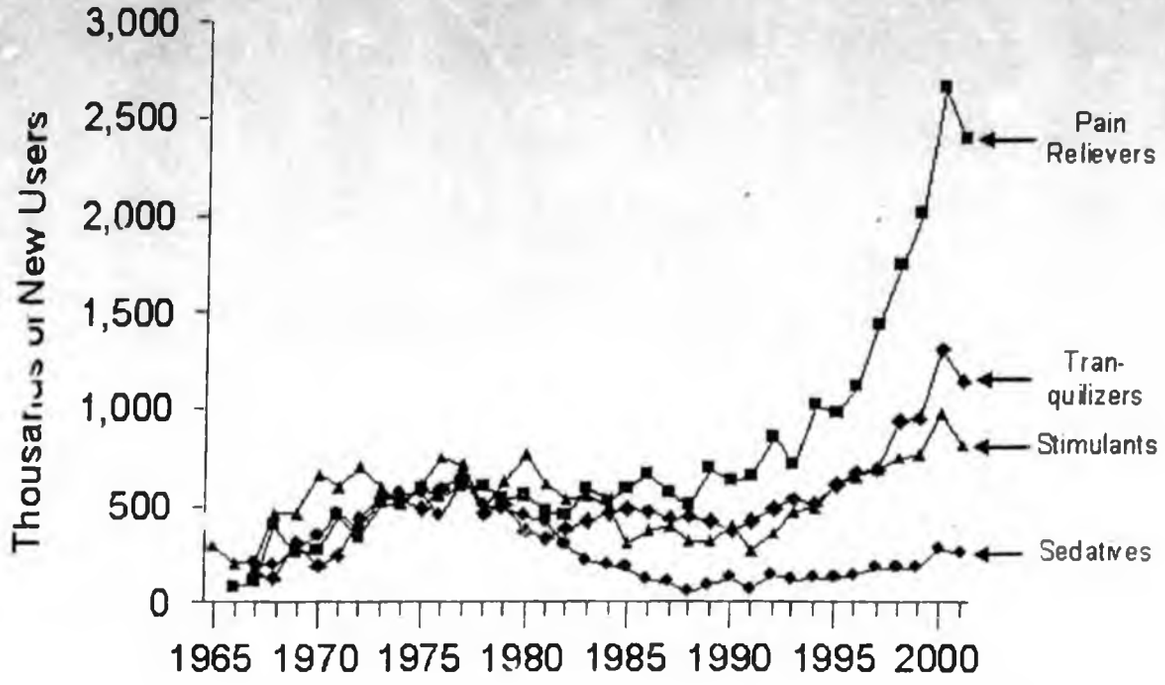
11778 SENATE HEALTH, EDUCATION & SOCIAL SERVICES



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- First use of stimulants increased during the 1990s from 270,000 in 1991 to 983,000 in 2000 and 808,000 in 2001.
- Incidence of methamphetamine use rose between 1991 (210,000 new users) and 1998 (454,000 new users). Since then, there have been no statistically significant changes. There were an estimated 326,000 methamphetamine initiates in 2001.
- Initiation of tranquilizer use increased steadily during the 1990s, from 372,000 initiates in 1990 to 1.3 million in 2000 and 1.1 million in 2001.
- The number of sedative initiates has remained below 300,000 per year after 1981. During the 1970s, the estimates had risen above 500,000 per year from 1973 to 1975 and peaked at 638,000 in 1977.

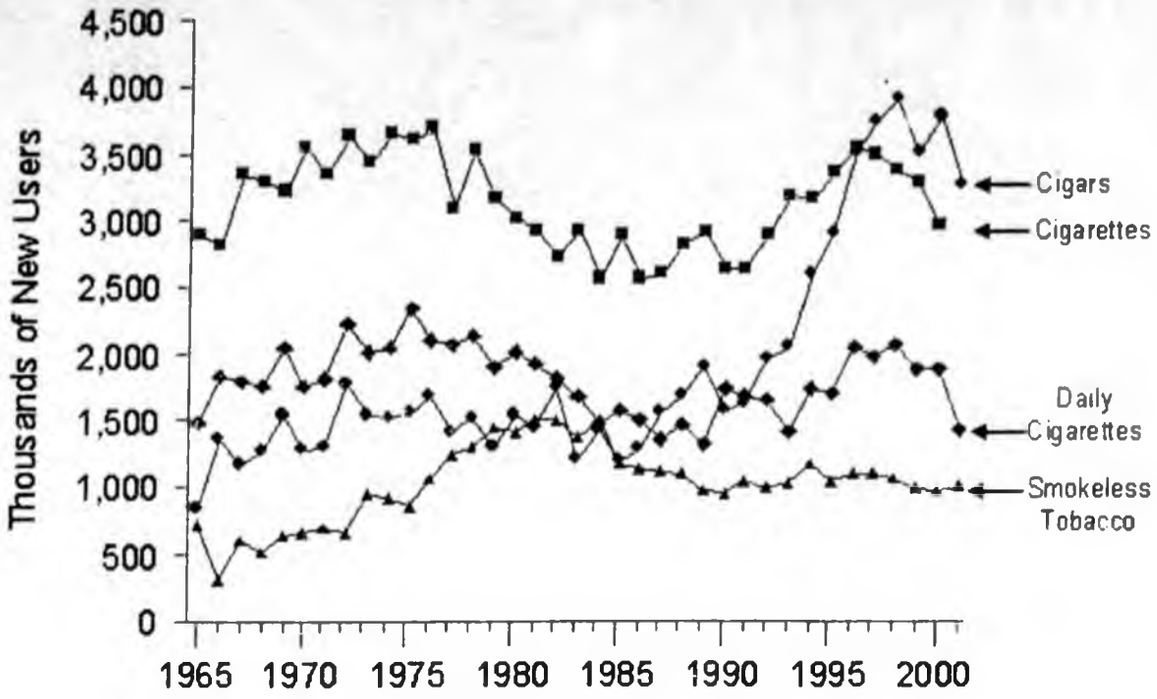
Alcohol

- Alcohol incidence increased steadily during the 1990s, from 3.3 million new users in 1990 to 5.6 million in 2000. Youths under 18 accounted for much of the increase, the number of adolescent initiates nearly doubling from 2.2 million in 1990 to 4.1 million in 2000. During this period, the increase was equally distributed among boys (1.1 million to 2.1 million) and girls (1.1 million to 2.0 million).

Tobacco

- Cigarette initiation increased from 2.6 million initiates in 1990 to 3.6 million in 1996, then decreased to 3.0 million in 2000. Initiation of cigarette use among youths under 18 significantly decreased from 2.8 million new users in 1996 to 2.2 million in 2000 (**Figure 6.4**).

Figure 6.4 Annual Numbers of New Users of Tobacco: 1965–2001



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- The number of new daily smokers decreased from 2.1 million in 1998 to 1.4 million in 2001. Among youths under 18, the number of new daily smokers decreased from 1.1 million per year between 1997 and 2000 to 757,000 in 2001. This corresponds to a decrease from about 3,000 to about 2,000 new youth smokers per day.
- Approximately three quarters (75 percent) of persons who tried their first cigarette in 2000 were under age 18. Among persons who first began daily smoking in 2001, about half (53 percent) were under age 18. Of the approximately 4,000 new regular smokers per day in 2001, approximately 2,000 per day were under age 18.
- Initiation of cigar smoking more than doubled during the 1990s, from 1.6 million new users in 1990 to 3.9 million in 1998. In 2001, the number of new users dropped to 3.3 million. Since 1990, youths under 18 have constituted an increasingly greater proportion of the number of new cigar smokers, from 21 percent in 1991 to 48 percent in 2001

P

10. Discussion

This report presents findings from the 2002 National Survey on Drug Use and Health (NSDUH). Conducted since 1971 and previously named the National Household Survey on Drug Abuse (NHSDA), the survey underwent several methodological improvements in 2002 that have affected prevalence estimates. As a result, the 2002 estimates are not comparable with estimates from 2001 and earlier surveys. The primary focus of the report is on the numbers of persons and rates for a variety of measures related to substance use and mental health in 2002, including comparisons across sociodemographic and geographic subgroups of the U.S. population. Some of the most important findings for 2002 are presented in the Highlights section of this report.

The prevalence estimates from the 2002 NSDUH are uniformly higher than the corresponding estimates from the 2001 NHSDA. Analyses to date of the effects of the methodological changes in 2002 (see **Appendix C**) indicate that the higher prevalences in 2002 mostly reflect an increase in the reporting of these behaviors by survey respondents due to the $30 incentive payment and other survey improvements, not actual increases in the prevalence of these behaviors and problems. The results of these analyses were presented to a panel of survey methodology experts, who concluded that 2002 estimates should not be compared with 2001 and earlier estimates. The panel also concluded that it would not be possible to develop a method of "adjusting" pre-2002 data to make them comparable for trend assessment.

Although traditional comparisons of estimates across years cannot be used to examine recent trends, it is possible to study trends by constructing "retrospective" estimates of lifetime prevalence and incidence produced from the 2002 NSDUH data alone (see **Chapters 5** and **6**). These trends can be compared with the results from Monitoring the Future (MTF), a study sponsored by the National Institute on Drug Abuse (NIDA). **Figure 10.1** shows the trends in lifetime marijuana use based on the 2002 NSDUH retrospective estimates for youths aged 16 to 20, as well as trends in lifetime marijuana use and past month marijuana use among the MTF 12th graders. The two data sources produce similar trends in lifetime prevalence, and the MTF trend in past month use also is similar to the trend for lifetime use. These trends also are consistent with trends for youths aged 12 to 17 and young adults aged 18 to 25 discussed in **Chapter 5**. They show very low rates of illicit drug use in the mid-1960s. In 1965, only 1.8 percent of youths had ever used marijuana. There were dramatic increases in use during the late 1960s and 1970s, and by 1979, 19.6 percent of youths had ever used marijuana. After that, use declined until 1991, when 11.5 percent of youths had ever used marijuana. The trend reversed during the 1990s, reaching 21.9 percent in 2001 before dropping slightly in 2002 to 20.6 percent.

