very hard

to balance and he had a group of chronic female users. In other words, in his high use group there were a group of females. He then did the analysis by gender, and all of the deficits melted away. That means that this evidence of chronic harm occurs only in male users. Well, what does that make one think of? It makes me think that this is not a pharmacological issue. This is not necessarily an effect of marihuana at all. I'm not automatically saying that chronic using women are smarter than chronic using men. They may be. But that males are heavier users than other drugs, play more football and soccer, a variety of other things that might diminish their cognitive abilities in this

kind of testing. And—but more important than that, merely dividing the subjects by gender and suddenly having all of your intellectual deficits disappear, raises questions about the probity of your publication, the probity of your scientific analysis. Because I can't think of any reason why all differences should disappear in women who've also been users at this high level for three years.

Thanks for reminding me of that. I'd forgotten.

Q All right. And the Schwartz study?

A Dr. Richard Schwartz is a pediatrician in Vienna, Virginia who very frequently is listed in the

anti-marihuana front. He's a hard fighter. He's a colleague of Dr. Nauhaus, colleague of Dr. Tenant. Works together with them to fight the marihuana reformers and to identify immoral actions on the part of the marihuana reformers.

Now, Dr. Schwartz has published a study which

has attracted a lot of attention. In fact, both Bloch and Pope refer to Dr. Richard Schwartz's study. Now I've been aware of Dr. Schwartz's study almost since the day it came out. I know him well. He published this paper in 1989. And I looked at the paper and I have previously criticized it. Let me tell you quickly what he found.

He—he was the medical director of a

treatment program based in the United States called Straight Incorporated. Straight Incorporated had come under lots and lots of criticism and in fact—although it may be a little unfair—its licence has been removed in the United States. It no longer exists. And the reason the licence was removed, because of multiple lawsuits and complaints by young people who were forced to go to Straight by their parents because they were marihuana users and users of other drugs and in trouble, but once they were in Straight they discovered that they were subject to corporal punishment by graduates of the program, they were subject to physical restraint and were not permitted to leave when they wanted to even though they had not been committed legally. But this treatment program looked like a jail.

Now many of the graduates of Straight say,

"It saved my life. It's wonderful." Many of the parents think it was the greatest thing in the world, etcetera. But its licence has been removed in four states in the United States, it no longer exists.

Now—so Dr. Schwartz published this

study which said the following. He studied ten heavy users of marihuana, adolescents whom he said were marihuana dependent, that is they were

using so much marihuana that it was their main medical problem. And they were admitted by their parents to this program. At the time of their admission he measured a number of tests of their memory. And in fact he measured it two to three days afterward and they had abnormalities in two tests of memory of the seven he administered. That's in itself an interesting finding.

And then he measured them six weeks later after

they'd been in the program, and although they'd improved somewhat, one of the tests was still abnormal, that is comparing inside the heavy use group, still abnormal.

Now of course what am I going to talk about now?

I'm going to talk about the control group. Dr. Schwartz generated two control groups for comparison of memory testing to these adolescent marihuana dependent people. And in two papers he's published about this study since, in conferences held in Paris by Dr. Nauhaus, he's taken on a pretty self-congratulatory tone about, "We had two control groups and they showed that this memory deficit persisted for six weeks, therefore if all of this previous research to say that the memory defect doesn't persist is wrong, all of you people are wrong."

Now the paper basically sat on my desk for

five or six years until Professor Zimmer and I began preparing this book and this chapter. And then we looked at it very carefully and discovered some pretty amazing problems. The abnormality in the memory two to three days after admission was abnormal compared to the control groups when the control groups were combined, nine people from one control group and ten people from another. So then if you compared the memory function in his marihuana using young people to the two control groups combined, seventeen people, they were abnormal, two to three days after admission.

Then when he compared them—on two tests. Then

when he compared them to his control group at the end of six weeks they were abnormal from the combined control group.

Now I need to tell you what the two control

groups were. One, like the adolescent marihuana users, were admitted to the program. They were committed to Straight Incorporated. They didn't have marihuana problems. In fact, it's a little difficult to tell what their problems were; they may have been alcohol, but it's not clear. And in fact at the time of admission he said they didn't have too many drug problems, but they were admitted.

Then his second control group were brothers

and sisters of the marihuana patients who had no drug use problems as far as anyone knows. And he tried to match them as to age and intellect, etcetera, etcetera.

Now the critical thing when he broke out

the two control groups, the abnormality in memory at both ends disappeared when he compared it to the committed patients. That means that the control group for the adolescent marihuana use which consisted of people who were also in the treatment program, there was no difference. There was no memory deficit between those two. So if there was a memory deficit from the community controls, the brothers and sisters, then the memory deficit didn't have to do with marihuana, it had to do with being admitted to this program. And having interviewed a number of people from this program, the most likely explanation for Dr. Schwartz's studies is how angry and furious those kids were at him as the medical director of this program and at their parents for forcing them to stay in this program where they were physically restrained, not permitted to leave, had to follow a series of rules, told they were marihuana addicted and that they were no good.

I believe that's the reason there was some

defects in this testing, because they didn't cooperate with the testing. And I have very strong evidence for that, since there was no difference in memory scores between the two admitted patient groups, and the only difference occurred when he added in the outpatient controls, who were not good controls. After all, they were not drug users. They were not in trouble. They were community samples.

So after five years of Dr. Schwartz's study

floating around, Professor Zimmer and I have read it carefully and think it adds very little to this argument.

Actually that's wrong. It adds something very

important to this argument. It shows that if the control group is constructed properly you may see no difference in chronic marihuana users. If you construct a control group of individuals who are admitted to a program, that looks like a good control group and you don't see any impact of the drug. If you compare them to outpatient, smart kids who are not in trouble, then you see differences. Outpatient, cooperative, smart kids, then you see differences. That shows that by constructing your control group properly you learn something, which is that marihuana had no impact on the memory of these children.

Q All right. Let's move on then to chapter thirteen, "Marihuana, Motivation and Performance". The myth there that we're dealing with is, "Marihuana causes an amotivational syndrome. Marihuana makes users passive, apathetic and disinterested in the future.

Students who use marihuana become underachievers, and workers who use marihuana become unproductive." What were the results of your investigations into that myth?

A In the late 1960's a couple of people writing clinically about marihuana patients said they had seen some heavy marihuana users who appeared to be withdrawn, listless, without energy, apathetic. And at least two of these writers coined the term, "They almost seemed amotivational. They had an amotivational syndrome."

And then in 1971 or '72 two people

in the group of scientists who were collected together to question what we believed about marihuana, two psychiatrists named Kalanski and Moore, published findings in which they showed that a group of adolescent marihuana users had a very distinct amotivational syndrome, that is they didn't—would not work, they had dropped out of their previous life, they were apathetic, listless, withdrawn, lethargic, had no taste for life, had anomie, and that this was caused by marihuana.

