The Myth of Drug-Induced Addiction

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Most Canadians believe that certain drugs cause catastrophic addictions in people who use them. This conventional belief is reflected in such familiar phrases as "crack cocaine is instantly addictive" or "heroin is so good, don't even try it once". It is also implied in the professional literature which routinely describes certain drugs as "addictive", "dependency producing", or "habit forming". The belief that drugs can induce addiction has shaped drug policy for more than a century.

However, the only actual evidence for the belief in drug-induced addiction comes 1) from the testimonials of some addicted people who believe that exposure to a drug caused them to "lose control" and 2) from some highly technical research on laboratory animals. These bits of evidence have been embellished in the news media to the point where the belief in drug-induced addiction has acquired the status of an obvious truth that requires no further testing. But the widespread acceptance of this belief is a better demonstration of the power of repetition than of the influence of empirical research, because the great bulk of empirical evidence runs against it. Belief in drug-induced addiction may have deep cultural roots as well, since it is a pharmacological version of the belief in "demon possession" that has entranced western culture for centuries.

This is more than an academic issue. Canadian policy decisions frequently are constrained by the public's strongly-held belief in drug-induced addiction. Draconian laws, sentencing, and even police violence have been justified by the need to keep addicting drugs out of the hands of the nation's youth at all costs. As well, it is almost impossible to experiment with medical administration of heroin or cocaine to addicts for fear that the medical profession would be seen as dispensing an addicting drug that could find its way to the public. Introduction of methadone maintenance into most parts of Canada was delayed for years, largely on the basis of the argument that, since methadone is pharmacologically similar to heroin, diversion of methadone from addicts to their neighbours would cause a new explosion of addiction (Alexander, 1990, chap. 8). No form of drug legalization can be credible when drugs provoke fear in the public mind of the sort that demons did in olden days.

This article briefly reviews the empirical evidence surrounding the belief that heroin and cocaine cause addiction. It does not evaluate the possibility that drugs other than heroin or cocaine induce addiction, although the claim has been made for many others, beginning with alcohol in the 19th century and marijuana in the 1920s. However, heroin and cocaine are currently regarded as the most addictive of drugs. If they do not cause addiction it would seem probable that no drug does. If the myth of drug-induced addiction can be dispelled, then Canadian drug policy can be formulated on a different basis, with fewer constraints

I have learned that raising this sensitive issue inevitably provokes misunderstanding. I entreat the Senators not to mistake my purpose. I have no wish to deny that some heroin and cocaine users become addicted, often with horrible consequences. I do not deny that some people sincerely believe that a few doses of a drug has robbed them of their self-control and deprived them of an otherwise normal existence. I do not deny that heroin and cocaine, and drugs in general, should be regulated in the public interest. However, I do deny that drugs cause addiction and I deny the utility of Draconian drug laws that have been based, in part, on the belief that they do.

Drug-induced addiction is a myth that has fanned the flames of the "War on Drugs". Now that this "War" has been discredited, we can discard its myths (along with its dysfunctional policies) in order to clear our vision for a realistic examination the terrible problems that the War on Drugs was intended to combat. There are many other harmful myths besides the myth of drug-induced addiciton (Alexander, 1990), but this submission addresses it alone.

Structure of the

Argument

The centerpiece of this submission is a review the empirical data. Some of the data come from my own thirty years of research on addiction in Vancouver, but the majority come from the publications of other professional researchers in the fields of epidemiology, psychopharmacology, neurobiology, psychotherapy, and history. Many published articles and books that use this same literature to explode the myth of drug-induced addiction on empirical grounds are also available, e.g., Trebach, (1982; 1993); Szasz (1985); Erickson & Alexander (1989); Alexander (1990; 1994), Davies (1992); Morgan & Zimmer (1997a, b); Peele & DeGrandpre (1998).

Analysing the vast and complex literature that relates to this topic becomes simpler if the general belief that heroin and cocaine cause addiction is resolved into two more specific claims, and each is evaluated separately. The two claims are:

> **Claim A: All or most people who use heroin or cocaine beyond a certain minimum amount become addicted.**
>
> **Claim B: No matter what proportion of the users of heroin and cocaine become addicted, their addiction is caused by exposure to the drug**
>
> .

The two claims are rarely stated this explicitly. Usually, they are either assumed, stated in a vague way, or combined. However, every professor who teaches a course in drug addiction knows that the majority of students firmly believe both of them at the beginning of the semester. Moreover, a careful reader will be able to unearth these two claims throughout both the popular and professional literature on addiction from the 19th century until the present. Here is a single example:

> As with any other measure of health, one's liability to chemical addiction lies on a continuum, with none of us entirely invulnerable...Addictive disease occurs when a person somehow exceeds his/her invisible and unknown threshold level on the continuum of vulnerability and triggers a certain biochemical response in the brain through repeated drug use. (Washton, 1989, p. 57)

The two claims both show through if this short quote from a prominent American researcher is carefully examined. Washton states that all people will become addicted to cocaine if they exceed a certain threshold level of use (Claim A), and that when addiction does occur it is because of the pharmacological effects of exposure to the drug (Claim B).

These two claims are fundamental to the belief in drug-induced addiction. If either one can be verified, it would comprise powerful support for the general belief. However, if neither can be verified, the general belief would lose any luster of empirical support that it may have had. By separately considering each claim as it applies to both heroin and to cocaine, I will show that existing evidence conclusively refutes the first and fails either to prove or disprove the second. Thus, the widespread and heartfelt belief that heroin and cocaine cause addiction has something other than an empirical basis. This submission also discusses some other possible bases for support of this belief. In my oral presentation to the Senators, I will discuss alternative explanations for the spread of addiction that are more plausible in the light of history (Alexander, 2000).

Claim A: Review of the Evidence

Claim A is usually asserted less strongly now than it has been in the past, when claims of "instant addiction" were often made for both heroin and cocaine and, earlier, for alcohol, marijuana, and numerous other drugs. More cautious contemporary statements of Claim A state that addiction only occurs after several exposures to the drug, although the minimum amount required to produce addiction is left unspecified. As well, it is now sometimes added that certain outside factors may abort the progression toward addiction, before the threshold has been crossed. For example, an article in *Science* magazine, while reasserting the belief that "cocaine causes a neurophysiological addiction", acknowledges that not all those who experiment with cocaine become addicted. It explains this fact by suggesting that those who do not become addicted either 1) do not have the normal euphoric reaction to cocaine, 2) cannot find or afford additional supplies of cocaine, or 3) see that they are becoming addicted early on and "are able to cease use" (Gawin 1991, p. 1580-1581). Thus, this cautious form of claim A would predict that repeated use of cocaine would cause addiction in a physiologically normal person unless the person were unable to obtain the drug or stopped using out of fear of addiction before crossing the threshold of addiction. Continued use would inevitably cause addiction. Although Gawin's statement is more cautious than some past rhetoric, it preserves the essence of claim A.