Retrospective estimates based on 2002 NSDUH data are presented in **Table 10.1** for selected substances along with related estimates from the 2002 MTF for youths and young adults. The NSDUH data show decreases from 2001 to 2002 in lifetime use of marijuana, LSD, and cigarettes among youths, but an increase for cocaine among youths. For young adults aged 18 to 25 during this time period, there was a slight increase in lifetime cocaine and Ecstasy use and a decrease in lifetime LSD use. These NSDUH results are generally consistent with MTF trends, with a few exceptions. MTF shows no change in lifetime cocaine use among youths, and it shows decreases in youth Ecstasy and alcohol use not found in

the NSDUH estimates.

Estimates of incidence, or first-time use, also suggest that illicit drug use prevalence had been very low during the early 1960s, but began to increase during the mid-1960s as substantial numbers of young people initiated the use of marijuana. As discussed in **Chapter 6**, annual marijuana incidence increased from about 0.8 million new users in 1965 until it reached a peak of 3.5 million initiates per year during 1973 to 1978, just before the prevalence rates peaked. Interestingly, the annual number of marijuana initiates reached a low point in 1990 (1.6 million), then increased, 2 years before the increase in youth prevalence occurred. This finding demonstrates the value of analyzing the incidence data and using it to forecast future trends in prevalence. Assuming this relationship between incidence and prevalence continues to hold, the continuing high levels (between 2.5 and 3.0 million initiates per year) of marijuana incidence between 1995 and 2001 indicate that substantial declines in youth prevalence may not occur in the near future. However, the NSDUH incidence estimates for youths under age 18 indicate a decline from 2000 to 2001 (from 2.1 million to 1.7 million), which suggests that youth prevalence may decline. The NSDUH youth lifetime prevalence and MTF past month prevalence estimates do show decreases from 2001 to 2002. High rates of marijuana initiation during the 1970s among the cohort identified as the "baby boomers" have resulted in an increase in the numbers needing treatment for substance abuse problems. The increase in marijuana initiation rates during the 1990s may have the same result.

Figure 10.1 Marijuana Use among NSDUH Youths Aged 16 to 20 and MTF 12th Graders: 1975–2002

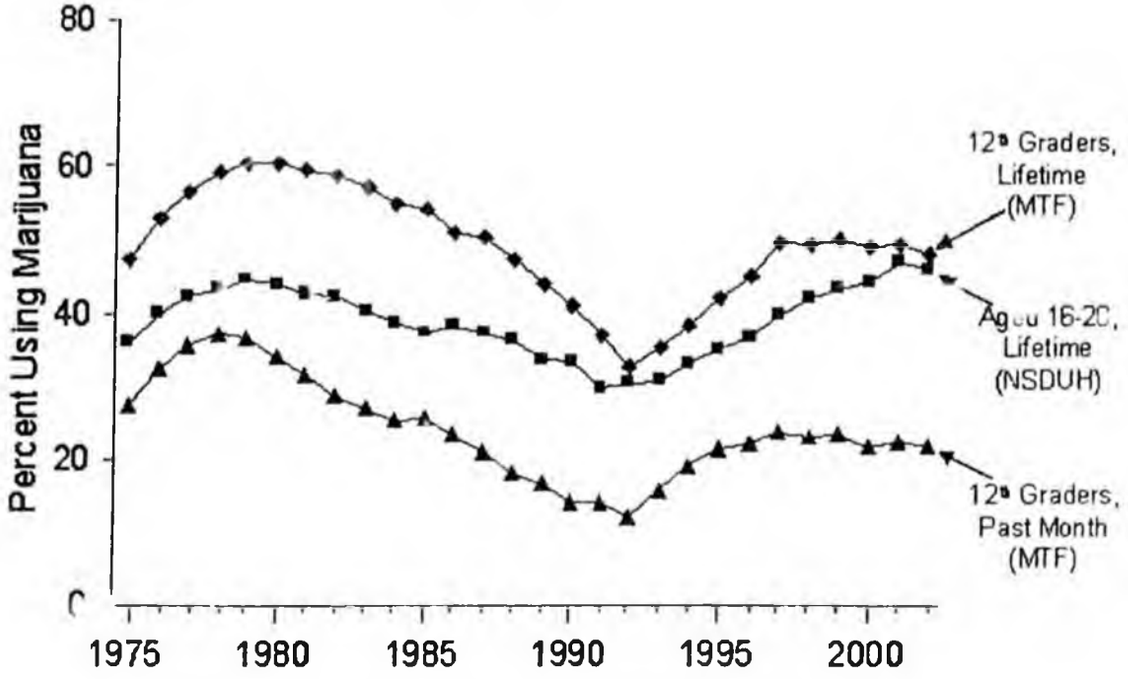

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Table 10.1 Comparison of NSDUH and MTF Prevalence Rates

	NSDUH 12–17		MTF 8th and 10th		NSDUH 18–25		MTF 19–24	
	2001	2002	2001	2002	2001	2002	2001	2002
Marijuana								
Lifetime	21.9	20.6	30.3	29.0	53.0	53.8	56.3	56.1
Past Month	--	8.2	14.5	13.1	--	17.3	19.6	19.8
Cocaine								
Lifetime	.3	2.7	5.0	4.9	14.9	15.4	12.4	12.9
Past Month	--	0.6	1.3	1.4	--	2.0	2.5	2.5
Ecstasy								
Lifetime	3.2	3.3	6.6	5.5	13.5	15.1	15.0	16.0
Past Month	--	0.5	2.2	1.6	--	1.1	2.2	1.6
LSD								
Lifetime	3.3	2.7	4.9	3.8	16.6	15.9	15.2	13.9
Past Month	--	0.2	1.3	0.7	--	0.1	1.0	0.4
Alcohol								
Lifetime	43.3	43.4	60.3	57.0	85.5	86.7	88.1	88.4
Past Month	--	17.6	30.3	27.5	--	60.5	67.1	67.7
Cigarettes								
Lifetime	37.3	33.3	44.7	39.4	71.3	71.2	--	--
Past Month	--	13.0	16.8	14.2	--	40.8	32.6	31.4

-- Not available.

Note: NSDUH data in this table are retrospective estimates from the 2002 data. MTF data for 8th and 10th graders are simple averages of estimates for those two grades reported in Johnston, O'Malley, and Bachman (2003b). MTF data for youths aged 19 to 24 are simple averages of estimates for youths aged 19–20, 21–22, and 23–24 reported in Johnston et al. (2003c).

Sources: SAMHSA, Office of Applied Studies, National Survey on Drug Use and Health, 2002.
The Monitoring the Future Study, University of Michigan, 2001 and 2002.

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[1] RTI International is a trade name of Research Triangle Institute.

Drug and Alcohol Services Information System

The DASIS Report

March 29, 2002

Treatment Referral Sources for Adolescent Marijuana Users

In Brief

- By 1999, more than half of all adolescent marijuana admissions were through the criminal justice system
- Adolescent marijuana admissions through the criminal justice system increased at a higher rate than admissions through other sources

Marijuana was the most common drug of abuse among admissions of adolescents aged 12 to 17 reported to the Treatment Episode Data Set (TEDS) in 1999. The number of adolescent marijuana admissions increased 260 percent between 1992 and 1999. TEDS collects data on nation admissions to primarily publicly f d substance abuse treatment faciliti "Admissions" represent annual treatment episodes rather than the number of individuals entering treatment.

Generally, adolescents entered treatment through the intervention of another person or agency. The Treatment Episode Data Set (TEDS) collects information on the referral source for people entering substance abuse treatment in the public sector. TEDS defines the primary referral sources as follows:

Court/criminal justice referral/DWI/DUI—police official, judge, prosecutor, probation officer or other person affiliated with a Federal, State, or county judicial system; court referral for DWI/DUI; referrals in lieu of or for deferred prosecution, during pretrial release, or before or after adjudication, pre-parole, pre-release, work or home furlough, TASC, or civil commitment

Self- or individual referral—self-referral; family member, friend, or other individual

School—school principal, counselor, or teacher; student assistance program, school system, or educational agency

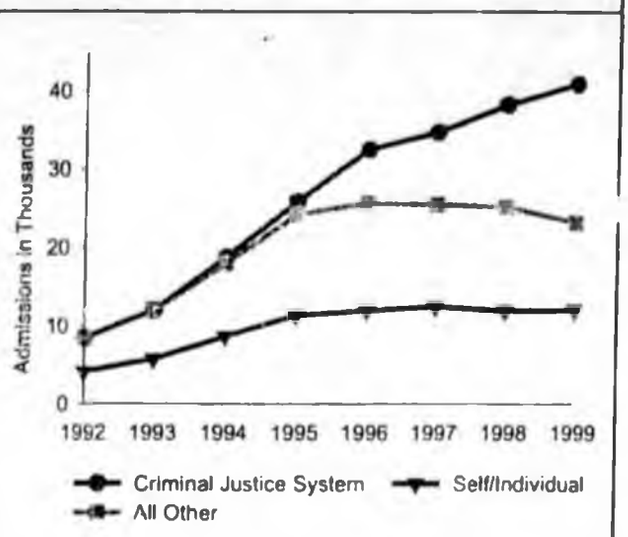
The DASIS Report is published periodically by the Office of Applied Studies, Substance Abuse and Mental Health Services Administration (**SAMHSA**). Other reports from the Office of Applied Studies are also available on-line: http://www.DrugAbuseStatistics.samhsa.gov/.