So Kalanski's and Moore's statement of

1971 or 1972 that marihuana causes an amotivational syndrome is perhaps the most important and frequently stated claim of those who are concerned about adolescent marihuana use, and it's stated over and over and over again. Our quotes are fairly contemporary. "The marihuana issue is about the costs to society of drug-related lost productivity." Donna Shelalagh said that in July of 1995, the Secretary of Health Human—Health and Human Services in the United States. "An amotivational syndrome has been reported in heavy chronic marihuana users. It is characterized by decreased drive and ambition." That's a NIDA publication. Although it's undated I know it came out in 1995.

"Marihuana keeps a person from functioning

at full potential. It makes an above average student average and an average student below average." That's the Attorney General of the state of Pennsylvania. Again, I don't know exactly the date but it was the 1990's.

"The amotivational syndrome is easily

recognized," etcetera, etcetera.

The amotivational syndrome is a wonderful

subject to review because there's so many ways to look at it. We found for instance a series of laboratory studies in which individuals who were willing to be admitted to a hospital site were given work tasks or ability to earn money or a variety of tasks and then they were given marihuana and their

productivity on the task was compared to their non-marihuana use or a group of controls. In fact, the most interesting and most—in many ways most important one of these was done in Canada. That would probably be—

Q Page 13-5.

A And the reference number is?

Q Thirty-one.

A Yes. C.J. Miles, "An Experimental Study of the Effects of Daily Cannabis Smoking on Behavioural Patterns". And it was published by the Addiction Research Foundation in 1974. And then Dr. I. Campbell, who was part of that same study with Dr. Miles, "The Amotivational Syndrome in Cannabis Use with Emphasis on the Canadian Scene".

So, a series of laboratory studies in which

we give the drug to individuals and see if it decreases their commitment to work in a variety of settings. We have those kinds of studies.

The three field studies that I talked to

you about, Jamaica and Costa Rica in particular, the economic capacity of those men who were cannabis users was compared to controls, and in fact in one instance there was actually a study done using a quantitative assessment of the amount of work that a Jamaican farm labourer performed after smoking marihuana. It was done a number of times to see if his commitment, his ability to work decreased because he smoked marihuana.

Then there are a series of studies in the

United States of grade—school grade performance of marihuana users versus non-marihuana users in high school and in college.

And then my favourite since I have been involved

in the arguments about workplace-based urine testing, there are now four publications coming out of the same database about wage-earnings in marihuana users versus non-marihuana users. In the United States there is something called the National Longitudinal Survey of Youth in which twelve thousand young people have been interviewed quite extensively over the years, then in two years, four years apart they were asked a series of questions about their drug use. And then there’s a New York State sample of young people who’ve been interviewed extensively by a group of epidemiologists at Columbia University, learning lots about their work, their life, their performance, their drug use, etcetera, etcetera.

Having looked at all of these parameters, there

is no support for an amotivational effect of marihuana. None. Zero.

Let me—

Q And that's a longitudinal study then?

A None. Let me—let me give you a couple of specific things.

Q But the study itself as its name implies is—is like the Tashkin study, a longitudinal study—

A Some of the studies have been longitudinal, although again most of them were not.

Q I'm talking about the New York State one.

A Yeah. Well, at least the New York State one—it's called a Longitudinal Study of Youth, but at only two points in time have people looked carefully at marihuana use and wage capacity. Let me tell you about that.

There have been four studies of that

data set, the National Longitudinal Survey for Youth, and somewhat to the embarrassment of the investigators, in one study the wages of marihuana users and

non-marihuana users was the same. In the other three the marihuana users all earned significantly more money. So the ability to generate wages, which is at least one measure of motivation, would seem to not be impacted by marihuana.

Now an earlier—an early analysis of the

1984 sample said that marihuana users actually were—even though they earned as much money or more, were absent more often, they were not so good workers. But then that scientist did a longitudinal study in which he looked at '94 and 1988, and found that there was no difference in the number of hours worked per year. That is, they didn't have more absences if they were marihuana users, and they earned significantly more wages. And in fact in one of the four studies I think the differential was twenty percent higher wages by marihuana users. Now that's a pretty strong blow against the idea that marihuana use causes amotivation.

I wanted to tell you something about the

Canadian study because it is—it in fact is really very impressive. I then will go back to the—the field studies.

"Canadian researchers designed a similar

token economy study,"—that is, individuals had to work. I can't remember whether they were putting together stools or—they had some task they had to do. And if you worked hard you got more tokens which you could cash in for money at the end of the study. Other studies have been done in which you could use your tokens to buy marihuana, but in this one that was not done.

"Canadian researchers designed a similar

token economy study to evaluate marihuana's impact on motivation. They found some reduction in work efficiency at the beginning of the marihuana use period. However, efficiency quickly increased and surpassed abstinence levels. And although subjects consuming the most marihuana spent the least amount of time working, they were no less productive because when they worked they worked harder. In addition, during the period of highest marihuana use subjects at the Addiction Research Foundation, organized a unified collective action and successfully demanded and negotiated for increased wages. At the end of that time they worked even harder."

I absolutely love that.

Q That was in 19 --

A That was just one of the—

Q -- 74?

A Reported in the Annals of the New York Academy of Medicine, by Dr. Campbell, a Canadian scientist.

Now—

THE COURT: I have a question—

A Yes, ma'am.

THE COURT: -- on those types of tests. Is there any

way to control for the influence that might be present by virtue of the fact that the subjects are actually participating in this study? In other—in other words, in their normal life they may behave one way but when they participate in this study they would—might behave another.

A The answer to your question is no, there is no adequate way. All studies in which individuals are isolated and pulled out of the real world and studied under a microscope as it were, in a laboratory, all have what actually people have called the Hawthorne effect, that the impact of the study may be clearly important. What investigators do is to try to repeat the study numerous times, do it with different mechanisms, for instance token economy, actual wage payments, sometimes people are paid in their freedom to socialize. But no, they're—they're all artificial. They're all artificial in the sense that I understand your question. People may behave differently

in the real world than they do in a hospital being studied for their marihuana behaviour. Absolutely.

MR. CONROY:

Q Does that—does that lead us to—do we then—if we're trying to ascertain the health risks or dangers of any—any drug let's say, can we rely solely on the scientific method and the control groups, or do we have to look at anecdotal evidence as well as the scientific to come to our conclusions?

A Well, I think we have to look at both. We have to look at different kinds of informations. We gain something from a laboratory study in which we give T.H.C. to animals. We gain something from reports from marihuana users. We gain more if those reports are controlled in some fashion. And in a claim of amotivation we gain something by putting people in a hospital and having them work for a particular kind of task and expose them to the drug and see if the drug affects them. And I like the amotivation very much because we have the laboratory of real wages and a reported sample. And I didn't mention to you the grade

point averages in which college students by and large where marihuana users have had either equal or better grades than non-marihuana users. High school it's a little different, a little more disparate and there are some evidences of heavy marihuana users being quite dysfunctional in high school, although almost all the studies we've found have indicated that their dysfunction preceded their heavy use of marihuana. They were already having grade trouble before—they cleaved to marihuana perhaps as an answer.