Heroin

. Testing Claim A is logically straightforward; it predicts that when people are sufficiently exposed to drugs they will all become addicted. Of course, some people do become severely addicted after a few exposures to heroin and cocaine. However, controlled observations contradict both the strong and the cautious form of Claim A for heroin, morphine, or any opiate drugs. The large majority of people exposed to these drugs, even many times, do not become addicted.

Clinical Research. Conventional wisdom notwithstanding, administering large doses of heroin and other opiate drugs over long periods of time to medical patients does not cause addiction. Certainly, there were many 19th century case reports from physicians stating that otherwise normal patients became addicted due to overprescription of heroin and other opiates, but systematic historical research has raised doubts about these case studies. Although the use of opiates in the U.S. and England during the 19th century was enormously greater than it is now, both through physician-prescribed injections and ubiquitous patent medicines which were used as tonics and for recreational purposes, the incidence of dependence and addiction never reached 1% of the population and was declining at the end of the century before the restrictive laws were passed (Brecher, 1972; Ledain, 1973; Courtwright, 1982).

In the U.K., heroin was widely used as a medication for cough, diarrhea, chronic pain from the beginning of the 20th century to the present. In the year 1972, for example, British physicians prescribed 29 kilograms of heroin—millions of doses—to medical patients. A major portion of this heroin is sold as an ingredient in cough syrups which are readily available. Careful examination of the British statistics on iatrogenic addiction ten years later revealed "there is a virtual absence of addicts created by this singular medical practice" (Trebach, 1982: p. 83). Heroin remains a staple drug in British medical practice along with morphine and other opiates. Fears of addiction amongst British physicians are minimal (White, Hoskin, Hanks, & Bliss, 1991).

About 20 years ago, an American research team began experimenting with a Canadian invention, a bedside self-medication machine programmed to deliver about 1 mg of morphine intravenously to patients who pressed a hand button. The machine limited infusions to one every six minutes. In one early study, fifty patients were kept on the regimen between one and six days. The self-administered doses were considerably less than the maximum the machine would allow. Rather than increasing as patients continued the regimen, the doses progressively declined (Bennett, et al., 1982).

From this small beginning this machine, now widely known as the "patient controlled analgesia" or PCA machine, has come into general use in hospitals. In spite of the misgivings of many hospital workers who believed that PCA machines would cause addiction in many patients, iatrogenic addiction has been extremely rare, even among those patients that were allowed larger doses over several days (Schug, Merry and Acland, 1991). The only patients deemed unsuitable for PCA are those with concurrent medical conditions that could be exacerbated by analgesics and, in some institutions, patients with a history of addiction to drugs or alcohol.

Epidemiological research**.** It can be argued that the clinical research does not provide support for claim A because of the special frame of mind of medical patients and the special circumstances provided by hospitals (Lindesmith, 1968). It this were so, evidence supporting claim A would be expected from systematic surveys of heroin users who are not medical patients. A number of careful studies have described casual or regular non-addicted users of heroin who have not become addicted in spite of years of use (Blackwell, 1982; Zinberg, 1984).

Here it is important to consider the meaning of the term "addiction". Some (not all) of the non-addicted users of heroin studied by Blackwell and by Zinberg were *regular* users of heroin. However, they did not feel out of control, the heroin habit did not consume their lives, they did not steal to obtain it, and they were not criminalized. Therefore, by all normal definitions of "addiction" and equivalent terms, these people were not addicted (for example, see definitions in Jaffe (1990) or the most recent *Diagnostic and Statistical Manual of the American Psychiatric Association*). If the term "addiction" is applied to mere occasional use or innocuous regular use the term becomes trivial--most people regularly and stubbornly use things that carry some substantial risk of harmful side effects, like automobiles, skis, computers, and birth control pills.

The non-addicted users described by Zinberg, including those who used regularly, were no more likely to escalate their use than they were to reduce it. Zinberg studied a group of "controlled users" of opiates 12-24 months after an initial interview. He was able to re-interview 60% of the original group. Of these, 49% were using drugs in the same way as at the first interview, 27% "had reduced use to levels below those required for them to be considered controlled users", and 13% were using more opiates than at the first interview (Zinberg, 1984: 71). There is no doubt that some long-time users of heroin and other opiate drugs do escalate their use to true addiction, but the frequency of this is far less than claim A implies.

Cocaine. In the case of cocaine, as with heroin, the evidence is consistently against both the strong and the cautious form of Claim A. In the case of cocaine less clinical research is available, since cocaine has less application in modern medicine, but there is more epidemiological research thanks to the great surge of recreational cocaine use in North America in the 1980s. I will discuss cocaine in general first and conclude by discussing the special case of "crack" cocaine.

Contemporary Clinical Research. Cocaine is administered in medical practice in the United States and Canada primarily as a local anaesthetic, although there are a variety of other uses described in the contemporary medical literature as well. Although reports of iatrogenic addiction to cocaine were common in the 19th century and early 20th century (Erickson, Adlaf, Murray, and Smart, 1987), none of the contemporary clinical reports has produced any indications of iatrogenic addiction.

Probably the most common contemporary medical use of cocaine is as a local anaesthetic in nasal surgery (Haddad, 1983; Moore et al., 1986; Gordon, 1987). Nasal surgeons apply cocaine to exactly the same area—the nasal mucosa—that cocaine "snorters" do. Although the textbook maximum doses are around 200mg of cocaine hydrochloride, the dosages that have actually been used in nasal surgery (Johns & Henderson, 1977) are comparable or higher than those that are actually taken by Canadian recreational users (Erickson et al., 1987). Moreover, the peak blood levels of cocaine following the medical doses are comparable to those found following administration of doses that produce a "high" in experienced users (Javaid et al., 1978). All anaesthetics are dangerous, so patients that receive cocaine in this manner closely monitored for side effects. The levels of side effects compare favourably with other anesthetics—this is part of the reason that cocaine is the anesthetic of choice for many doctors. A survey of plastic surgeons revealed five deaths and 34

severe but non-fatal reactions in 108,032 medical applications of cocaine (Feenan and Mancusi-Ungaro, 1976)—but not a single case of iatrogenic addiction has been reported.