Table 1. Percent Distribution of Adolescent Marijuana Admissions, by Referral Source: 1992-1999*

	1992	1994	1996	1997	1998	1999
No. of Admissions (Thousands)	22	47	73	76	81	79
	Percent Distribution					
Criminal Justice	40.2	41.3	46.2	47.7	50.6	53.6
Self/Individual	19.6	19.0	17.1	17.2	15.9	15.9
School	16.3	16.0	13.2	12.1	11.3	10.5
SA/HC Provider	13.9	14.1	14.0	13.6	12.6	11.7
Other	10.0	9.5	9.4	9.3	9.3	8.3
Total	100.0	100.0	100.0	100.0	100.0	100.0

*1993 and 1995 not shown.
SA=Substance Abuse; HC=Health Care

Figure 1. Number of Adolescent Marijuana Admissions, by Referral Source: 1992-1999



Source: 1999 SAMHSA Treatment Episode Data Set (TEDS)

Other referral sources—substance abuse or health care providers; community, religious, or governmental organizations or agencies providing social services

Trends in Referral Source

In 1999, there were about 79,000 admissions for marijuana/hashish treatment among adolescents aged 12 to 17 (Table 1). This represented an increase from 22,000 admissions in 1992 and 47,000 in 1994. The most common referral source for adolescent marijuana admissions was the criminal justice system. Together with self- or individual referral, these sources account for 60 to 70 percent of all adolescent marijuana admissions in all years.*

In 1999, over half (54 percent) of all adolescent marijuana admissions were through the criminal justice system. This represented a significant increase from the 40 percent referred through the criminal justice system in 1992. The proportions of admissions from other referral sources declined. For example, from 1992 to 1999, self- or individual referrals declined from 20 percent to 16 percent, and referrals through schools declined from 16 percent to 11 percent.

The number of adolescent marijuana admissions increased from 1992 to 1995 for all referral sources (Figure 1). However, while the number of admissions from other referral sources stabilized after 1995, those from the criminal justice system continued to increase.

Referral Source by Sex

There was an overall increase in marijuana admissions for male adolescents from 1994 to 1999 attributable primarily to increased criminal justice referrals (Figure 2). While admissions for marijuana abuse increased by 67 percent from 1994 to 1999 among males, the percentage of those admissions due to criminal justice referrals increased by 108 percent. For females, marijuana admissions between 1994 and 1996 increased 67 percent and criminal justice referrals increased 111 percent. Between 1996 and 1999, however, there was only a 2 percent increase in female marijuana admissions but a 29 percent increase in criminal justice referrals.

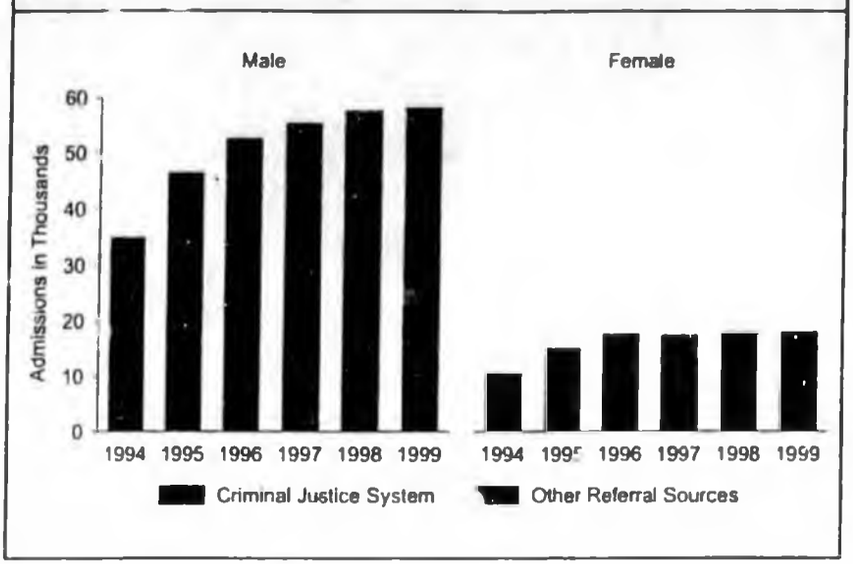
Among all adolescent marijuana admissions, there were three males for every female from 1994 to 1999. Among criminal justice referrals for marijuana abuse, the ratio was six males for every female in 1994. The ratio had declined to 4.5 to 1 by 1999 as more females were referred to treatment for marijuana abuse through the criminal justice system.

Referral Source by Age

Marijuana admissions for 12 to 14 year olds increased 56 percent

*Self-referral is the most common source for adolescent heroin admissions.

Figure 2. Number of Adolescent Marijuana Admissions, by Sex and Referral Source: 1994-1999



The Drug and Alcohol Services Information System (DASIS) is an integrated data system maintained by the Office of Applied Studies, Substance Abuse and Mental Health Services Administration (SAMHSA). One component of DASIS is the Treatment Episode Data Set (TEDS). TEDS is a compilation of data on the demographic characteristics and substance abuse problems of those admitted for substance abuse treatment. The information comes primarily from facilities that receive some public funding. Information on treatment admissions are routinely collected by State administrative systems and then submitted to SAMHSA in a standard format. Approximately 1.6 million records are included in TEDS each year. TEDS records represent admissions rather than individuals, as a person may be admitted to treatment more than once.

The DASIS Report is prepared by the Office of Applied Studies, SAMHSA; Synectics for Management Decisions, Inc., Arlington, Virginia; and RTI, Research Triangle Park, North Carolina.

Information and data for this issue are based on data reported to TEDS through April 16, 2001.

Access the latest TEDS reports at: www.DrugAbuseStatistics.SAMHSA.gov
Access the latest TEDS public use files at: www.icpsr.umich.edu/SAMHDA/teds.html

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Office of Applied Studies — www.samhsa.gov

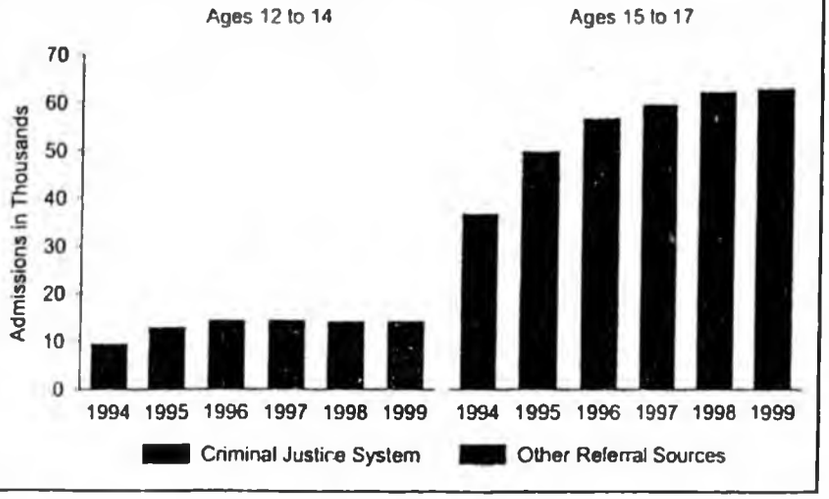
between 1994 and 1996, but decreased slightly (2 percent) from 1996 to 1999 (Figure 3). Marijuana admissions for 15 to 17 year olds increased by 54 percent between 1994 and 1996 but slowed to an 11 percent increase between 1996 and 1999. Criminal justice referrals among marijuana admissions for 12 to 14 year olds and 15 to 17 year olds have increased 21 and 27 percent, respectively, from 1996 to 1999.

Referral Source by Race/Ethnicity

Adolescent marijuana admissions averaged 66 percent White, 21 percent Black, and 13 percent Hispanic between 1994 and 1999 (data not shown). Each racial/ethnic group exhibited the same pattern over that time period—increased numbers of marijuana admissions driven by an increase in treatment referrals through the criminal justice system.

The numbers of Hispanic and Black adolescent marijuana admissions increased by 73 percent each, while the number of White admissions increased by 60 percent. Criminal justice referrals among Hispanic adolescents increased by 129 percent compared with 26 percent for referrals from other sources. Among White adolescents, criminal justice referrals increased by 119 percent compared with 26 percent from other sources. Black adolescents had the lowest percentage increase in criminal justice referrals for marijuana, 91 percent, while referrals from other sources for Black adolescents increased by 50 percent.

Figure 3. Number of Adolescent Marijuana Admissions, by Age Group and Referral Source: 1994-1999



FOR DEBATE

Reassessing the marijuana gateway effect

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ABSTRACT

Aims Strong associations between marijuana use and initiation of hard drugs are cited in support of the claim that marijuana use *per se* increases youths' risk of initiating hard drugs (the 'marijuana gateway' effect). This report examines whether these associations could instead be explained as the result of a common factor—drug use propensity—influencing the probability of both marijuana and other drug use.

Design A model of adolescent drug use initiation in the United States is constructed using parameter estimates derived from US household surveys of drug use conducted between 1982 and 1994. Model assumptions include: (1) individuals have a non-specific random propensity to use drugs that is normally distributed in the population; (2) this propensity is correlated with the risk of having an opportunity to use drugs and with the probability of using them given an opportunity, and (3) neither use nor opportunity to use marijuana is associated with hard drug initiation after conditioning on drug use propensity.

Findings Each of the phenomena used to support claims of a 'marijuana gateway effect' are reproduced by the model, even though marijuana use has no causal influence over hard drug initiation in the model.

Conclusions Marijuana gateway effects may exist. However, our results demonstrate that the phenomena used to motivate belief in such an effect are consistent with an alternative simple, plausible common-factor model. No gateway effect is required to explain them. The common-factor model has implications for evaluating marijuana control policies that differ significantly from those supported by the gateway model.