But again returning to Your Honour's

question, all laboratory studies are artificial. Almost all studies in which drugs are evaluated in humans under controlled circumstances are artificial. They give us important information but they form only some part of the picture. And real world studies, surveys, questionnaires, observations all make up part of the package of information, usually inadequate, with which we have to determine policies, decisions and regulation.

I'd like to—again, I'm sorry but let me

quickly mention to you that after all of these years, twenty years of amotivational studies, no one had ever found anything to support the amotivational syndrome. And then a NIDA-funded scientist in the late—in the early 1990's named Fulton did an astonishing experiment in which people were allowed to work—people were—under the influence of marihuana were assessed on four tasks. They were all terrible tasks. They were all extremely

boring. One of them was to sort pieces of metal by colour and size into different bins. One of them was to alphabetize a series of

seven-letter nonsense words generated by a computer. Just had to alphabetize it as a-b versus c-d, even though the word made no sense. So there were four terribly boring tasks.

The measure these scientists used for

motivation was how hard would someone work on his most boring task, the one he hated the most, so he could then spend some time on his least boring task. Strange set of ideas to begin with. However—and I believe it was designed to show that marihuana would have an amotivational effect, that is people wouldn't work very hard to escape their most boring task. They all did. On marihuana they worked the hardest to get rid of the boring task.

But unconvinced by the results of their laboratory

study, Fulton and his associates conclude that the complicated effects of smoked marihuana on the motivational aspects of human performance need to be studied more rigorously, under a wider range of clinical epidemiological and experimental conditions. Remember I told you that's the language that people use in grant applications.

There is no evidence for an amotivational

effect of marihuana.

Q Anything you want to comment on Kandel[phonetic], or is that much the same?

A Kandel has been a very important researcher in the stepping stone, gateway hypothesis. But we've used her here because it's some of her work that's reported higher wages in the marihuana users. She also recently has published an explanation, which I guess I should cite, in which she says marihuana users earn higher wages because they take risky jobs which have—they choose risky jobs which have higher wages, and she thinks over the long run the wage differential will decline because these jobs will not give them continued high earning. It's a strange speculation. I'm not sure she has much reason for it. But she's offered an explanation for why drug users have higher wages.

Q All right. Should we move on to fourteen?

A Yes.

Q "Psychological Disturbance and Mental Illness". The myth there that we're dealing with is, "Marihuana causes

psychological impairment during—sorry, marihuana causes psychological impairment. During intoxication marihuana users become irrational and often behave erratically. Chronic marihuana use causes permanent mental illness, including schizophrenia."

A Maybe I can do this one quickly. That there's little evidence that marihuana intoxication causes people to behave in a crazed or irrational manner. There's no convincing scientific evidence that marihuana causes psychological damage or psychiatric illness. There are reports of course, particularly of naive users, becoming panicked and frightened the first or second time they smoke marihuana, although the number of such panic anxiety attacks apparently declines as people become more used to the effects of the drug. And it's quite unusual now for an individual to arrive in an emergency room with a panic reaction due to marihuana. It was not so unusual in the sixties and seventies. I saw a number of such people, young people.

Now there has been a claim that there's

a specific cannabis or cannabinoid psychosis. It's very rarely been reported in the western world. It may have—it may occur occasionally in individuals who consume large doses of cannabis orally, which I've already discussed with you as being a different issue. But I do—I must focus on one thing because I'm pretty sure it's going to come up, and this is the Swedish conscripts study in which Swedish scientists have looked at a group of schizophrenic patients and discovered that they had lots of data on them because they were once in the military. And as they came into the military they answered very extensive questionnaires and demographic and other data in hopes that one would learn something from this kind of study. And what—I—Dr. Andreasson found was he described cannabis use as a risk factor in the occurrence of schizophrenia. That is, in the conscripts who became schizophrenics a few of them had significant consumption of marihuana before they became schizophrenic. So Dr. Andreasson and some critics of marihuana reform have decided that marihuana can cause schizophrenia in some people.

There are many, many other interpretations.

The most common one is that if you look at schizophrenic patients who are using marihuana, versus those who are not, that in general you find a pattern of less severe schizophrenia, less florid hallucinations, less life problems, less withdrawal. So the assumption is either that marihauna's helping them and they're using marihuana to treat some of their own symptoms, or that they are a little bit better than the other schizophrenics and were social enough, involved enough in social lives that they could get marihuana. And so there's all these interpretations to detract from Andreasson's idea that in a few young men marihuana use was related to the later occurrence of schizophrenia.

I don't have much more to say about it. It was

about six percent of the schizophrenics who had been marihuana users beforehand. If marihuana has an impact on schizophrenia it may be more likely to be used by people to diminish the symptoms, although in some schizophrenic patients marihuana use might precipitate the schizophrenia or

might cause it to emerge, might interfere with other medication. So I'm not saying that marihuana can be used without risk by individuals with serious mental illness, but I don't think there's any evidence that marihuana causes crying mental illness.

Q I understand your evidence though like the pregnant woman, you wouldn't recommend that people with a mental illness of any kind would smoke marihuana.

A I wouldn't.

Q Just a point on the panic attacks, you said that they're now rare. When you did see these were they long-lasting—

A Oh no, no.

Q -- were they short-lived, what—

A Quite—quite short-lived. Most of us who worked in hospital emergency rooms in the late sixties and early seventies saw people who were panicked because they had tried some marihuana for the first time, heart pounding, couldn't talk, mouth dry, funny thoughts rushing in. And they had used it in a setting in which they were maybe not thoroughly protected. So the advice to people was don't use marihuana for the first time in those settings, and if you're going to use marihuana use it with people you can trust and people who will help you and people who will talk to you calmly.

But despite that advice and despite the fact

that it occurred, most of these young people were naive users and given a little reassurance and a little time, little talk, they were fine.

Q You said that the incidence of that seems to have gone down. Is this from the Dawn studies or statistics—

A From the Dawn and other studies, that the acute panic reaction is rarely reported nowadays in the western world. Although it still happens, but the prevalence seems to be significantly less. And the assumption is that's because this is no longer a strange substance in this culture. Marihuana is here and it's here to stay. And people have learned something about its use. And this kind of strange, fearful reaction is now unusual.

Q Let's go to fifteen then, "Relationship Between Marihuana and Crime". The myth there is that, "Marihuana causes crime. Marihuana users commit more

property offences than non-users. Under the influence of marihuana people become irrational, aggressive and violent." What do you say about that?

A Well, we thought perhaps—let me start this way. If you look at Dr. Grinspoon's[phonetic] very widely received book called "Marihuana Reconsidered", he thought the issue of marihuana causing crime and violent crime had been put to rest. Because every government commission, every study that had looked at it said that marihuana users, if anything, are less aggressive than

non-marihuana users and there's no real association of marihuana with criminal behaviour, except for the crime of the possession of marihuana. And that marihuana users are not over-represented in any kind of criminal statistic.

What we have found much to our amazement, like

all marihuana myths none of them ever die. And this one has come back again. The strong use(sic) between marihuana use and violence which was stated by the last American drug czar, "Chronic effects of frequent marihuana use may include pervasive anger with easy provocation to hostile aggression even against loved ones." And so we now hear these things being stated once again.