Cocaine also appears to be a valuable treatment for older people who suffer from chronic rheumatoid arthritis. A small group of doctors in California in the 1970s reported good success in relieving the pain and depression of this disease with "Esterene" which is simply "free-base" cocaine prepared for nasal application. In this form, cocaine is released slowly into the blood stream. The arthritis sufferers recovered some strength and showed some reduction of inflammation. In the most successful cases, bedridden patients were sometimes able to resume normal activities that they had given up years before. Every one of the two hundred or more patients used the drug only as directed, even though they did experience a mild euphoria from it (see *Arthritis News Today*, 1980). Ronald Siegel (1989, p. 308-312), who reviewed the effects on the entire patient population, reported that Esterene seemed to have the same effect as chewing coca leaves.

When the Esterene story hit the newspapers, the government shut down the California clinic where Esterene was being administered and disciplined the doctors prescribing it, without investigating its efficacy. As well, sufferers from rheumatoid arthritis began to experiment with intranasal free base cocaine outside of the medical setting. Siegel (1989) was able to track down 175 illegal arthritic users in the Los Angeles area:

> Surprisingly most were not experiencing problems. They reported antifatigue effects, as well as suppression of chronic pain and discomfort, but they failed to experience the rapid and reinforcing euphoria that gives cocaine its addictive potential. Unlike daily cocaine hydrochloride users who repeatedly dose themselves throughout the day, people sniffing cocaine free base administered the drug infrequently and did not show signs of dependency. Some had financial or legal problems associated with their use; several also experienced loss of appetite or sleep. Yet their ability to maintain daily doses as high as 1,000 milligrams without severe dysfunction suggested that safe use was possible even in nonmedical settings (pp. 310-311).

Epidemiological Research. Numerous surveys in the U.S. and Canada indicate that cocaine use peaked in the 1980s at levels that had not been seen since early in the 20th century. However, in spite of widespread availability and moderate prices, a majority of North Americans never used cocaine; of those who did, most used it only once or a few times; of those who became more regular users, most did not become addicted; and of those whose addiction became serious enough to require treatment, most had lives that were marked by severe alienation or misfortune before they first used cocaine, suggesting that their addiction had causes other than mere exposure to the drug. These facts come from field studies from various countries.

An American national survey, conducted annually since 1975, involves random sampling of two groups: high school seniors and high school graduates up to age 32. In 1990, for example, 8.6% of high school seniors reported having used cocaine (other than "crack") at some time in their life, 1.7% reported using it once or more in the month that they were interviewed, and 0.1% reported using it at least 20 days in the month that they were interviewed. Thus, in contradiction to the strong form of Claim A, less than 1 student in 80 who admitted to having used cocaine could be considered a current addict, if addiction is assumed to require use on at least 20 days out of a month (Johnston, O'Malley, and Bachman, 1991). Moreover, data reported below indicates that only a fraction of those who use regularly can be considered addicted if a fuller definition of the term is applied.

The likelihood of a cocaine user becoming an addict seemed even lower among the high school graduates. In this group, 41% reported having used cocaine at some time in their life, 3% reported use of cocaine at least once in the month of the interview and less than 0.1 % reported using it at least 20 days in the month of the interview. Thus, less than one student in 400 who reported having used cocaine could be considered a current addict (Johnston, O'Malley, and Bachman, 1991).

Other American surveys have produced similar results and revealed some further patterns. Kandel, Murphy, and Karus (1985) randomly selected a group of people approximately 25 years of age in New York State. Of the 30% of this group who had ever used cocaine, about 60% had used it less than 10 times in their entire lives, 31% had used it 10-99 times, 6% had used it 100-999 times, and about 3% had used it 1000 times or more. Approximately 2% of the respondents had used cocaine daily at some time, but only about 4/10 of 1% continued to do so in the year of the study (based on re-calculation of data from Kandel, Murphy, and Karus, 1985: 80-1). If using cocaine 10-99 times is a reasonable minimum necessary to lead to addiction under the cautious form of Claim A, and if we take daily use at some time in a person's life as a reasonable minimum that would be expected in an addicted person, we can say that no more than 1 in 15 of the respondents who used cocaine enough to be addicted according to the cautious form of Claim A could ever have been addicted and no more than 1 in 77 could have been addicted at the time of the interview.

Some American studies appear more supportive of claim A, but these generally are based on populations of people in treatment, or on a loose definition of addiction. For example, Kilbey, Breslau, and Andreski (1992) found that 124 of 1007 largely middle class people in the Detroit metropolitan area had used cocaine more than 5 times. Of these 124 people almost 10% had fulfilled the definition for cocaine dependence at some time during the year of the interview. Thus, almost 1 in 10 people who used cocaine as often as 5 times were diagnosed as cocaine dependent at some time in the year of the interview. These data support a weak form of Claim A.

The difference between these results and those of other American studies which suggest a much lower addiction rate probably lies in the inclusive definition of "dependence" used by Kilbey et al. In their diagnostic manual--the DSM-III-R (American Psychiatric Association, 1987)--a person is said to be dependent on a drug if they fit any 3 of 9 criteria. This flexibility makes it possible to diagnose as dependent both people who are fully addicted in the traditional sense of the word as well as people who would not be considered addicted in ordinary language. The DSM-III-R is explicit about this, for it describes one form of dependence ("mild dependence") as the case in which ..."the symptoms result in no more than mild impairment in occupational functioning or in usual social activities or relationships with others" (p. 168). This kind of dependence is not addiction in the ordinary sense--unless we are willing to say that people are "addicted" to all the activities that mildly interfere with their work performance or social life. It is not that mild dependence on cocaine is unimportant. It causes some problems but, apart from the risk of arrest on drug charges, these are no greater than those associated with regularly overeating or watching too much television. Addiction, as usually defined, is a much more serious problem than this.