KEYWORDS Adolescents, drug use, marijuana gateway effect, mathematical model.

INTRODUCTION

Alcohol, tobacco and marijuana are widely regarded as 'gateway' drugs. Although the gateway concept admits a number of definitions, one in particular predominates in drug policy discussions: use of gateway drugs causes youths to have an increased risk of progressing to other, more serious drugs. For instance, in debates on marijuana decriminalization or the medicinal use of marijuana, policy makers frequently suggest that use of marijuana increases youths' risk of initiating more dangerous drugs such as cocaine and heroin (US Congressional Record 1998, 1999). Although marijuana is the least prevalent of the three principal gateway drugs, it is currently the focus of extensive policy reassessment in the United States, Canada, Western Europe and Australia. Using a simulation model, we demonstrate that the primary evidence supporting the marijuana gateway effect can be explained completely by the order in which youths first have the opportunity to use marijuana and other drugs, and by assuming a non-specific liability to use drugs, without any assumption that use of marijuana contributes to the risk of initiating use of hard drugs. We argue that although marijuana gateway effects may truly exist, available evidence does not favor the marijuana gateway effect over the alternative hypothesis that mari-

juana and hard drug initiation are correlated because both are influenced by individuals' heterogenous liabilities to try drugs.

The popular concern that marijuana use increases the risk of progressing to other, more serious drugs is a long-standing one, and has influenced US drug policy since at least the 1950s (Goode 1970; Whitebread & Bonnie 1972; National Research Council 1982). Some social scientists have also suggested that marijuana gateway effects probably account for several phenomena observed in adolescent drug use initiation patterns (e.g. Goode 1970; O'Donnell & Clayton 1982; Yamaguchi & Kandel 1984b). Three such phenomena represent the primary evidence for a marijuana gateway effect. The first concerns the *relative risk* of hard drug initiation for adolescent marijuana users vs. non-users. In general, marijuana users in many countries appear to have a significantly elevated risk for drug use progression (Adler & Kandel 1981; Kandel 1975; Blaze-Temple & Lo 1992; Stenbacka, Allebeck & Romelsjö 1993; Beenstock & Rahav 2002). Indeed, one US study found their risk to be 85 times those of non-users of marijuana (Center on Addiction and Substance Abuse 1994). Another form of relative risk that is occasionally cited in support of the gateway effect is that younger marijuana initiates have a higher risk of initiating hard drug use than older marijuana initiates (O'Donnell & Clayton 1982; Yamaguchi & Kandel 1984b; Kandel & Yamaguchi 1993; Center on Addiction and Substance Abuse 1994). This relative risk differs from the first only insofar as it finds that risk of hard drug initiation is conditioned on a characteristic of the user (age), rather than on marijuana use alone. Therefore, it does not provide strong evidence supporting a gateway effect.

The second observation routinely cited in support of the marijuana gateway effect concerns the remarkably invariant *ordering* in adolescents' initiation of different drug classes. Adolescents rarely initiate hard drug use before marijuana (Ellickson *et al.* 1992; Kandel, Yamaguchi & Chen 1992; O'Donnell & Clayton 1982; Yamaguchi & Kandel 1984a). For instance, in a longitudinal sample of 1265 New Zealand youths between the ages of 15 and 21, Fergusson & Horwood (2000) found only three cases reporting use of hard drugs before marijuana. This figure is dramatically lower than the roughly 124 such cases that would be expected from annual incidence rates if use of marijuana and hard drugs were independent.

The third phenomenon used to support claims of a marijuana gateway effect concerns the strong relationship between the frequency of marijuana consumption and the risk of hard drug initiation: as the frequency of marijuana use increases, so too does the risk of initiating hard drug use (Ellickson *et al.* 1992; Kandel *et al.* 1992; Fergusson & Horwood 2000). Fergusson & Horwood (2000), for instance, developed a proportional hazards model suggesting that youths reporting 50 or more uses of cannabis in the past year had hazards of progression to hard drugs that were more than 140 times greater than those for youths reporting no use of cannabis. Findings like this suggest an even stronger form of the marijuana gateway effect defined earlier: not only does marijuana use increase youths' risk of hard drug initiation, but every instance of marijuana use adds to that risk. For convenience, we refer to this phenomenon as marijuana's apparent *dose–response effect* on hard drug initiation.

The three phenomena of relative risk, ordering in drug use initiation and dose–response are not sufficient to prove that use of marijuana, rather than some associated factor, increases the risk of hard drug initiation (Joy *et al.* 1999). Indeed, a frequently cited alternative explanation is that a common factor, which we might refer to generically as a propensity for drug use, could influence use of both marijuana and hard drugs, thereby causing initiation of these drugs to be correlated (Goode 1972; Huba *et al.* 1981; Donovan & Jessor 1985; Hays *et al.* 1987; MacCoun 1998). For instance, if high drug use propensities elevate individuals' risk for use of both marijuana and hard drugs, this could explain why marijuana users have a higher relative risk of hard drug initiation in comparison with non-users.

This 'common-factor' model does not immediately account for the ordering and dose–response phenomena. To make sense of these observations, proponents of the common-factor approach suggest that ordering in drug use initiation results from the order in which opportunities to use marijuana and hard drugs are presented to young people (Goode 1972; Jessor & Jessor 1980). Those with the highest propensities to use drugs are likely to use the first one offered to them, and that happens to be marijuana in most cases. Moreover, if a high drug use propensity is associated with greater frequencies of drug use, the common-factor theory can also account for the dose–response phenomenon: marijuana use frequency is associated with risk of hard drug initiation because both are controlled by drug use propensity.

The common-factor model is appealing in part because it takes account of what is a substantial scientific literature demonstrating the existence of genetic, familial and environmental characteristics associated with a generalized risk of using both marijuana and hard drugs. For instance, several studies examining drug use among monozygotic and dizygotic twins in the USA demonstrate genetic and family environment contributions to the likelihood of any drug use (van den Bree *et al.* 1998) and any drug use initiation (Tsuang *et al.* 1998; Kendler *et al.* 1999, 2000). Similarly, community drug use or drug

availability may contribute to individuals' risk of using drugs (Lillie-Blanton, Anthony & Schuster 1993).

Although the common-factor model is plausible, previous research has not demonstrated that propensities to use drugs and environmental factors such as drug use opportunities could, in fact, account for the strong relative risk, ordering and dose–response phenomena observed among adolescents. Indeed, two lines of research provide some evidence that the common-factor model cannot account for drug use initiation without assuming a marijuana gateway effect. Firstly, several studies examine the association between marijuana use and the risk of hard drug initiation after controlling for a large number of risk factors, such as delinquency and peer drug use (Yamaguchi & Kandel 1984b; Fergusson & Horwood 2000). By the logic of this approach, any residual marijuana effect on hard drug initiation that remains after controlling for these candidate common factors lends credence to the suggestion that marijuana use *per se* increases the risk of hard drug initiation. However, if the selected covariates are less good proxies for the propensity to use drugs than is marijuana use itself, these findings are perfectly consistent with a strict common-factor model. Because this approach does not observe all or even most individual risk factors, it provides little persuasive evidence against a common-factor explanation. We will illustrate this point with data derived from the model described later on in this paper.

A second approach to contrasting the gateway and common-factor models of drug use initiation uses instrumental variables in an effort to account for both observed and unobserved person-level risk of initiation. Two of these studies (DeSimone 1998; Pacula 1998) suggest that common factors alone cannot explain observed gateway phenomena. The third (Beenstock & Rahav 2002) provides qualified evidence that observed marijuana gateway phenomena are not attributable to a gateway effect, but instead derive from individuals' predispositions to use both marijuana and hard drugs. However, none of these studies take into account the observation that opportunities to use marijuana precede those for hard drugs, and may themselves be associated with propensity to use drugs through, for instance, drug-seeking behavior. This is a critical omission, since proponents of the common-factor model have consistently cited the ordering in drug use opportunities as an essential part of the explanation of ordering in drug use initiation. Indeed, in a series of analyses on US and Panamanian data, Anthony, Van Etten and colleagues have shown that gender, race and neighborhood differences observed in rates of drug use initiation are attributable, to a large extent, to differences in the rates at which groups are exposed to drug use opportunities (Crum, Lillie-Blanton & Anthony 1996; Van Etten, Neumark & Anthony 1997, 1999; Deiva *et al.* 1999; Van Etten & Anthony 1999). Thus, econometric models have not tested the common-factor model adequately.

In this report we describe a Monte Carlo model of drug use initiation with parameters selected to match the drug use experiences of the population of US residents under the age of 22. The model describes the joint distribution of four events: the ages of first opportunity to use marijuana and hard drugs, and the ages of first use of marijuana and hard drugs. Each of these events depends on a common factor—drug use propensity—but conditional on this factor, the ages of first opportunity to use and first use of marijuana are independent of opportunity to use and use of hard drugs. Thus, the model is designed to exclude any causal gateway effect. Random draws from the modeled joint distribution are used to examine the relative risk, ordering and dose–response phenomena that might be expected by chance in the US if model assumptions are accurate.

METHODS

Procedure

We build a common-factor model of adolescent drug use initiation using parameter estimates derived from a representative sample of youths in the US population. With this model, we observe the rates at which phenomena of relative risk, ordering and dose–response can occur when no causal gateway effects are present. We compare these rates to those observed in the sample of US youths to demonstrate that a common-factor model designed to match US rates of drug use initiation and drug use opportunities without a gateway effect can still reproduce all of the gateway phenomena observed in the population. In the remainder of this section we describe the model specification and the statistical methods used to estimate the values for the model parameters and the gateway effects observed among youths in the US.