Throughout the twentieth century every serious

scholar and government commission examining the relationship between marihuana and crime reached the same conclusion, including prominently the—the Ledain Commission. The vast majority of marihuana users do not commit crimes. Among marihuana users who do commit crimes, marihuana does not play a causal role. Almost all human and animal studies show that marihuana decreases rather than increases aggression. So the idea of the crazed marihuana user painted by Harry Anslinger, which we thought had gone away, has not entirely gone away and is being described again. But there is not any evidence, any evidence that such a thing is true.

Q The Reefer Madness movie.

A Yeah. The movie was never called Reefer Madness except by latter day people. It was initially marketed as Tell The Children. It was one of three movies that Anslinger encouraged Hollywood movie makers to make around the time of the marihuana tax act to show that marihuana's a terrible, terrible drug causing violent criminality, insanity, sexually rapacious behaviour and other horrible things.

Q All of the studies, whether it's been specifically those looking into the causal relationship with crime or the earlier one in terms of mental illness and so on, did they ever have any of the subjects in the studies start to act out in those ways depicted in that movie?

A Well, it's—no. It's very important that we now have an enormous experience of giving large doses of marihuana to people in laboratories and people in hospital settings, and I've never heard anyone report an outburst of violent behaviour among an individual—by an individual given a large dose of marihuana.

I'll make one quick comment. Reference

number thirteen was recently cited in the debate about the impact of marihuana on homicide. A prominent and respected investigator in New York interviewed a number of men who had been admitted—admitted, had been incarcerated for homicide in New York State. And fifteen percent of those men said that they had been on marihuana at the time of the homicide. And I think of those maybe half of them said they'd been only on marihuana at the time of the homicide, which cut it then down I guess to about six percent. And in the investigator—and in questioning the individual felt that there was no evidence that marihuana had contributed to the homicide, although this is well after the fact.

The reason I tell you all this story is because

it was recently cited in the debate as proof that marihuana caused homicidal behaviour in humans. The fact that some people who had committed a homicide said that they had been on marihuana was accepted by him as evidence that marihuana caused the homicide. There is no evidence that's acceptable to say such a thing.

Q Is there anything else specifically that we should be aware of arising under this chapter? You mention the—at page 15-4 the National Commission on Marihuana and Drug Abuse appointed by President Nixon. Any comment on that?

A Well, the Schaeffer Commission, which published its report in 1972, concluded by saying, "Some users commit crimes more frequently than

non-users, not because they use marihuana, because they happen to be the kinds of people who would be expected to have a higher crime rate wholly apart from the use of marihuana. In most cases, the difference in crime rates between users and non-users are dependent not on marihuana use per se but on these other factors." And we quoted that because it was stated by President Nixon's conservative commission in 1972, and it seems to us to still be true.

Q And that commission was otherwise known as the Schaeffer Commission.

A The Schaeffer Commission.

Q All right. Let's move on then to sixteen, "Marihuana Influence on Driving Ability". The myth stated there is, "Marihuana use is a major cause of highway accidents. Like alcohol, marihuana impairs psychomotor function and decreases driving ability. If marihuana use increases an increase in traffic fatalities is inevitable." What do you have to say about that?

A Well, it may be one of the most important things that we discuss here. Since alcohol is—is irrefutably associated with vehicular accidents and vehicular mayhem and death, it has been a proper concern of people to say—to question, to query is marihuana associated with driving misadventures. What we have are three kinds of studies. Actually, let me say four kinds of studies, the fourth one being an epidemiological survey. We have a series of studies in which individuals have looked for deficits in psychomotor performance in individuals in a laboratory dosed on marihuana. And as I said before, there is no surprise that you can find in a laboratory that people given marihuana may have a deleterious impact on their performance. And a scientist named Herbert Moskowitz, who has been very prominent looking at the impacts of marihuana on driving-related behaviours, has published a number of studies in which he shows that people on marihuana, they don't do divided attention tasks very well. That is, you tell them every ten seconds you have to beep the horn—no, let me—let me describe it more accurately.

You give them a task in which they're

> required to pay attention to two things at once. They are not driving a car, but they're sitting there monitoring a set of lights which when it comes up red they have to push a button. But at the same time they're monitoring those set of lights they have to monitor something else a little bit out to their periphery. So they have to keep looking back and forth.

Moskowitz has shown quite convincingly that

> marihuana diminishes people's ability to perform this kind of psychological function well, a divided attention task. Okay?

Now, so Dr. Moskowitz's papers all say, "Since

> this kind of skill is related to automobile driving, I fear that marihuana may cause people to drive badly."

Now in addition to Dr. Moskowitz's studies

> there are many such laboratory studies to show a diminution in psychomotor skills, reflex time, etcetera, etcetera, that might relate to driving. So let's set those on one side, large numbers of studies that say that in laboratory assessment that marihuana may diminish some of the skills that might relate to driving. An important issue.

However, then we move to the next set of

> studies, which are driving simulator studies and you know, almost like the car tracking things in penny arcades. Investigators have done many studies with the driving simulators in which you may be even given a panoramic t.v. screen and the road moves in front of you, and you may be given noises and you're asked to brake or pass or do various things. You're actually not driving a car but you're in a simulator.

Now the simulator studies are very intriguing

> because by and large marihuana, even at relatively high doses, has relatively little impact on people in driving simulators. There's no—not much of an increase in braking errors. People can put on their brakes just fine. They'll swerve the wheels when you send something into them. They'll do pretty well. In driving simulator studies marihuana's been shown to have an effect on some performance measures, particularly those involving divided attention. However, overall impairment from marihuana is much less severe than that produced by alcohol at doses below intoxicating levels. That is, that two drinks which gives you a blood alcohol level of .04 percent or .05, you see a much greater impact on simulator studies than people given larger doses of marihuana. I'll explain larger dose of marihuana later. It becomes real important.

So that simulator studies did not bear out

> the fears that Dr. Moskowitz and other students of driving skills had.

Now, then the next thing that happens is that

> you give people actual on the road tests of their driving ability. There are many, many of these and they've been done in many ways, sometimes with an observer sitting next to the driver, sometimes with an observer with a dual set of controls in case somebody gets into trouble. Sometimes the observer is in the back seat giving a global assessment of driving. And of course certain other things can be measured such as distance behind a car that's being tracked, does the marihuana user get too close, a driving task in which the individual is told to pass as soon as the road is clear ahead, measurement of braking time, how often the individual makes braking or steering errors. And again the general finding is that low doses of marihuana caused little or no impairment on on-road studies. Even high doses of marihuana caused less impairment than low doses of alcohol. And the study which causes marihuana critics to laugh derisively but which has been shown over and over again, that people under the influence of marihuana become more cautious. When they perceive themselves to be high they drive more cautiously. They slow down, they increase their distance behind a car they're following, and they are very hesitant to pass in any sort of situation even though they've been directed to pass. If there's a hill way up ahead, they still have a dotted line but they can see the white line up in front, they're hesitant to pull out. And if you interview them, the reason is quite clear. "I felt high. I was afraid I couldn't do it so I slowed down."