Canadian survey data are usually far less extensive than American data, but the results are similar. The "Ontario Household Surveys" have provided the best trend data available for Canadian adults. The proportion of Ontarians reporting ever using cocaine nearly doubled, from 3.3% to 6.1% of the population between 1984 and 1987 and thereafter remained relatively stable or declined. Of those who had ever used cocaine, 95% reported using it less than once a month in 1987.(Smart and Adlaf, 1988; 1992).

A survey conducted by the Co-ordinated Law Enforcement Unit (1987) in British Columbia revealed higher rates of experimental use in B.C. than in Ontario, but again showed that most of those who use cocaine do not become frequent users. Of a random sample of respondents from throughout the province, 11.2% reported having used cocaine at least once in their lifetime. Of these, 56% had used it less than 10 times in their lives, 36% had used it 10 -99 times, and 8% had used it 100 times or more. (These data are recalculated from the original report into a form comparable to data presented above).

The highest report of cocaine use in any single Canadian subpopulation not in treatment comes from my own research at Simon Fraser University, in British Columbia, where my student interviewers took the time to interview each subject at considerable length under conditions of confidence and security about the way in which they used drugs as well as the frequency. Of 107 students interviewed, 40.2% reported using cocaine at some time in their life. However, only 4 of these had used cocaine at all in the previous 30 days and none had used it daily during that period. One student reported having been a regular user of cocaine in the past, but was no longer. No student reported being or having been addicted to cocaine, although reports of addiction to other drugs were not unusual (Alexander, 1985). These data contradict the strong form of Claim A which holds that any exposure to cocaine at all should yield a high rate of addiction.

Taken together, the American and Canadian population surveys indicate that merely having used cocaine is associated with less than a 10% chance of having it as often as 100 times. Virtually all addicts use it far more than 100 times.

There are several studies of cocaine users in various countries who were located through advertising and/or personal networks. Since these respondents are volunteers, they do not represent all cocaine users. They do, however, provide in-depth information on heavier users who are not in treatment, a group of cocaine users who show up only rarely in random surveys and never in studies of addicts in treatment. These studies show that, contrary to Claim A, there are many regular users of cocaine who cannot be considered addicted, and many who have passed into and out of regular use without lasting addiction or outside intervention. These "snowball sample" studied come from Canada (Cheung & Erickson, 1997; Erickson et al., 1994); the United States (Murphy, Reinarman, and Waldorf, 1989; Waldorf, Reinarman, and Murphy, 1991; Reinarman & Levine, 1997); the Netherlands (Cohen, 1989; Cohen &

Sas, 1993); and Australia (Mugford & Cohen, 1989). I supervised a study of this type for the World Health Organization in Vancouver (Matthews et al., 1994; WHO/UNICRI, 1995)

In a study conducted in the San Francisco area, a group of cocaine users who were initially interviewed in 1974-1975 was followed up after 11 years (Murphy, Reinarman, and Waldorf, 1989; Waldorf, Reinarman, and Murphy, 1991). Of the original 27 respondents, 21 were re-interviewed. There had been no formal contact with the respondents in the 11 year hiatus between interviews. The original sample was characterized as a "naturally occurring friendship network" in which the age range was 16 to 51, the sex ratio was approximately equal, and most of the respondents were university students or graduates. [how much did they use?] In 1977, the investigators did not consider any of the 27 respondents to be addicted. Most were described as casual users, However 4 used daily.

The follow-up interviews revealed that all 21 respondents were gainfully employed, many in professional and managerial positions. One of the 21 respondents, i.e., 5% of the group, was currently a compulsive cocaine user. Eleven others reported having used cocaine daily at some point, but were no longer doing so. Seven of these eleven had reduced their consumption from as much a 3 grams a week to one quarter gram or less but continued to use in a controlled way. Four had adopted abstinence after periods of heavy uncontrolled use. Seven other subjects were characterized as "continuous controlled users" who maintained moderate use patterns throughout the eleven year period. Two other past users of this continuous controlled type had stopped using entirely for two and five years prior to the follow-up interview.

With respect to claim A, this study shows that 20 of 21 people who had used cocaine for years retained, or lost and then regained, a pattern of controlled use of cocaine. For 4 of the respondents this eventually meant abstinence. Between the beginning and the end of the study a minority of the respondents passed through periods during which they could have been considered addicted.

The fact that people pass into and out of addiction contradicts an implicit supposition that accompanies Claim A. This is the supposition that entry into addiction is an irreversible transition. If this were so, the possibility that cocaine may cause addiction in some users would be extremely serious. However, beyond the San Francisco study reviewed above, many interview studies have now demonstrated that it is common for people to pass through a period of dependence of cocaine addiction and to return to moderate use or abstinence without social intervention or any dramatic discomfort (Cohen, 1989; Erickson, Adlaf, Murray, and Smart, 1987; Matthews et al., 1994). Addiction to cocaine is typically a temporary rather than a permanent condition.

In another study, Erickson, et al. (1994) interviewed 111 Canadian cocaine users who had at least one experience with cocaine in the past three years. Attempts were made to attain a typical sample of users. Two-thirds of the respondents were males, the age range was 21 to 44, and all had been employed in the year prior to the interview.

Nearly all respondents reported favouring the intranasal route of administration, and the average duration of time since first exposure to cocaine was 7 years. While a majority of the respondents (58%) had used less than 10 times in the previous year, only 9% reported using on 100 or more occasions during that period.

Although quantities consumed on any single occasion were generally small, i.e., 6 "lines" or less over several hours, many of the respondents reported occasional binges of intensive use and "runs" lasting 2 days or more at some time. Respondents reported considerable fluctuation in their consumption of cocaine. About half (51%) reported more intensive periods of use in the past, usually of short duration and mainly in response to greater availability. Over half (61%) reported cutting back on their cocaine use at some time and provided a variety of reasons including less availability, concern with physical risks and overuse, loss of interest, and lifestyle changes.

Most of those interviewed by Erickson et al., (1994) were infrequent cocaine users who clearly were able to limit their use of cocaine. Restricting use to party situations or special occasions, buying little or none at all, having a stable employment and/or domestic situation, and appreciating the risks of cocaine were some of the factors that appeared to reinforce the controlled use of cocaine. Between 5% and 10% of these Canadians developed very heavy or compulsive cocaine use at some time. The majority of those who had engaged in more intensive periods of cocaine use cut back on their own initiative. Seven individuals had sought treatment related to cocaine use, mainly for medical complications rather than for addiction. These observations would seem to directly contradict even the cautious form of Claim A.