Model specification

Drug use propensity

Each case is assigned an arbitrary propensity to use drugs, θ, which we conceptualize as the resultant shared risk of reporting use of both marijuana and hard drugs after accounting for all person-level risk factors that remain more or less constant during adolescence. Examples of such invariant predispositions to report drug use could include genetic and family environmental history factors (Kendler *et al.* 1999; Tsuang *et al.* 1998; van den Bree *et al.* 1998; Kendler *et al.* 2000) and community

drug use or drug availability (Lillie-Blanton, Anthony & Schuster 1993). We assume that propensity is correlated not just with the probability of drug use, but also with the probability of having the opportunity to use drugs at any particular age. This assumption is supported by several considerations. Firstly, we define propensity as resulting, in part, from environmental risk factors. Local drug use norms and the availability of drugs are examples of environmental influences likely to affect both individuals' risk of drug use and their risk of having an opportunity to use drugs (Lillie-Blanton *et al.* 1993). Drug use propensity is also likely to be correlated with age of first opportunity to use drugs because individuals with greater propensities are more likely to seek out drug use opportunities, or recognize them when they present themselves. Finally, empirical studies document a strong association between the risk of drug offers (and, by extension, opportunities) and a range of characteristics likely to correlate with drug use propensity, such as smoking, alcohol use and parental substance use (Stenbacka *et al.* 1993). Each of these considerations suggests propensity will be correlated with drug use opportunities as well as drug use.

Although epidemiological studies provide evidence supporting the existence of a drug use propensity (Tsuang *et al.* 1998; van den Bree *et al.* 1998; Kendler *et al.* 1999, 2000), no information exists about its distribution in the population of adolescents. Thus, in the model we draw drug use propensities, θ, at random from a standard normal distribution.

Drug use opportunities

We assume that for any individual, the age of first opportunity to use marijuana, Y_M, and the age of first opportunity to use hard drugs, Y_H, are drawn at random from distributions describing the risk of first marijuana or hard drug use opportunity at each age. These risk distributions are functions of the individual's drug use propensity: higher propensities shift the risk curves so that exposure to a drug use opportunity is more likely at earlier ages. Thus, between the ages 0 and 22, we define the cumulative distribution of age of first opportunity to use marijuana as $1 - S_{YM}(t, \theta)$, where $S_{YM}(t, \theta)$ is the survival function describing the probability that age of first opportunity to use marijuana exceeds t, conditional upon θ. Similarly, the distribution of age of first opportunity to use hard drugs is given by $1 - S_{YH}(t, \theta)$. We use a frailty model to construct these conditional survival functions. Frailty models are a standard approach to describing joint survival functions when risks for each modeled event are presumed to correlate due to heterogeneity across individuals in the population (Hosmer & Lemeshow 1999; Therneau & Grambsch 2000):

$$S_{YM}(t, \theta) = S^{*}_{YM}(t)^{f(\theta)} \text{ for marijuana, and} \quad (1)$$

$$S_{YH}(t, \theta) = S^{*}_{YH}(t)^{f(\theta)} \text{ for hard drugs.} \quad (2)$$

The function f transforms θ to the corresponding value from the Gamma distribution with mean of 1 and variance β_1. Under this parameterization the frailty model produces a correlation between age of first use opportunities Y_M and Y_H that increases as β_1 grows. We estimate β_1 and the functions $S^{*}_{YM}(t)$ and $S^{*}_{YH}(t)$ from US data on adolescent drug use opportunities, as described below. The estimated functions are defined so that marginal survival functions for the model (expected values over θ of $S_{YM}(t, \theta)$ and $S_{YH}(t, \theta)$) equal marginal survival functions fit to our sample of US data.

Drug use initiation

For any individual, first use of marijuana, Z_M, is a random variable drawn from a distribution describing the individual's risk of initiating marijuana at each age. Each individual's risk distribution depends on his or her drug use propensity and age when first presented the opportunity to use marijuana, Y_M. Youths with greater propensities have a greater risk of use at every age, beginning with their age at first opportunity to use a drug.

Specifically, given an individual's drug use propensity and age of first opportunity to use marijuana, the cumulative probability distribution for age of marijuana initiation is given by $1 - S_{ZM}(t, \theta, Y_M)$, where $S_{ZM}(t, \theta, Y_M)$ is the conditional survival function for marijuana initiation. For $t = 8, 9 \ldots 22$,

$$S_{ZM}(t, \theta, Y_M) = \begin{cases} 0, \text{ if } Y_M > t \\ 1 - \pi_M, \text{ if } Y_M \le 8,\ t = 8 \\ F_{\beta_2}(\psi^{*}_{Mt} - \theta)\, S_{ZM}(t-1, \theta, Y_M), \\ \quad \text{if } Y_M \le t,\ t > 8. \end{cases} \quad (3)$$

where F_{β_2} is the cumulative probability function for a normal distribution with mean 0 and variance β_2. Thus, the parameters π_M and ψ^{*}_{Mt}, $t = 9 \ldots 22$ define for the model the marginal probabilities $Pr\{Z_M < 9 \mid Y_M < 9\}$ and the marginal probabilities $Pr\{Z_M = t \mid Y_M \le t,\ Z_M \ge t\} = E[F_{\beta_2}(\psi^{*}_{Mt} - \theta_i)]$, respectively. Age of initiation of hard drugs, Z_H, is drawn independent of Y_M and Z_M from an analogous distribution defined by the parameters β_1, π_H and ψ^{*}_{Ht}, $t = 9 \ldots 22$.

The value of β_2 is the same for both marijuana and hard drugs. This parameter affects the correlation between Z_M and Z_H by controlling the influence of propensity on the probability of initiation. It is chosen so that the model produces a correlation between $L_M = Z_M - Y_M$ and $L_H = Z_H - Y_H$ for youths who used both marijuana and hard drugs by age 22 that matches the same correlation observed in data on adolescents in the US. We set the remaining parameter values so that the marginal

probabilities in the model (i.e. $Pr(Z_M < 9 \mid Y_M < 9)$, $Pr(Z_H < 9 \mid Y_H < 9)$, $Pr(Z_M = t \mid Y_M \leq t,\ Z_M \geq t)$ and $Pr(Z_H = t \mid Y_H \leq t,\ Z_H \geq t)$, $t = 9 \ldots 22$) match the corresponding estimates from our sample of data from the US population.

The joint distribution for Y_M, Z_M, Y_H and Z_H is:

$$\begin{aligned}&Pr\{Y_M = y_M,\ Z_M = z_M,\ Y_H = y_H,\ Z_H = z_H\} = \\ &\int [S_{ZM}(z_M - 1,\ y_M,\ \theta) - [S_{ZM}(z_M,\ y_M,\ \theta)] \times \\ &[S_{YM}(y_M - 1,\ \theta) - S_{YM}(y_M,\ \theta)] \times \\ &[S_{ZH}(z_H - 1,\ y_H,\ \theta) - S_{ZH}(z_H,\ y_H,\ \theta)] \times \\ &[S_{YH}(y_H - 1,\ \theta) - S_{YH}(y_H,\ \theta)]]\ \phi(\theta) d\theta.\end{aligned} \quad (4)$$

where ϕ denotes the density function for a standard normal random variable.

Figure 1 depicts our procedure for drawing random observations from this distribution.

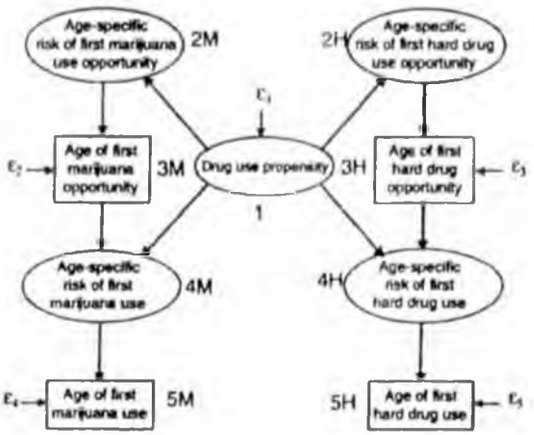

Figure 1 Procedure for generating simulated observations from the model of adolescent drug use initiation. Firstly, a random drug use propensity, θ, is drawn (1), which uniquely determines the risks (or probabilities) of having an opportunity to use marijuana, $S_{YM}(t, \theta)$, and hard drugs, $S_{YH}(t, \theta)$, at every age $t = 0$–22 (2M & 2H; note that epsilons in the figure are used to denote draws of separate, independent random variables from the indicated distributions). A random variable is then drawn from the distribution defined by S_{YM} to determine the age of first opportunity to use marijuana, Y_M, and an independent random variable is drawn to determine the age of the first opportunity to use hard drugs, Y_H (3M & 3H). The values of θ and Y_M then uniquely determine the risk of marijuana initiation at every age greater than Y_M, $S_{ZM}(t, \theta, Y_M)$, and similarly the values of θ and Y_H uniquely determine the risk of hard drug initiation for every age greater than Y_H, $S_{ZH}(t, \theta, Y_H)$ (4M & 4H). Another set of random variables that are independent of each other and all previous random variables are then drawn from the distributions determined by $S_{ZM}(t, \theta, Y_M)$ and $S_{ZH}(t, \theta, Y_H)$ to set age of initiation of marijuana, Z_M, and hard drugs, Z_H (5M & 5H). The additional parameters of the model were all determined to match the marginal distribution of a sample of US youths from the NHSDA

Marijuana use frequency

To examine the dose–response relationship between marijuana use frequency and the risk of hard drug initiation, we categorize each case that initiated use of marijuana into one of five past year use frequencies (no past year use, 1–2 times, 3–11 times, 12–51 times and 52 or more times) at each age, beginning with the age of marijuana initiation. Cases are assigned a marijuana use intensity random effect, ξ_u, which is used to draw a marijuana use frequency from the distribution of use frequencies observed in the US sample with corresponding ages and number of years since marijuana initiation.