Now the reason I mention this is because

there is then a significant literature that says

risk-taking while driving a car on marihuana is reduced. And that's compared to a literature regarding alcohol which says risk-taking while individuals taking alcohol is markedly increased. Individuals who are under a psychomotor deleterious effect of alcohol seem not to realize it. They drive faster and more aggressively and take more risks and their judgment is more impaired.

So a little bit high on alcohol appears to be

much dangerous than a little bit high on marihuana.

I quickly say, should people drive while high

on marihuana? No, no, no, no. But giving those two things, giving those three things I've mentioned, psychomotor tests, driving simulators and on the road driving, there is relatively little evidence that marihuana is an important contributor to vehicular mishaps.

And then I have two other things I want to say.

You wanted to ask me something.

> Q The effect, you've told us before, is often in that first—the acute effects, the first half hour but can be up to two, four hours, sometimes longer.
>
> A That's right.
>
> Q And if I'm understanding you correctly, it's primarily an effect on psychomotor skills, at least insofar as when we're looking at the driving end of it.
>
> A Well, you know it's very hard to say, isn't it? Because if you find out the individual's psychomotor skills are affected you say, "Well, it's his psychomotor skills." But it turns out his psychomotor skills are impaired because his perception's impaired. He doesn't respond quickly because he doesn't see it quickly.

So Dr. Klanoff, who's also a Canadian

investigator, has published a number of studies in which he's talked about the fact it's very hard to know what's going on. You say it's reflex time, but it may be perception. It may be motor—muscle relaxation. It may be impaired memory. All of those things can influence what looks like a simple outcome of psychomotor impairment.

Q Assume we have a person who has recently smoked marihuana and is under the influence of it. Could a policeman on observing that person and having them perform a number of tests such as walking a straight line or heel—are you familiar with the basic—

A The roadside—

Q -- roadside type—

A -- sobriety tests—

Q -- tests?

A Yeah.

Q You know, finger-to-nose, so on.

A Yeah.

Q Would a policeman observing a person who's intoxicated going through those tests be able to determine anything?

A Sometimes he can, sometimes he cannot. Has to do with of course the level of intoxication. But yes, it is quite clear now that police will pull someone over for reckless driving, do the roadside sobriety test and see that it's not—that the individual cannot perform, and then collect the breathalyzer and find out that there's no alcohol here. So then, under certain circumstances in certain jurisdictions the policeman has every right to ask for a blood specimen or a urine specimen and if the individual refuses to give it he may then have the right to put him under notice as driving while intoxicated anyway.

MR. DOHM: I do think he's gone far beyond the psychopharmacology and into law, probably that of another country.

MR. CONROY:

Q I'm curious about the effects, the psychomotor effects and the ability to detect them.

A Yeah. To answer your question in an acceptable way, it's not always true but at significant levels of intoxication yes, people under the influence of marihuana will fail a roadside sobriety test.

Q Is there some—

A Not always.

Q Is there some—I read recently in a newspaper—I don't know how accurate it is, I don't usually believe anything I read in the newspapers at least not about this topic or criminal justice topics—that there's some machine that they've now developed that—in some countries, for determining levels of marihuana use. Are you aware of anything like that?

A Well, people have tried a variety of things, and I guess this is germane. People have looked at saliva levels to try to measure T.H.C. and T.H.C. metabolites, and of course we're quite good at measuring them in the blood and in the urine. The dilemma here is that even if we measure T.H.C. in the blood the answer is not like the answer for alcohol. Alcohol is quite rapidly distributed throughout the body and the amount that's in the blood has a strong correlation with motor dysfunction. Not absolute. Even though we say we'll take .1 as a per se level, .1 which is a hundred milligrams of alcohol per one hundred c.c.'s of blood, we'll say that's a per se level of intoxication. It's a statistically good level. Most people at .1 will be impaired. Some will not be. Some will be quite impaired at .05. Most will not be. But we've decided that the correlation is strong enough and the cost to the culture is strong enough that we're going to say, "No argument. You're intoxicated."

You cannot do it with blood levels of T.H.C.

You cannot do it. And I don't think we'll ever be able to do it. We're not going to be able to have a blood T.H.C. measurement that will tell us whether the influence was under—whether the individual was under the influence of marihuana because the range is so high. There are people with a hundred nanograms of T.H.C. per millilitre who are not at all high. There are people with ten nanograms who cannot walk. So we—and that's because of the compartmentalization. It's not the amount that's in the blood, it's the amount that's in the brain. And the amount that goes into the brain is delayed and then it comes out quickly and looking at the blood levels we don't know where we are.

Usually—people have done studies in which

if the blood level is below four or three or two, the assumption is the individual is not likely to be under the influence. And that's germane in something else I'm going to talk about.

But we don't have a per se blood level and I

don't believe we shall ever have a per se blood level, as we do for alcohol. It's going to have to be something else.

Q Have you ever heard of the Barringer Ionizer Scan that they use in prisons to detect contact with various drugs?

A Well, Barringer is now the name of the company—was once called Syva, S-y-v-a. And Syva was the developer of the immunoassay, the emit test which is the most common test of cannabinoid metabolite in the urine. And when I saw that note I assume that's what it referred to. Barringer now markets the emit test, which is the most common assay for marihuana metabolite and it's quite sensitive. It will read out and will tell you this individual's been exposed to marihuana. The dilemma is you don't know whether he took it an hour ago or five days ago. That's the problem.

Q Or you could—you could be in a bar where—or a rock concert when people have been smoking marihuana.

A You could, if it were read down very low. It's usually not read that low. False positives due to exposure, passive inhalation, do occur but they're really quite rare and it depends on where you set your cutoff level. In the workplace I think passive inhalation is a rare cause of a positive, although I'll hold out that it happens sometimes.

Q I understand the most recent studies on influence of marihuana on driving were H.W.J. Robe, is that right?

A Robe.

Q Robe?

A Robe.

Q And that was funded by the U.S. again—

A Yes.

Q -- but conducted in the Netherlands.

A Correct. The National Institute of Highway and Traffic Safety decided to give to Dr. Robe a significant grant to study the impact of marihuana on driving behaviours under a number of circumstances. There's very good reason for doing that. Dr. Robe and his colleagues at Maastricht, the Institute for Human Pharmacology, have been very involved in doing driving studies. They for instance have done driving studies on the impact of Valium, the impact of Benadryl, the impact of

imipromine[phonetic], on the impact of a lot of prescription drugs and over the counter drugs.

So to try to draw this quickly to a

- to a conclusion, Dr. Robe did the most extensive and most comprehensive and to me, and I think to most people, the most believable study of the impact of marihuana on driving ability. And I'll give you his conclusion. "Of the many psychoactive drugs licit and illicit that are available and used by people who subsequently drive, marihuana may well be the least harmful."

Q And so that includes comparisons to things like Valium.