Crack Cocaine. There was an explosion of publicity in the American and Canadian media a few years ago about crack being the "most addictive drug on earth", and causing "instant addiction" (Trebach, 1987; Reinarman and Levine, 1997, chap. 1). Sufficient time has now passed to evaluate these claims empirically. They are simply false.

The great majority of users of crack and other forms of smokable cocaine are experimenters who smoke cocaine a few times and subsequently lose interest. There are also a number of users who use it intermittently over longer periods without serious difficulty. It is true that some people who smoke cocaine become rapidly and tragically addicted, and others discontinue use because they feel that they are "losing control" over their intake, but these are a small minority of users, comparable in size to the minority of users of alcohol, heroin, credit cards, computers, and sex who either become dangerously obsessed or learn that they must abstain (Inciardi, 1987; Waldorf, Reinarman, and Murphy, 1991; Morgan & Zimmer, 1997a; Peele & DeGrandpre, 1998).

The evidence concerning the addictive potency of crack cocaine is consistent over time. In fact, there never was any empirical data to support the claims of "instant addiction". The picture has remained consistent since the very earliest research on the first "crack epidemic" reported in Miami, Florida.

For example, Inciardi (1987) reported that juvenile delinquents in Miami generally preferred cocaine hydrochloride to alkaloid cocaine, because its effects lasted longer. On the other hand, many of them used crack in addition to cocaine hydrochloride because it was sold in smaller, cheaper doses. Inciardi reported that addiction to crack was rare among the delinquents he interviewed.

The 1990 survey of 19-32 year old American high school graduates cited above reported that 5.1% had used crack at least once in their life, but only 0.4% had used it once or more in the month of the interview, and less than 0.05% had used 20 or more days in the month of the interview. Thus, the "most addictive drug on earth" caused persisting addiction in no more than 1 experimental user in 100 (Johnston, O'Malley, and Bachman, 1991), contrary to Claim A.

Indirect evidence that has been used to argue that smokable cocaine carries an especially high addictive liability is also weak. For example, there are a number of longtime heavy users of cocaine who became addicted only after they switched to smoking cocaine (Siegel, 1985; Waldorf, Reinarman, and Murphy, 1991). Does this necessarily mean that "crack" and "freebase" cocaine have a high "addictive liability", even if cocaine hydrochloride does not? A more parsimonious interpretation of these results would be that the people who exposed themselves to alkaloid cocaine were especially needful of cocaine's stimulation. Waldorf et al. estimate that their original sample of 267 cocaine users came from among the heaviest 1% of cocaine users in the U.S. (p. 2). One-fifth of this group, 53 people, had switched from cocaine to smoking crack. The main reason was that they were seeking a more intense, less expensive experience:

> ...All that is necessary to explain most freebasers' entry into this method is a relatively common desire to achieve what might be called more bang for the buck...For example, one of my respondent said that he first freebased when a cousin told him he was "wasting coke" by merely snorting it...(110-111).

Thus, the cocaine smokers came from the 1/5 of the top 1% of American cocaine users who had such a strong desire to intensify their experience that they switched from cocaine hydrochloride to smoking crack or freebase, in spite of the incessant media reminders that smoking cocaine carried a high risk of addiction. It is hard to conclude from the addiction of some members of this special group that crack would cause addiction in people in general.

Abundant later evidence has continued to demonstrate the falsity of Claim A for crack cocaine, the drug to which it is almost universally believed to apply (Reinarman & Levine, 1997).

I believe it is of the utmost importance for the Senate to contemplate why a totally false, never-documented belief could have had the universal support of the newsmedia, reputable scientists, the then-government of Canada, and the people. I will take up this question after consideration of the evidence for Claim B, where the situation is logically more complex than in the case of Claim A.

Claim B: Review of the Logic and Evidence

Logic Used to Support Claim B. Whereas Claim A assumes that sufficient exposure to heroin or cocaine will cause addiction in virtually any person, Claim B allows for the possibility that only certain people are at risk. It asserts the existence of a subpopulation of "vulnerable" or "predisposed" people for whom exposure to heroin or cocaine causes addiction.

It is of course true that a small percentage of people who try heroin or cocaine do become addicted to it, but this in itself does not provide any evidence for Claim B, if the normal rules of logic are applied. Claim B asserts a cause—exposure to the drug—that transforms people into addicts against their own will. Claim B comprises a type of pharmacological determinism that is compatible with the overall assumptions of neurochemistry and psychopharmacology. Although a great deal of technically sophisticated research has been devoted to Claim B in these fields, the evidence is very far from a conclusive proof of it (Nadeau, in preparation). Some of this evidence will be examined below.

A non-deterministic explanation is logically just as plausible as a deterministic one. It is possible that a small percentage of people choose, consciously or unconsciously, to adopt drug use permanently following an initial experience, in the same way that other people, following an initial experience, commit themselves strongly and permanently to a religion, a political position, a mate, a life of crime, or a thousand other lifestyle commitments which sometimes have catastrophic consequences (Alexander, 1990; Schaler, 2000).

Careful scrutiny of the existing evidence shows that the deterministic hypothesis (Claim B) is no better substantiated than any of a large number of non-deterministic alternative hypotheses. For example, there is evidence that many heroin and cocaine experimenters who become addicts suffer from chronic, severe depression, anxiety, or physical pain. If the pain is sufficiently intense, people who become aware of the analgesic or tranquilizing effects of heroin or cocaine are likely to cling to the drug to relieve this pain, in spite of all the difficulties that this entails. The expense and legal obstacles to heroin or cocaine use would require many desperate people to engage in criminal activities to obtain the relief they need and, in extreme cases to become overwhelmingly involved in obtaining their drug. This idea is sometimes called the "self-medication hypothesis". It finds considerable support in the literature of clinical and research psychiatry, although the evidence is far from conclusive (Khantzian, 1985; 1997). If this interpretation were correct, the drug would not be the cause of addiction, but instead the pre-existing pain and the person's desperate attempts to control it would be. It would be assumed that if the pain were removed, the person would abandon their compulsive use of the drug.