We hypothesize that marijuana use frequency is positively correlated with propensity to use drugs. However, because propensity is unobservable, we know of no good data for estimating this correlation. Therefore, we conduct a sensitivity analysis in which the risk of hard drug initiation at each marijuana use frequency is examined as the correlation between ξ_u and θ, ranges from 0 to 1.

Parameter estimation

This section summarizes the statistical methods used to estimate values for each of the model's parameters, β_1, S^*_{YM}, S^*_{YH}, β_2, π_M, π_H and ψ^*_M, and ψ^*_H $t = 9 \ldots 22$. It also describes the methods used to estimate the observed values of the relative risk, ordering and dose–response effects from a sample of data from the US population.

Data source

Estimates for the model parameters and observed values of the relative risk, ordering and dose–response effects were derived from the National Household Survey of Drug Abuse (NHSDA). The NHSDA is an ongoing probability sample survey of the US civilian, non-institutionalized population aged 12 years and older (US Department of Health and Human Services 1999). Data on all 58 846 respondents, 12–25 years of age, from birth cohorts 1964 through 1982, were pooled from the 1982 through 1994-A NHSDA in order to create stable estimates of quite rare events. This pooling was justified by preliminary analyses suggesting that drug use opportunity and initiation survival probabilities were similar across birth cohorts. NHSDA sample weights were applied to make the pooled sample representative of the included birth cohorts. The selected survey years included questions on the ages of initiation and first opportunities to use marijuana, heroin, cocaine and hallucinogens. More recent data on drug use opportunities are not available because these questions were dropped from subsequent administrations of the NHSDA. First opportunity to use and initiation into use of hard drugs were defined as the earliest

reported age of opportunity and use of heroin, cocaine or hallucinogens. Because these data are self-reports of illicit behavior and improper events, they are subject to a variety of well-known biases. Nevertheless, longitudinal investigations indicate that ordering of drug use initiation, a central concern of the present analysis, is reported reliably (Golub *et al.* 2000). Therefore, for our purposes, recall bias is unlikely to significantly affect our principal findings.

Statistical methods

Estimation of β_1 To best match the correlation in ages of first opportunities to use marijuana and hard drugs observed in the NHSDA, we selected $\beta_1 = 3.22$, the maximum likelihood estimate of β_1 for model (1 & 2) fit to a 5% random sample of the NHSDA data, stratified by year of survey administration and birth cohort. The estimate was obtained using the S-Plus Software (MathSoft, Seattle, WA, USA) using the methods described in Therneau & Grambsch (2000).

Estimation of S^*_{YM} and S^*_{YH} The estimates of S^*_{YM} and S^*_{YH} derived directly from the marginal survival functions $S_{YM}(t)$ and $S_{YH}(t)$, which we estimate using data from the NHSDA. We used the actuarial life table method (Miller 1981) to estimate the survival function for first opportunity to use marijuana, $S_{YM}(t)$, defined as the probability that a randomly chosen individual's first opportunity to use marijuana occurs after age t. The actuarial life table method estimates the probability of a first opportunity to use at age t as the ratio of the number of individuals who report the first opportunity at age t to the number of individuals eligible to have a first opportunity at age t. Individuals are ineligible if they had a previous opportunity to use or if they are censored. Respondents are censored if they are interviewed before age t and report no opportunities to use prior to the interview. We used weighted sums in the ratio to account for unequal probability of selection in the NHSDA. The survival function at age t is obtained by multiplying the age-specific probabilities of an opportunity to use. We used analogous procedures to estimate a survival function for the first opportunity to use hard drugs, $S_{YH}(t)$, and survival functions for initiation of marijuana use, $S_{ZM}(t)$, and hard drug use, $S_{ZH}(t)$ (Fig. 2).

Because $f(\theta)$ is distributed as a Gamma random variable with mean one and variance β_1, $E[S^*_{YM}(t)^{f(\theta)}] = 1 - \beta_1 \log S^*_{YM}(t)^{-1/\beta_1}$ (Hogg & Craig 1978). Setting $E[S^*_{YM}(t)^{f(\theta)}]$ equal to the estimated marginal survival function for the NHSDA sample yields, $S^*_{YM}(t) = e^{(1-\hat{S}_{YM}(t)^{-\beta_1})/\beta_1}$. Again, a similar procedure is used to estimate S^*_{YH} from the estimates of β_1 and $S_{YH}(t)$.

Estimation of π_M and π_H Since $\pi_M = Pr\{Z_M < 9 \mid Y_M < 9\}$, we estimate this probability directly from the NHSDA as the sum of the weights from respondents who report marijuana initiation before age nine divided by the sum of the weights from respondents who report an opportunity to

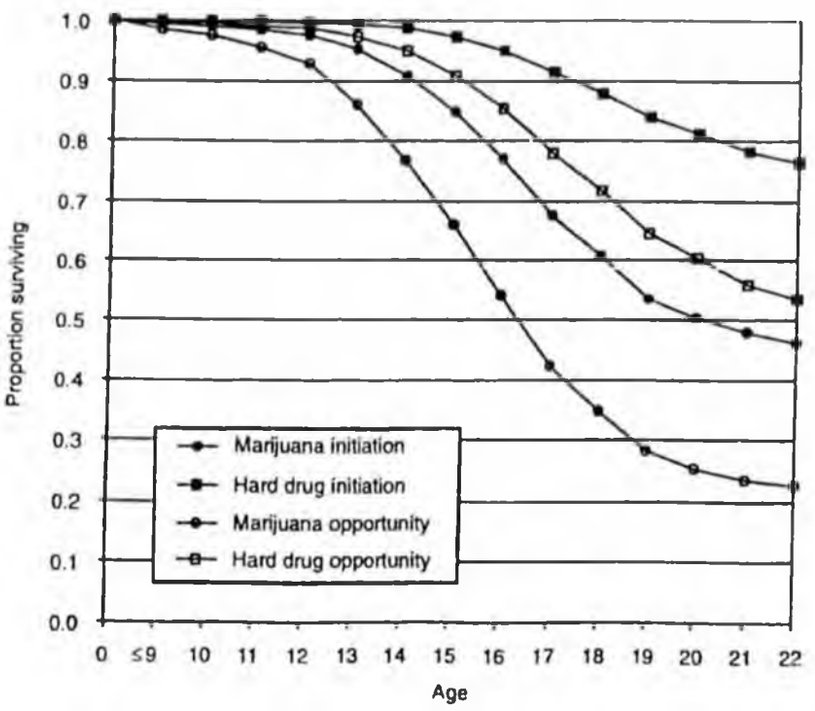

Figure 2 Survival functions estimated from the NHSDA describing the probability of surviving until age t without having the opportunity to use marijuana, the opportunity to use hard drugs, initiating marijuana and initiating hard drugs

use marijuana before age nine. The procedure is repeated for hard drugs.

Estimation of β_2 As noted earlier, we set β_2 so that the model correlation between L_M and L_H matches the NHSDA estimate. For all NHSDA respondents who reported using both marijuana and hard drugs by age 22, we calculated L_M and L_H and their correlation, r. We do not have a closed form for the correlation between L_M and L_H as a function of β_2, $\rho(\beta_2)$ in our simulation model. Therefore, we estimated the function via simulation. For a given value of β_2 we simulated 10 000 observations from the distribution and calculated the correlation between L_M and L_H. We then used the bisection method to search over the values of β_2 to find the value that solved $\rho(\beta_2) - r = 0$.

Estimation of ψ^*_{Mt} and ψ^*_{Ht} To estimate ψ^*_{Mt} we first estimate from the NHSDA $Pr\{Z_M = t \mid Y_M \leq t, Z_M \geq t\}$ as the ratio of the sum of the weights for respondents who initiate use at age t to the sum of the weights of respondents with first opportunity to use marijuana before age $t+1$ who did not initiate use prior to age t. We again did not have a closed form for $E[F_{\beta_2}(\psi^*_{Mt} - \theta_t)] = Pr\{Z_M = t \mid Y_M \leq t, Z_M \geq t\}$ as a function of ψ^*_{Mt} given a value for β_2. Instead we used the bisection method to find a value ψ^*_{Mt} so the $E[F_{\beta_2}(\psi^*_{Mt} - \theta_t)]$ from the simulation model equals our estimate of $Pr\{Z_M = t \mid Y_M \leq t, Z_M \geq t\}$ from the NHSDA. Values for the corresponding hard drug parameter were calculated in a similar manner.

Estimation of the relative risk effect We estimate the risk of hard drug use by 21 using the sample of NHSDA respondents aged 22 or older. We estimate the probability of hard drug use separately for respondents who reported marijuana use by 21 and those who did not. Estimates equal the weighted proportion by stratum. The relative risk is the ratio of the risk for hard drug initiation for marijuana users to the risk for others.

Estimation of ordering effect We used life table methods to estimate the rate at which hard drug initiation precedes marijuana initiation. For each age, t, we summed the weights for respondents who had used neither marijuana nor hard drugs by age t. Call this sum E_t. We subtracted from E_t, C_t the sum of the weights for respondents who were surveyed at age t and had used neither marijuana nor hard drugs—i.e. the weights for censored observations. Let U_t equal the sum of the weights for youth who report initiating hard drugs at age t before initiating marijuana use. We then estimate the probability that hard drug use preceded marijuana use at age t as $P_t = A_t U_t / (E_t - C_t)$, where A_t equals our estimate of the probability that a youth's first use of hard drugs or marijuana is after age t. The survival curve for initiating hard drug use prior to marijuana use equals $S(t) = 1 - \sum_{j=1}^{t} P_j$.