A Furthermore, he had a very quantifiable measure. The most sensitive indicator of drug effect on an individual's driving is his ability to hold himself steadily in the middle of the road. And what Robe and his group have done—others have as well—is to generate something called the standard deviation—standard measure of lateral deviation, standard—lateral deviation. So if I'm driving and I'm very good I'll pretty much stay in the middle of the road with the white line on the left side and the edge of the road on the right side. If I'm under the influence of a drug I'll deviate a little bit. Now you can measure the deviation when you cannot see that the individual's impaired. When there's no other measure of impairment, observation, braking time, etcetera, etcetera, but you can get an impact on lateral deviation with low doses of a drug. And in fact Valium, Benadryl, imipromine or tofranil[phonetic] had a greater impact on lateral deviation than did these doses of marihuana. So Valium has a greater impact on driving than marihuana according to Robe's studies, at least with this measure, the ability to hold the car in the middle of the road, which is the most sensitive measure. He used others. That's the most sensitive one.

Now in terms of dosage, which is a critical

issue, Dr. Robe gave three doses of marihuana. And he determined them in part by allowing people to smoke and telling him what they liked as their high dose. And it wasn't much difference than had been done in other studies. It was, in the way pharmacologists like to calculate the dose, by weight, it was either one hundred, two hundred or three hundred micrograms of Delta 9 T.H.C. per kilogram of body weight. That's the equivalent of two to three milligrams in most people and four to five in big people. Six to seven in real big people.

Now the important thing is that in all three

doses—incidentally, Dr. Moskowitz, whom I mentioned to you before, has found this impact on selective and divided attention at doses one half Robe's lowest, at fifty micrograms per kilogram. So Robe's doses were clearly enough to affect the kind of measures that Moskowitz and others have worried about as important in driving behaviours.

Now Robe did—he actually did three kinds

of studies. He gave people the hundred or two hundred or three hundred and compared it to placebo smoke, and then he did a number of studies, such as the memory, the ability to estimate the passage of time, measured their heart rate, looked at their red eyes, a variety of other things that are generally accepted as evidence of a marihuana high, including the individual's rating was, "Am I high on marihuana?" So he did those to see that these were effective doses.

Then he evaluated their driving performance

in two separate trials, the first on a highway closed to traffic. And I actually saw a videotape of Robe's operation in which the Maastricht police have cooperated with them to give them a real section of road, but it's easily closed to traffic with a bypass for the regular traffic. And people drove on that road which was controlled access, and the vehicle ahead of them was instructed on how to track and how to slow and how to speed ahead.

The second was on an occupied highway in which

- this was a similar stretch of highway but there were real cars there. The individual really had to drive.

And the third, using only the lowest dose of

a hundred micrograms per kilogram in busy city driving in Maastricht, which is a small to medium Dutch town.

Okay. In the first two trials on the

closed highway and on the occupied highway, marihuana's effect on driving performance is found to be insignificant on nearly every measure. Marihuana did impair the driver's ability to maintain a steady lateral position and higher doses produced greater impairment. That is, they would weave more. However, even with the highest dose marihuana's effect was relatively minor, similar to that observed in drivers using legal medications in therapeutic doses and similar to that of drivers with blood alcohol concentrations well below intoxicated levels. In fact, in the urban driving trial, which compared marihuana's influence on driving to alcohol at a .04 percent blood level, which is two beers in an average weight person, alcohol produced significant reductions in driving ability while marihuana produced none.

So Robe did everything that could have

been expected of him. He gave the marihuana, assessed that it had an impact, did a series of physiological and other studies. He then put people on the road in three different settings, a highway with controlled access, and it always included following behind a car at a certain distance and then responding to that car as the driver took his foot off the accelerator and let the car slow and to see how the marihuana driver would behave. That was a standard part of the test.

And then on the occupied highway and the city

driving the drivers were evaluated by two means, one in which someone sitting in the car was given a checklist and was supposed to every few seconds check and see if he was maintaining distance where he was, was his speed okay, etcetera, etcetera. And then another assessor in the same car did a global evaluation, was this skilled driving at this level or skilled driving at a different level. And basically, marihuana produced no

impact on these global driving evaluative scores. Produced an impact on lateral deviation. And the marihuana drivers tended to stay further behind the car they were to track than they were supposed to. In other words they slowed down because of the apparent cautionary influence. The marihuana drivers told in the city driving test that they were worried, they were scared because they felt high on marihuana and a few of them said, "I don't want to drive." But they were convinced to do so. And they tended to be very careful because of this. While the alcohol drivers at .04 percent, although they had significant impairment, felt just fine.

So Robe's studies are I think critically

important.

There has been some criticism of the only

urban driving trial was at a low dose and some people have said it was too low a dose to have an impact. It was a dose sufficient to produce marihuana effect in almost every sphere. A hundred micrograms per kilogram is a standard social dose of marihuana. Everybody gets high on it. So I don't believe the criticisms are apt, but you will hear those criticisms, that the Robe study should be discounted because the lowest dose was too low.

The Robe study is really—well, in terms

of those of us who care about marihuana and its impact on the culture and its impact on human performance, the Robe study is almost revolutionary. It has to me answered every question that people have asked about marihuana and driving. Maybe not every, but an awful lot of them.

Now let me in two minutes tell you

the—the final piece of information. So we've got psychological, psychomotor tests, we've got driving simulators, we've got on-road performance, we've got Robe's study which in a certain sense combines almost everything. And all of those say, "Well gee, I don't see much evidence that marihuana is an important contributor to highway problems." Then in our last—almost—not our last, but on 16-4, "More compelling evidence of marihuana's minimal effect on driving comes from epidemiologic surveys of drivers involved in fatal highway accidents." And I want to particularly comment on that, because in Dr. Kalant's commentary in Haman[phonetic], he discussed the fact that there were some studies which seemed to indicate that marihuana positives

were over-represented in serious accidents, and some early studies may indeed have said that. But there's been significant information generated in the past few years. "Compelling evidence of marihuana's minimal effect on driving comes from epidemiologic surveys of drivers involved in fatal highway accidents." That is in the United States, Canada and Australia. "And found T.H.C. in the blood of from three to eleven percent of fatally injured drivers. However, in the majority, seventy to ninety percent of these cases, alcohol was detected as well and probably contributed to the outcome. To evaluate marihuana's specific contribution to accidents some researchers have rated the culpability of drivers who test positive for marihuana only." That is, someone using the road report, the police report, the characteristics of the accident have said, "Now was this driver responsible for this accident? Did he bear responsibility to a large degree or to a small degree? Is there any evidence that maybe he was not guilty of anything at all, just had the bad fortune to be ploughed into by another driver?"

And one of the culpability studies is related

to an important Canadian study of T.H.C. in the blood of fatally injured drivers. That's the study by Sinbora[phonetic] which is referred to here.

So in the—"In rating the culpability of drivers

who test positive for marihuana only one study found a higher culpability for marihuana positive drivers than drug-free drivers. That means that marihuana appeared to be at fault. But it relied on a very small sample. Three other studies found not only that marihuana positive drivers were less culpable than alcohol positive drivers, but were also less culpable than drug-free drivers. That is, fewer drivers in the marihuana positive group than in the drug-free group were judged to be responsible for the accidents. The author of one of these studies suggests that either the cannabis actually increases driving ability or that drivers taking cannabis overcompensate for loss of driving skills." And obviously I quoted that because I believe that's what happens. The cannabis drivers overcompensate, they slow down, they won't pass, they won't speed, they're very careful.