Another plausible alternative to Claim B is that desperately alienated and isolated people choose to immerse themselves in the drug addict subculture because they find that it provides a substitute society that they desperately need, in spite of the hazards associated with drug use. Again, if this alternative is valid, the drug is not the cause of addiction. Rather, certain people choose an addictive lifestyle as a lesser evil in desperate circumstances. The same people would be expected to turn to a variety of other drugs and lifestyle addictions if their drug of choice were not available. This alternative explanation for addiction is sometimes called the "adaptive model". Like the "self-medication hypothesis", the adaptive model finds extensive support, but not conclusive proof, in the clinical, research, and historical literature (Alexander, 1990, chap. 8; Alexander, 2000).

Evidence used to support Claim B

. Although Claim B has not been resolved by empirical test, and quite possibly cannot be, there are three kinds of evidence that are widely seen as validating it. All three can only be accepted if people think loosely, for none comes close to providing conclusive support for Claim B.

Personal Testimony of Addicts. Many addicts and former addicts describe with powerful intensity how using heroin or cocaine instilled an irresistible drug appetite in them and thus destroyed an otherwise satisfactory life (See Burroughs, 1959; Courtwright, Joseph, and DesJarlais, 1989, chap 3; Lemere and Smith, 1990; Waldorf, Reinarman, and Murphy, 1991). However, these sincere reports must be viewed with the same skepticism as all other evidence.

Davies (1992) has shown that the attribution of causal power to drugs by addicts can be best explained by a set of psychological principles known as "attribution theory". Consciously or unconsciously, most people (not just addicts) attribute causes to their own behaviour for the purpose of maximizing their self-esteem, rather than describing reality. Thus, it is unlikely that what addicts say about the cause of their own drug use provides any evidence about Claim B. To claim that one has been transformed into an addict by exposure to a drug can serve the same function as pleading guilty to a lesser offence: Rather than bearing responsibility for unacceptable behaviour, the person is only responsible for the unwise experimentation that transformed him or her into an addict, against their best intentions. There is good experimental support for attribution theory in many areas of human behaviour including addiction. For example, Davies (1992) shows that addicts describe themselves more in terms of Claim B when they are being interviewed by an authority figure than when they are being interviewed by another drug user.

Of course it is possible that cocaine addicts and junkies are more honest than ordinary citizens, and therefore the "attribution theory" that applies to the general population does not apply to them. But this strains credulity, since in most other circumstances cocaine addicts and junkies are regarded as "in denial", as unreliable witness, and, quite often, as pathological liars. My own experience with cocaine and heroin addicts is that they are quite divided on this question. A few consistently describe their addiction in terms of Claim B, a larger number consistently describe it in non-deterministic terms, and perhaps the largest number are inconsistent in their explanation, like those described by Davies above.

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Animal Behaviour Research

. The belief in drug-induced addiction is often justified by reference to research on the self-administration of drugs by laboratory animals. However, when carefully scrutinized, this research too affords little real support for the belief.

In the early 1960s, researchers at the University of Michigan perfected devices which allowed relatively free-moving rats to inject themselves with drugs. After implantation of a needle in one of their veins connected to a pump via a tube running through the ceiling of a special Skinner box, rats could inject themselves with a drug simply by pressing a lever. By the end of the 1970s, hundreds of experiments with apparatus of this sort had shown that rats, mice, monkeys, and other captive mammals will self-inject large doses of heroin, cocaine, amphetamines, and a number of other drugs (Woods, 1978).

Many people concluded that these data also constituted proof for the belief in drug-induced addiction. If most or all animals in an experimental group inject heroin and cocaine avidly and to the detriment of their health (as they did in some experiments), does it not follow that the power of these drugs to instill a need for future consumption transcends species and culture and is simply a basic fact of mammalian existence? One eminent American scientist put it this way:

> If a monkey is provided with a lever, which he can press to self-inject heroin, he establishes a regular pattern of heroin use--a true addiction--that takes priority over the normal activities of his life...Since this behaviour is seen in several other animal species (primarily rats), I have to infer that if heroin were easily available to everyone, and if there were no social pressure of any kind to discourage heroin use, a very large number of people would become heroin addicts. (Goldstein, 1979, 342).

But this statement is in direct contradiction to careful observations of the era in which heroin was freely available in North America and observations of people given relatively free access to opiate drugs in PCA machines, described above. Moreover, some researchers have pointed out that opiate ingestion by laboratory animals could be understood as a way that the animals cope with the stress of social and sensory isolation and the restriction of movement that is imposed by the complex self-administration apparatus. Other researchers pointed out that the mere regular use of a drug by animals or human beings is not equivalent to addiction, as discussed above.

To determine whether the self-injection of heroin in these experiments could be an artifact of social isolation, a group of researchers at Simon Fraser University began a series of experiments in the late 1970s which were eventually dubbed the "Rat Park" experiments. Albino rats served as subjects and morphine hydrochloride, which is equivalent to heroin, as the experimental drug. Laboratory rats are gregarious, curious, active creatures. Their ancestors, wild Norway rats, are intensely social and hundreds of generations of laboratory breeding have left many social instincts intact. Therefore, it is conceivable that the self-medication hypothesis might provide the most parsimonious explanation for the self-administration of powerful drugs by rats that were raised

in isolated metal cages and subjected to surgical implantations in the hands of a eager (but seldom skillful) graduate student followed by being tethered in a self-injection apparatus. The results of self-injection experiments would not show that claim B was true so much as that severely distressed animals, like severely distressed people, will relieve their distress pharmacologically if they can (Weissman and Haddox, 1989).

My colleagues at Simon Fraser University and I built the most natural environment for rats that we could contrive in the laboratory. "Rat Park", as it came to be called, was airy and spacious, with about 200 times the square footage of a standard laboratory cage. It was also scenic, (with a peaceful British Columbia forest painted on the plywood walls), comfortable (with empty tins, wood scraps, and other *desiderata* strewn about on the floor), and sociable (with 16-20 rats of both sexes in residence at once).

In the rat cages, the rats' appetite for morphine was measured by fastening two drinking bottles, one containing a morphine solution and one containing water, on each cage and weighing them daily. In Rat Park, measurement of individual drug consumption was more difficult, since we did not want to disrupt life in the presumably idyllic rodent community. We built a short tunnel opening into Rat Park that was just large enough to accommodate one rat at a time. At the far end of the tunnel, the rats could release a fluid from either of two drop dispensers. One dispenser contained a morphine solution and the other an inert solution. The dispenser recorded how much each rat drank of each fluid.