Estimation of the dose–response effect To examine the marginal dose–response effect of the simulated marijuana use frequency on the age of hard drug initiation, a generalized linear model with a complimentary log-log link (Hosmer & Lemeshow 1999) was fitted to the marijuana use frequency and hard drug initiation data from a random sample of 30 000 simulated cases. Whether or not a case initiated hard drug use in a given year was modeled as a function of past year marijuana use intensity (no past year use, 1–2 times, 3–11 times, 12–51 times and 52 or more times) at age t ($t = 12 \ldots 21$). Those who did not initiate hard drug use were censored after age 21.

To compute a corresponding hazard ratio for the NHSDA, we first selected the subset of respondents who reported no hard drug initiation prior to the year preceding the survey. We then stratified these respondents by age at the time of the survey. For each respondent age group we calculated the weighted proportion reporting initiation of hard drug use in the past year by past year marijuana use frequency. For example, we divided 12-year-old respondents into those who did not use marijuana in the past year, those who used marijuana 1–2 times, 3–11 times, 12–51 times or 52 or more times. Within each use group we estimate separately the proportion that initiated hard drugs. We repeat this for all other age groups. The resulting proportions define the hazard of hard drug initiation by age for each level of marijuana use. We assume that the five resulting hazard functions (one for each level of marijuana use) are age-specific, but that a single proportional hazards model describes the relative sizes of the five hazards at all ages. We compute the proportionality constants as the weighted average of the hazard ratios across ages in order to allow ages with less variability to have more weight in the calculation. This procedure for establishing dose–response hazards in the NHSDA is inaccurate, since marijuana use frequency could change after hard drug initiation in the past year. However, we use this NHSDA estimate only for purposes of comparison to analogous hazards observed in our modeled data, not to establish parameters for the model. For this comparison, our NHSDA dose–response estimates are sufficient.

MODEL RESULTS

We drew 1 000 000 observations from the joint distribution, from which three sets of outputs are recorded: simulated marijuana and drug initiation survival functions, $\hat{S}_{2M}(t)$ and $\hat{S}_{2H}(t)$; relative risk of initiating hard drugs by

age 21 for cases with and without prior marijuana initiation, and the percentage of simulated cases for which hard drug use preceded marijuana use by at least 1 year prior to age 22. Because the NHSDA and our simulated cases record age of drug use initiation in whole years, it is not possible to determine if hard drug initiation preceded marijuana initiation if both occurred in the same year. Therefore, we describe hard drug initiation as preceding marijuana initiation only if $Y_H < Y_M$.

Model precision

By design, modeled marijuana and hard drug initiation survival functions, $\hat{S}_{2M}(t)$ and $\hat{S}_{2H}(t)$, closely matched those for the US population shown in Fig. 2. Indeed, actual and modeled survival rates differed by 0.009 or less for each drug at every age.

Gateway effects

Relative risk

In our model, by age 21, users of marijuana were 157 times more likely than non-users to have initiated a hard drug. In comparison, respondents aged 22 or older in our NHSDA sample who initiated marijuana use by age 21 were just 24 times more likely than non-marijuana users to initiate hard drugs. Thus, our model produces a relative risk phenomenon even greater than that observed in the US data, even though the model incorporates no gateway effect. We attribute little significance to the fact that our model produces a relative risk substantially greater than that observed in the NHSDA. The denominator in this ratio is so small that even a slight imprecision in its estimate could more than account for the difference between observed and modeled risk ratios. Thus, for instance, if the true value of $P_r\{Z_H < 22 \mid Z_M > 21\}$ differs from our NHSDA estimate by as little as 0.013, the true relative risk could be greater than 190.

In addition, the elevated relative risk of hard drug initiation among younger marijuana initiates vs. older ones is also reproduced in the model. Among those aged 22 and older in our NHSDA sample, those who initiate marijuana by age 15 have 1.60 times greater risk of becoming a hard drug user by age 22 than those whose marijuana initiation occurs after age 15. Our model produces the larger, but still comparable, relative risk for these groups of 3.44.

Ordering

The proportion of simulated cases for which hard drug initiation preceded marijuana initiation was 0.011. This compares with the corresponding estimate of 0.016 from the NHSDA. Thus, initiation of hard drugs before marijuana was even more rare in our model than in the US household data, suggesting that no gateway effect is required to explain the strong ordering effect observed in youths' drug initiation experiences.

Dose–response

Hazard ratios for hard drug initiation among users of marijuana vs. those who did not use it in the past year are presented in Fig. 3. The figure exhibits a strong dose–response response relationship between marijuana use frequency and the hazard of hard drug initiation at each hypothesized correlation between the marijuana use intensity random effect, ξ, and propensity, θ. Indeed, even assuming zero correlation between these effects, a rising dose–response curve is found, with the heaviest users of marijuana having hazards of hard drug initiation more than 10 times greater than those of non-users.

The corresponding dose–response curve from the NHSDA data is plotted as a series of stars in Fig. 3. This curve bears a striking resemblance to those produced by the model. For the first two levels of marijuana use frequency the US data corresponds closely to the assumption that marijuana use frequency and drug use propensity have a moderate correlation ($r = 0.4$). For the highest marijuana frequencies, US hazard ratios fall between the moderate and high ($r = 0.8$) correlation assumptions.

DISCUSSION

Adolescent drug use initiation

The results reported here demonstrate that a simple common-factor model with population-based parameters can reproduce each of the phenomena previously used to support claims of a marijuana gateway effect. Thus, the strong relative risk, ordering and dose–response relationships observed between marijuana use and hard drug initiation do not require an assumption that marijuana initiation, or even the first opportunity to use it, increases the risk of either hard drug initiation or the opportunity to use hard drugs. While not disproving the existence of a marijuana gateway effect, our findings demonstrate that the primary evidence supporting gateway effects is equally consistent with an alternative model of adolescent drug use initiation in which use, *per se*, of marijuana has no effect on the later use of hard drugs.

Once a general propensity to use drugs is posited, the relative risk of hard drug use among marijuana users vs. non-users can be completely accounted for as a simple consequence of the fact that users of any drug are likely to have higher drug use propensities than non-users. Indeed, our model produced hard drug initiation risk

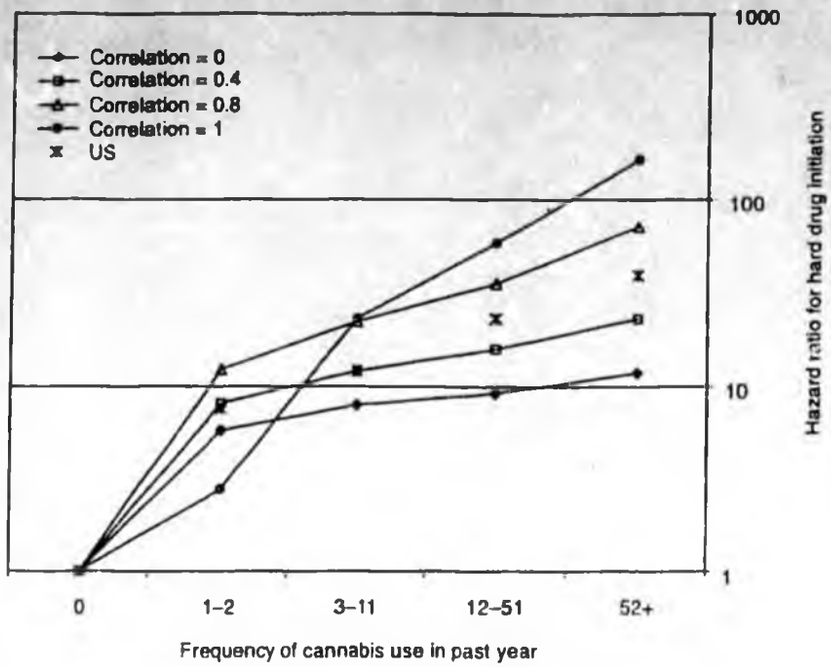

Figure 3 Hard drug initiation hazards, given past-year marijuana use frequency (expressed as a proportion of the hazard given no past year marijuana use) under four assumptions concerning the correlation between the marijuana use intensity random effect and the drug use propensity random effect

ratios greater than those observed in the NHSDA both for users vs. non-users of marijuana and for younger vs. older initiates of marijuana.

With the assumption that use of any drug is conditioned only on an individual's age, drug use propensity and opportunity to use drugs, the observed ordering in drug initiation can be attributed to the fact that opportunities to use marijuana routinely precede opportunities to use hard drugs—often by many years. Using just these assumptions, our model produced rates of hard drug use preceding marijuana use of just 11 per 1000 individuals, reflecting an even more invariant ordering than that found in our NHSDA sample, in which 16 of every 1000 individuals try hard drugs before marijuana.

Finally, even without the reasonable assumption of a correlation between marijuana use intensity and the more general propensity to use drugs, the assumptions of the model suffice to produce a strong dose–response relationship between marijuana use frequency and the risk of hard drug initiation. However, introducing such a correlation strengthens the dose–response relationship considerably. Indeed, as demonstrated by our sensitivity analysis, adjustments to the correlation between marijuana use intensity and drug use propensity suffice to account for the magnitude of the dose–response relationship observed for populations of youths. Again, the observed dose–response relationship between marijuana use frequency and the risk of hard drug initiation requires no marijuana gateway effect for its explanation.