I don't believe that marihuana improves driving

ability and I think no one should drive having smoked marihuana in the last few hours. No one should drive. However, there is no acceptable evidence, no convincing evidence to me that marihuana is an important contributor to vehicular accidents or vehicular fatalities on the roads of North America.

MR. CONROY: I note the time, Your Honour. Will we

be going 'til four or later today? There's the ceremonies for the judges that are being called. I don't—

THE COURT: I'll leave that to counsel. I'm quite

prepared to sit through the regular court day, knowing that we have an out-of-town witness.

MR. CONROY: We've got roughly four more to go.

A Do we really?

MR. CONROY: Well, we're up to seventeen.

A But there is no nineteen.

MR. CONROY: That's right, there's no nineteen, so we've

got three to go.

THE COURT: Is Dr. Morgan prepared to come back tomorrow?

MR. CONROY: Oh, yes.

A Oh yes, ma'am.

MR. CONROY: He's here until—

A I'm—my plane is not scheduled to leave until Thursday.

MR. CONROY: Thursday night, so.

A Thursday—I can't remember.

MR. CONROY: Thursday evening.

THE COURT: I'm not sure you should have told us that.

A Well—

MR. CONROY: Thursday evening.

A -- I felt I had to.

MR. CONROY: But he's available in the morning, so

let's take the break, get as much as we can—

THE COURT: Whether counsel wish to attend the

ceremony or not, I'll leave that up to you. It appears that Dr. Morgan will be here tomorrow anyway. I don't know if you want the extra half hour today or if you wish to attend—

MR. CONROY: Well, I'd like—I'd like us to do a little

bit more maybe until four and then—

THE COURT: We'll come back at least until—

MR. CONROY: Four.

THE COURT: -- five to four.

MR. CONROY: I'll speak with my friend at the break and

see what his wishes are.

THE COURT: All right.

(WITNESS ASIDE)

(PROCEEDINGS ADJOURNED)

(PROCEEDINGS RECONVENED)

MR. CONROY: We would like to break just before

four, if that's possible.

THE COURT: Fine.

JOHN PAUL MORGAN, recalled, testifies as follows:

EXAMINATION IN CHIEF BY MR. CONROY continuing:

Q The—just on the driving issue, before we move on to the next one, I found my newspaper clipping that I was talking about in terms of a device. And it refers to a cannabis intoxication factor, c.i.f. blood test. And according to the article—I'll just give you very, very quickly—it's apparently being developed in Germany. Police are testing this method with driver levels of intoxication from hashish or marihuana. And according to the article the test promises to show anyone with a c.i.f. of ten is about as incapable of safe driving as someone with a blood alcohol level of 0.11. Do you know anything about that?

A I do.

MR. DOHM: With respect, Your Honour, I'd submit

that my learned friend is cross examining his witness. He asked him a question identical in substance to that before the break and the witness said he wasn't aware of a device that would measure the person's level of intoxication, and he went further and said that he didn't think that one would ever be created.

MR. CONROY: Is this a device or is it just a factor

that people look at?

THE COURT: I don't—I'm going to allow the question.

I think counsel's entitled to ask about—if he

- if this witness has any knowledge of this particular c.i.f. or factor or blood test.

A Thanks for the opportunity. Because it—it illustrates a couple points and clears things up. The factor that's being referred to there has—I think was chiefly developed in the United States and again, it's not a factor of intoxication. It is a means by assessing two cannabinoids in the blood of estimating within fairly narrow limits when the material was consumed. What it simply does is to measure the Delta-9 T.H.C. in the blood, which gives you a certain reading somewhere between one and a hundred, but it also gives you the amount of T.H.C. carboxylic acid, which is the chief metabolite, the weightiest metabolite of marihuana, of T.H.C.

So Dr. Kohn and Dr. Hustus[phonetic]

have published two papers now in which they've given marihuana of a known amount to individuals with no T.H.C. in their bloodstream and then measured these two factors. And by a ratio of the metabolite to the parent compound they have been able to do pretty well at saying this was consumed within an hour, this was consumed within five hours. So the newspaper article is incorrect. It doesn't tell you whether the individual is intoxicated or not, and my answer is that I don't know that we'll ever be able to do that (indiscernible). However, this may be an important estimate of when the material was consumed. And so for all I know a state legislature might then say, "Well, if it was consumed within the last hour we're going to presume the individual was." But that's all speculative. That's what's going on with that test.

MR. CONROY:

Q So it measures how recent or how long ago, not how much.

A Correct.

Q Okay. All right, let's go to chapter seventeen then, "Marihuana and Hard Drugs", which is also the gateway theory topic, as I understand it.

A Yes.

Q The myth, "Marihuana is a gateway drug. Even if marihuana itself causes minimal harm it is a dangerous substance because it leads to the use of harder drugs such as heroin, L.S.D. and cocaine." What can you tell us about that?

A Earlier on in my testimony I talked about

medi-toxicity, and this is of course a very important one, that after you've argued with people about the harms of marihuana and whether you've convinced them of the minimal harms of the substance, then the idea of the gateway is trotted out, that somehow the use of marihuana is associated with a cause, some kind of factor that will lead to the use of other drugs. We're fond of pointing out that in the sixties all discussions of gateway had to do with progression from marihuana to heroin use. No one has those discussions any longer because we now have plenty of evidence that almost no marihuana smokers progress to heroin use and the level of heroin use has remained quite low and fairly steady in this culture, with some ups and downs.

But then the gateway progress—the gateway

then became if you use marihuana you'll use L.S.D. Well, that has not worked out. And in the States now

- I don't know how it is in Canada—the gateway is always cocaine; if you use marihuana you'll progress to cocaine.

Now what—we have said two or three things

that we think the evidence merits. There is no pharmacological basis for the gateway theory. There is no impact of marihuana on the brain which leads individuals to need, want, search for, suck up cocaine or any other drugs. The gateway is a description of a very logical statistical observation which goes as follows. In a culture there are a group of drugs, intoxicants available. If an individual has used an intoxicant of very low prevalence, such as cocaine or heroin, then he is very likely to have used an intoxicant of high prevalence. Therefore, if you examine users of heroin, users of cocaine, you will almost always find that they have heretofore used marihuana, tobacco and alcohol. Those are the intoxicants of high prevalence and their use almost always precedes the use of a low prevalence intoxicant such as cocaine.

Now—but to describe marihuana as a

gateway to cocaine is not supportable by any evidence. In other words, true, most users of cocaine have used marihuana. But only a minority of marihuana users ever use cocaine. The last number, the high school senior survey in the United States was that of all of the high school seniors who had experimented with marihuana only fourteen percent had tried cocaine. So, for eighty-six

percent of high school seniors marihuana is not a gateway drug, it's a terminus drug.