A number of experiments were performed in this way (for a more detailed summary, see Alexander et al., 1985), all of which indicated that rats living in Rat Park had little appetite for morphine. In some experiments, we forced the rats to consume morphine for weeks before allowing them to choose, so that there could be no doubt that they had developed strong withdrawal symptoms. In other experiments, I made the morphine solution so sickeningly sweet that no rat could resist trying it, but we always found less appetite for morphine in the animals housed in Rat Park. Under some conditions the animals in the cages consumed nearly 20 times as much morphine as the rats in Rat Park. Nothing that we tried instilled a strong appetite for morphine or produced anything that looked like addiction in rats that were housed in a reasonably normal environment.

I will illustrate the kinds of results we found by describing a single experiment in a slightly simplified way (for a complete description see Alexander et al., 1981). This experiment entailed making a morphine solution progressively more appealing to the rat palate, in order to determine at what point the rats that were isolated in cages and those housed in Rat Park would drink large quantities of it.

Morphine in solution is unpleasantly bitter to human beings and apparently also to rats, since they reject it with the same signs of distaste they direct towards extremely bitter quinine solutions. Offered a simple choice between water and morphine solution, rats typically sample a drop or two of the morphine solution, shake their heads in distaste, and thereafter ignore it. We set out to overcome the taste barrier by making adding enough sugar to the morphine solution to make it sickeningly sweet, a taste experience that rats normally find extremely enticing.

The experiment involved four groups of rats. Group CC was isolated in laboratory cages when they were weaned at 22 days of age and lived there until the experiment began at 80 days of age. Group PP housed in Rat Park for the same period. Group CP was moved from laboratory cages to Rat Park at 65 days of age, and group PC was moved from Rat Park to cages at 65 days of age. All tests of morphine preference were conducted around the clock in the animal's home environment.

We pretested the rats with a choice between water and a bitter sweet, pharmaceutically inert sugar-quinine solution to see if there was any pre-existing taste preference between the four groups, which their was not. Then I gave them a continual choice between water and a bitter sweet morphine solution. Every five days the drug solution was changed so that it tasted better and had less drug effect--in other words I progressively reduced the morphine concentration from 1 mg morphine hydrochloride per ml of water to .15 mg morphine hydrochloride per ml of water. At the 1 mg/ml level the solution was too bitter for all the rats and they consumed only water. At all subsequent levels the caged rats (groups CC and PC) drank much more morphine than the rats that lived in Rat Park (groups PP and CP). At concentration level, the caged males drank 19 times as much morphine as the Rat Park males. The differences for females were similar, although not as extreme. The most interesting group was the rats brought up in cages but moved to Rat Park before the experiment began (group CP). These animals shunned the morphine solution when it was stronger, but as it became more tasty and more dilute, they began to drink almost as much as the rats that had lived in cages throughout the experiment.

I believe these results, which have subsequently been extended by other experimenters (e.g., Bozarth, Murray, and Wise, 1989; Schenk, et al., 1987; Shaham, et al., 1992) show that the earlier animal self-administration studies provide no real empirical support for the belief in drug-induced addiction. The intense appetite of isolated experimental animals for heroin and cocaine in self-injection experiments tells us nothing about the responsiveness of normal animals and people to these drugs. Normal people can ignore heroin and addiction even when it is plentiful in their environment, and they can use these drugs with little likelihood of addiction, as discussed above. Rats from "Rat Park" seem to be no less discriminating.

Physiological Research. Another line of animal research is often interpreted as showing that certain "dependency producing" drugs can instill a powerful appetite for continued use. This research can be taken to support Claim A as well as Claim B, depending on whether the process that is seen as instilling the powerful appetite in animals is said to exist in all or some human beings.

A group of researchers at MacMaster University in Canada has focussed on the ability of heroin, after it has been administered a number of times, to produce distressing "drug-compensatory responses" in rats, which create a powerful need for the drug when it is not available--in other words, withdrawal symptoms. This group has shown that these compensatory responses can be re-instituted as a conditioned response when a rat is abstinent, which might cause relapse (see Siegel, Krank, and Hinson, 1987).

A group of researchers at Concordia University in Montreal have marshaled evidence that cocaine and other drugs potentiate the action of the neurotransmitter dopamine in the medial forebrain bundle and possibly elsewhere. By mimicking and overshadowing the effect of natural rewards on this powerful and important reward system heroin and cocaine, and presumably other "dependency-producing" drugs, are thought to become almost irresistible once a rat or person has been exposed to them several times (see Wise, 1988a, b).

Although this research is sophisticated and technically impressive, it has not demonstrated that heroin and cocaine instill an addictive need for heroin or cocaine in rats or human beings outside of the restricted laboratory environment. Rather, this research simply *assumes* that claim B is true, and explores physiological responses which might explain why. So powerful is the mystique of high-tech research, However, that it is easy become convinced of the assumptions that it makes.

An experiment by Gill, et al., (1991) illustrates the power of assumptions in this type of research. The title of the article indicates that Gill et al. (1991) intended a direct test the "dopamine depletion hypothesis of cocaine dependence". This hypothesis holds that exposure to cocaine causes addiction by weakening the brain's capacity to produce endogenous dopamine, thereby requiring the person or animal to rely on a continuous supply of cocaine as a replacement. Neurochemical tests were conducted on abstinent cocaine addicts in a treatment program, but no convincing signs of dopamine depletion were found in their brains. Having failed to confirm their hypothesis, the researchers did not question the underlying assumption that exposure to cocaine causes addiction, but rather suggested that it was more likely that the addiction was produced by a desensitization of dopamine receptors in the brain than by a depletion of the supply of dopamine. In this line of research, Claim B is simply assumed; the task is to uncover the physiological explanation. Obviously merely assuming a hypothesis does not make it true, but when a large number of sophisticated scientists consistently assume a hypothesis, it can create an illusion of truth is the general public.

Summary of Support for Claims A and B

Thus, Claim A is demonstrably false and Claim B is no more than one of a number of unsubstantiated, but plausible explanations for the fact that some drug users become addicts. The belief in drug-induced addiction, at least with respect to heroin and cocaine, has no status as empirical science, although it has not been disproven. It is believed for some reason other than its empirical support.

I believe that the universal acceptance of the belief in drug-induced addiction by Canadian should be a topic of serious contemplation at a time when major changes in drug policy are under consideration. Why have the media, the general public, reputable scientists, and the government of Canada expressed near-universal belief in the unsubstantiated idea of drug-induced addiction for so many years?

Bases of Acceptance of the Myth of Drug-Induced Addiction

Historians have identified a number of apparent reasons for the acceptance of the "War on Drugs", including the belief in drug-induced addiction, which has been one of its major sustaining myths (Alexander, 1990, chap. 8). I will review some of these reasons briefly, and add some more from other sources. I hope that this review may convince the Senators that none of these reasons are sufficiently weighty to deter the Government of Canada from unburdening itself from the myth of drug-induced addiction in its search for more efficacious policy. I will not distinguish between reasons for supporting Claim A and Claim B in this section, since they are most often lumped together.

Reasons that the belief in drug-induced addiction has received public support

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Shifting patterns of drug use. In the 1980s there was a substantial increase in people seeking treatment for cocaine addiction in the United States and Canada . This fact is sometimes taken to indicate a great "addictive liability" inherent in cocaine. However this conclusion is unwarranted. It turned out to be more the case that the increase in clients seeking help for cocaine addiction resulted from a shifting preference by polydrug addicts, from alcohol and heroin to cocaine, as the latter became popular and inexpensive.

Rationalisation of intractable problems.

Addicts can solve an intractable problem by believing in drug-induced addiction. If they accept this belief, they can escape an enormous burden of guilt for their catastrophic lives, because the active agent is not themselves, but the drug. They made only one mistake and forgot to "just say no": The rest was out of their control.

Often such rationalisation provides a merciful relief for a suffering addict, at least for a time. Sometimes it is useful for drug abuse counselors to accept this belief during therapy. But why do authorities on drug addiction believe addicts when they voice such transparent rationalisations? At least part of the explanation is that the same rationalizations serve to relieve the intractable problems of people other than the addicts themselves.

For example, Claim B provides parents of addicts a guarantee that the blame for an offspring's addiction will not be directed at themselves. I saw this first hand during the years that I worked as a family therapist with families of heroin addicts. It was painfully apparent how much the mothers and fathers of heroin addicts needed to believe that drugs and bad companions had addicted their child, and that the child's upbringing had nothing to do with their current disastrous plight. (As a parent myself, I find this totally understandable, so my observation here intends no disrespect to these anguished and well-meaning people.) It is a fact that most parents will not participate in family therapy sessions unless they are assured in advance that the therapist believes that they are not responsible, as parents, for the catastrophe that has struck their family. As therapy proceeds, many parents gradually come to perceive a more complex reality, but this only develops gradually in most cases (Alexander & Dibb, 1975).

The uncritical acceptance of the rationalisations of addicts concerning the cause of their addition is also comprehensible from the standpoint of professionals in law enforcement, who can justify budgets on the basis of the urgent need to keep addictive drugs out of the hands of the unsuspecting. It is also comprehensible from the standpoint of the intractable problems of the larger society. In what kind of a society would addiction run rampant, not just to drugs but to money, power, sex, work, etc.? This is a question that I have tried to analyse historically in recent years (Alexander, 2000), but it is one that must be approached with great sensitivity. Nobody wants to rush into a consideration of the possibility that Canadian society mass-produces addiction. It is quite understandable that people would rather suppose that we can solve our growing social problems by getting rid of the drug lords who are distributing irresistibly addictive drugs that cause the problems. If it were so, a War on Drugs could solve our problems. It seems, however, that we are being gradually pushed by circumstances into considering a more comprehensive set of possibilities.

Money. There is no pharmacological reason that heroin and cocaine are prohibited for virtually all purposes, whereas drugs which share all the same positive effects as well as addictive qualities—for example, meperidine (tradename: Demerol) and methylphenidate (tradename: Ritalin)—are legally prescribed (Perrine, 1996, chaps. 2 and 4). There is however an important commercial reason. Heroin and cocaine can be produced as cheaply as any other agricultural products and are therefore potentially formidable competitors to the alternative drugs that produce substantial profits for the drug companies.

Drug companies have been among the most willing contributors to expensive publicity campaigns designed to promulgate the myths of the drug war, including the myth of drug-induced addiction. This is perfectly understandable in terms of the drug companies' conscientious devotion to shareholder value, but it is not a basis for allowing these myths to constrain public decision making.

International Politics. It seems inconceivable to me, and to many other citizens of my acquaintance, that there would not be a international political explanation for the Canadian government's wholesale acceptance of the myths of the War on Drugs during the Mulroney years or behind the subsequent reluctance of the Canadian government to adopt new policy directions. Since the Senators' expertise on political factors is infinitely greater than mine, I will not here venture any of my thoughts about what the political reasons might be. I would venture to suggest, however, that the politics of the drugs wars will have to become public knowledge before any complete removal of drug war myths from the public mind can be achieved.

Conclusion

I hope that this short review is sufficient to show that the conventional belief that heroin and cocaine cause addiction is very far from an empirically supported fact. By the normal, skeptical standards of science, Claim A is false and Claim B is an unsubstantiated hypothesis.

Moreover, the conventional belief in drug-induced addiction appears to persist because it serves personal, social, professional, commercial, and political needs. I do not mean to imply, however, that these needs are unimportant. Empirical science is not the only road to truth--conventional wisdom must be evaluated pragmatically as well as scientifically. However, at this point in history, the conventional belief in drug-induced addiction may be doing more harm than good.

There was a time when society spoke with unshakable certainty of the terrifying dangers that resulted from even a word of religious heresy and of the incurable consequences of occasional childhood masturbation (Bullough, 1987). At the time, terrifying rhetoric seemed necessary to frighten people away from socially unacceptable behaviours. But the consequences were brutal. Moreover, the scare tactics eventually lost their power anyway. Much the same seems to be occurring now, as both the brutality and the futility of the "War on Drugs" are becoming more and more evident. There are times in history when society is better served by dispassionate information than by manufactured fear.

My hope is that this quick survey of the illusory scientific support for the conventional belief that heroin and cocaine cause addiction can help to show why society should turn away from this unsupported belief. Understanding that there may not be any inherent addictive power in drugs could help to turn us toward a broader, more efficacious formulation of the causes of addiction in our time, and of the huge, dismal saga of tragedy that it produces.

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Home | Important Notices