Those data hold up fairly well into adult

life, although they drop somewhat, logically enough. And I believe of individuals who are twenty-nine or thirty who have used marihuana about two-thirds of them have never used another illegal drug, in other words, sixty-seven percent. And that tends to hold fairly well throughout the culture.

So marihuana cannot be described as a gateway

drug if only a minority of individuals who use it go on to other drugs.

And I'll say two other quick things.

In the United States we have been presented with a sound byte which goes as follows, "Users of marihuana are eighty-five times more likely to use cocaine than non-users of marihuana." This number has come from something called the Centre for Addiction and Substance Abuse affiliated with Columbia and headed by a man who was once in the drug czar's office and headed by another man who was once Secretary of Health, Education and Welfare in the United States in the Carter administration, a man named Joseph Calofono[phonetic]. So let me state it for you again. "Users of

marihuana are eighty-five times more likely to have used cocaine than those who have not used marihuana." Now this is not at all a statement of the gateway. What it is is the statement of how unusual it is for someone to have used cocaine to have never used marihuana before.

In other words, if you take—this actually was

done a year before the fourteen percent so it was sixteen percent. So you put it in your numerator sixteen, that is the percentage of marihuana users who have used cocaine, and then your denominator you put the percentage of cocaine users who have never used marihuana, which happens to be .02. And then you divide sixteen by .02 and you get eighty-five. Clear? It's—maybe I can make it clearer with one

other thing that Professor Zimmer and I have used in this chapter. Anytime there are two associated events of high prevalence and low prevalence you'll see this association. For instance, only a few Americans ride motorcycles. Of those Americans who ride motorcycles, almost all of them have ridden bicycles before. Yet, of those individuals who ride bicycles only a small percentage of them go on to use—to ride motorcycles. Is therefore bicycling a gateway to motorcycling, or does bicycling cause motorcycling? There is no gateway theory at all and there is no evidence that the use of marihuana leads in any causal, pharmacological factor to the use of other drugs.

And one last point, we have detailed in here

a lot about the shifting prevalence of drugs over time. Well, marihuana use declined in the United States from 1979 'til 1991. For the first part of that cocaine use went up. And while marihuana use declined for a long period of time L.S.D. stayed about the same.

The most interesting statistic that we were

able to generate was that—you heard me say before that the prevalence of cocaine experimentation in individuals who had used marihuana who were high school seniors, was fourteen percent. That's the lowest it has been in a decade. In 1986 the percentage of marihuana users who had experimented with cocaine was thirty-three percent. That's when cocaine was on its upsurge in the United States. So it's fallen every year since that time. This gateway factor has fallen every year since 1986. 1986, thirty-three percent of marihuana smokers who were high school seniors had tried cocaine. In 1995 fourteen percent of high school seniors who had used marihuana had used cocaine. There is no gateway. There is no gateway. There is no stepping stone. There is no theory here at all.

Q All right. Chapter—I should just add, figure two in chapter seventeen illustrates that to some extent, doesn't it?

A Yeah. Actually figure one may illustrate it even better. Figure one illustrates what I just said. Proportion of marihuana users ever trying cocaine among high school seniors. You'll see the peak was in 1986 at thirty-three, and you'll see it's fallen every year since that time.

Q Figure one—sorry, I've got—

A That's this one.

Q -- is—

A The—there, you've got it.

Q -- the data for number seventeen. I had it out of order.

A Okay.

Q Sorry. Figure one does what?

A It just shows what I just talked about. In

1986 --

Q All right.

A -- thirty-three percent of high school seniors who had tried marihuana had tried cocaine and it's fallen every

year to the low of fourteen percent in 1995. There's no relationship between cocaine and marihuana use.

Q All right. Number eighteen is "Punishment for Marihuana". Now this—the myth stated here is, "Marihuana offences are not severely punished. Few marihuana law violators are arrested and hardly anyone goes to prison. This lenient treatment is responsible for marihuana's continued availability and use."

A I wanted—we wanted to do this because these statements are commonly made in the United States. "Why are you all so concerned with marihuana? Hardly anybody's punished for marihuana use anyway. Marihuana's openly smoked in New York City and nobody ever gets arrested and if they do it's a misdemeanour," blah, blah, blah.

Well, our experience has told us that this

is not true, that marihuana enforcement is very important in the United States. And so we simply engaged in gathering data to see what marihuana law enforcement in the United States is like. I have no idea what it's like in Canada, but I can tell you that the number of people arrested for marihuana offences in the United States reached an all-time high in 1995. That year there were more than a half million marihuana arrests, eighty-six percent of which were—were for marihuana possession. Tens of thousands of people are now in prison for marihuana offences and an even greater number are punished with probations, fines and civil sanctions such as having their property seized, their drivers' licences revoked and their employment terminated.

Q And this is set out in your table—

MR. DOHM: Excuse me, Your Honour, before—before Mr.

Conroy asks another question, I'm trying to figure out whether this is anywhere near the Doctor's qualifications either as a medical doctor or as a psychopharmacologist.

MR. CONROY:

Q I assume the answer to that question would be that it doesn't fall within your—your expertise as a medical doctor or as a pharmacologist. But why did you gather this data in your manuscript? How do you—can you relate it to your expertise as a pharmacologist or doctor?

A Well, I don't know. I gathered the data because of the frequent statements that marihuana punishment is very minimal in the United States.

Q So this—this data is gathered from—from what sources?

A You'll see in the reference, that's the reports of New York State and Texas and Michigan on the percentage of individuals charged with marihuana offences who were subsequently imprisoned, a report from the federal government of the United States, the Bureau of Prisons and the Bureau of Criminal Justice, the number of people arrested and the number of people sentenced.

We did a number of things to try to gather

this information and to portray the fact that nothing has changed from the opinion of the Schaeffer Committee, which is that the greatest hazard facing a user of marihuana is not toxic, but the likelihood that he'll be arrested and prosecuted.

Q Do you—I can't remember if you were in the courtroom when Dr. Connolly—I know you were here for when he testified in chief and I think that's when he gave this evidence. He related punishment and so on in—from a doctor or a medical perspective. Do you do that?

A Well, I don't often do it. In terms of assessing the harm done to an individual, I think oftentimes the discovery that he's a marihuana user by a urine test or by a criminal justice investigation will bring him more sorrow, harm, difficulty than any biomedical harms that are likely to come from marihuana. So then I guess I am competent to make that judgment, that I know what the biomedical harms are. It's been my life to study them. And they are minimal compared to the life disruption that occurs because a marihuana user falls into the hands of his employer, his government, his criminal justice supervisor because his marihuana use is detected. So I guess I can make some kind of judgment about harm. Whether we specifically consider that incorporated by my expertise in toxicology I'm not entirely sure. I'll let you all decide.

MR. CONROY: Well, perhaps we can consider that

over the evening. It's a few minutes to four. And if it's not within the expertise we'll leave it and go on to the next.

THE COURT: All right, we'll adjourn then until tomorrow

morning at nine thirty.

(WITNESS ASIDE)

(PROCEEDINGS ADJOURNED TO 29 JANUARY 1997 AT 9:30 A.M.)