Exhibiting gateway effects by controlling for common factors

Earlier studies have sought to support claims of a gateway effect by showing that marijuana use, *per se*, remains a powerful predictor of hard drug initiation, even after controlling for a wide range of candidate 'common factors' such as individuals' background characteristics, their risk behaviors and the behaviors of their peers (Yamaguchi & Kandel 1984b; Fergusson & Horwood 2000). This approach presumes that the included factors are sufficiently powerful indicators of any unobserved drug use propensity that their inclusion should eliminate any spurious appearance of a relationship between marijuana use and hard drug initiation. Since we know drug use propensities in our simulation model, we can examine the limits of this assumption using a random sample of cases drawn from our model. To do so, we construct variables that are more or less reliable indicators of drug use propensity, where the variances of the normally distributed error terms are used to control the correlation between the drug use propensity and its indicator. These indicators are next included as covariates along with a marijuana use indicator, m, in the following logistic model of hard drug initiation by age 22:

$$P_r\{Z_H < 22\} = \frac{1}{1+e^{-(\alpha+\beta m+\gamma X_1)}} \quad (5)$$

Figure 4 presents the hard drug initiation odds ratios for marijuana users vs. non-users, after controlling for drug use propensity indicators (X) with reliabilities ranging

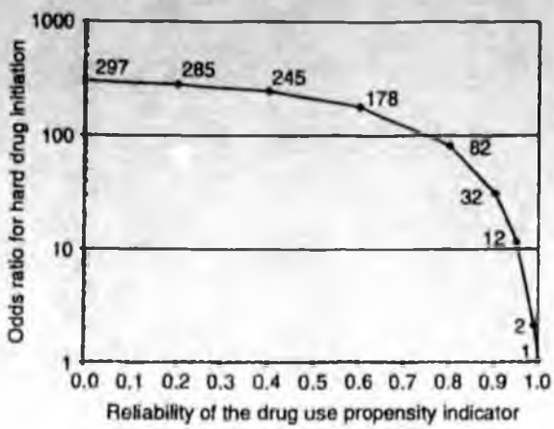

Figure 4 Odds ratios for initiating hard drugs given marijuana use (vs non-use), after controlling for presumptive 'common-factor' variables, as a function of these variables' reliability as indicators of true drug use propensity

between 0 and 1. This figure demonstrates that drug use propensity indicators need to be almost perfectly correlated with true drug use propensity before strong relationships between marijuana use and hard drug use are eliminated. Even when the indicator fails to capture just 2% of the variance in drug use propensity (i.e. its reliability is 0.99), marijuana users appear to have odds of initiating hard drugs that are twice as great as non-users of marijuana. Because it is very unlikely that the covariates included in prior studies have anything like a 0.99 correlation with drug use propensity, it is hardly surprising that controlling for these covariates does not eliminate the association between marijuana and hard drug use.

Model limitations

Several limitations and clarifications on the results are warranted. Firstly, our model relies on a number of untested assumptions and simplifications, such as a normal distribution for propensity and the frailty model for the joint distribution of age of first opportunity to use marijuana or hard drugs. To the extent these assumptions do not approximate corresponding phenomena in the population of youths, the model represents the process of adolescent drug use initiation less well. However, to the extent the model assumptions are plausible, we have demonstrated one possible process of drug use initiation that produces all of the gateway phenomena without requiring a gateway effect. The plausibility of our model is demonstrated through comparisons with estimates from the NHSDA. However, our estimates of the rate at which gateway phenomena occur in the NHSDA also depend on assumptions that may be wrong, like that the hazards of hard drug initiation for different marijuana use frequency groups remain proportional across age groups. If the assumptions are wrong, our NHSDA estimates will be biased and the comparisons provide less good evidence for the plausibility of our simulation model.

Secondly, we have produced a plausible model of adolescent drug use initiation that derives many of its parameter estimates from the NHSDA, a survey of US residents. However, it is quite clear that many of these parameters, like marijuana use prevalence, are specific to the population of youths in the US during the period in which the data used in this study were collected. As such, our estimates of the rate at which gateway effects occur in the NHSDA should not be expected to generalize to other places or times. Similarly, our model is calibrated to correspond to this US data, and might produce quite different results if parameter estimates from a different country or a different time were substituted for those estimates we used.

A third clarification concerns the possible effects of response bias on the appearance of gateway effects in this study, and every other study relying primarily on self-reports of drug use to demonstrate gateway effects. Suppose, for instance, that the likelihood of initiating hard drugs is, in fact, independent of whether or not someone has initiated marijuana or the frequency with which they use it. If there was a systematic under-reporting bias that led some marijuana users to claim to have never used either marijuana or hard drugs, or to under-report their marijuana use frequencies and their use of hard drugs, these biases could lead to the appearance of both the relative risk and dose–response gateway phenomena, although neither truly existed. If response bias accounts for the gateway phenomena, then the propensity factor we include in our model may correspond more to some heterogeneous response bias trait than it does to a true propensity to use drugs. It is for this reason that we have been careful to define propensity in terms of the likelihood of reporting drug use, rather than of engaging in drug use.

Fourthly, we constructed the model in such a way that use of hard drugs is independent of use of marijuana, except insofar as they share a common propensity to use drugs. This feature of the model holds true regardless of the particular values selected for drug use opportunities, use given opportunities or the correlation parameters. Therefore, even though the data set we use to derive these parameters might reflect the operations of a true gateway effect (the NHSDA), we can be certain that model's outputs do not result from any such effects.

The status of the marijuana gateway effect

The model and analyses described above do not disprove the gateway effect. Instead, they demonstrate that each

of the phenomena that appear to support such an effect are, in fact, equally consistent with a plausible alternative that accounts for the known general liability to use drugs and the known differences in when youths receive their first opportunities to use drugs.

Something like a marijuana gateway effect probably does exist, if only because marijuana purchases bring users into contact with a black market that also increases access to hard drugs (Goode 1970). However, this observation does not refute the analysis presented above, since there are at least two ways that gateway effects could exist without undermining a model of drug use initiation that fails to include them. Firstly, it is possible that any true marijuana gateway effects can explain only a tiny fraction of individuals' risk of hard drug use in comparison with the risk attributable to their propensities to use drugs, and is therefore a negligible factor in our model. A second possibility is that marijuana use could increase the risk of hard drug use for some youths, while decreasing the risk for others. As such, true marijuana gateway effects may be counterbalanced in the population by negative marijuana gateway effects, with the net effect of marijuana use on hard drug use being insignificant. Negative gateway effects could occur if, for instance, marijuana sated some youths' desires to experiment with illicit drug use, or if unsatisfying (or penalized) marijuana use experiences discouraged drug use progression among some youths.

The purported marijuana gateway effect is frequently invoked by policy makers as among the primary reasons to resist efforts to relax marijuana policies, such as permitting the medicinal use of marijuana (US Department of Health and Human Services 1999). Whereas social scientists often acknowledge that relative risk, ordering in drug use initiation and dose–response phenomena do not prove the existence of a marijuana gateway effect, they too have frequently drawn policy conclusions that presuppose such an effect. For instance, many have concluded that by postponing youths' marijuana initiation, prevention efforts will reduce the likelihood of hard drug use and abuse (Yamaguchi & Kandel 1984b; Kandel *et al.* 1992; Golub & Johnson 2001). Our model demonstrates how the observed correlations in the use of marijuana and hard drugs may be entirely due to individuals' propensity to use drugs and their opportunities to use them. As such, marijuana policies would have little effect on hard drug use, except insofar as they affected either an individuals' propensity to use any drugs (as might be the case with drug use prevention programs) or they resulted in hard drugs becoming less available or available later in youths' lives.

Because our model provides a straightforward, parsimonious and plausible explanation for each of the phenomena used to support claims of a marijuana gateway effect, we believe the validity of that effect must remain uncertain until new evidence is available directly comparing it with the alternative common-factor model.

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STEPPING STONE TO OTHER DRUGS?

The stepping stone theory holds considerable sway in debates on marijuana. In fact, the concern is that cannabis use leads to the use of other drugs, in particular, the so-called hard drugs, such as heroin and cocaine.

> *It logically follows that more people using drugs will increase the number of people being harmed by them. Cannabis is believed to be the foundation upon which most young people begin experimenting with illicit drugs. (...) The "gateway" concept has been around for a long time, and again, although there is no definitive evidence, the National Institute on Drug Abuse has reported that neuro-toxicological research suggests that marijuana "may alter the brain in ways that increase the susceptibility to other drugs." Many believe that cannabis use provides the impetus for those people looking to increase the psychotropic effect a drug has on them.* [51]

We should first define our terms. The "stepping stone" theory holds that cannabis use inevitably leads to use of other drugs. In this theory, cannabis use would lead to neurophysiological changes, affecting in particular the dopaminergic system (also called the reward system), thus creating the need to move on to the use of other drugs. This theory has been completely dismissed by research. We share this conclusion with several international bodies doing drug research, including the British organization *DrugScope*.

> *The Stepping-Stone theory has proved unsustainable and lacking any real evidence base. The "evidence" that most heroin users started with cannabis is hardly surprising and demonstrably fails to account for the overwhelmingly vast majority of cannabis users who do not progress to drugs like crack and heroin. The Stepping-Stone theory (often confused among the general public for the Gateway theory) has been dismissed by scientific inquiry. The notion that cannabis use "causes" further harmful drug use has been, and should be, comprehensively rejected.* [52]

The "gateway" theory suggests that users' trajectories offer them choices as they start their trajectory of use and that one of these choices is to use other drugs. According to this theory, certain factors, such as early initiation and more regular and heavier use, reinforce this possibility. However, these factors themselves, and early initiation to cannabis in particular, are related to earlier factors, arising from the family environment and social living conditions, that predispose the more vulnerable young people to this early initiation and more rapid progress towards regular and heavy use.

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[51] M. J. Boyd, Chair of the Drug Abuse Committee and Deputy Chief of the Toronto Police Service, Canadian Association of Chiefs of Police, testimony before the Special Senate Committee on Illegal Drugs, Canadian Senate, first session of the thirty-seventh Parliament, Issue 14, page 75.

[52] DrugScope (2001) *Evidence to Home Affairs Committee Inquiry into Drug Policy.* Available on-line at: http://www.drugscope.org.uk/druginfo/evidence-select/evidence.htm

The link between cannabis and other drug use, according to this explanation, is thus a reflection that there are a number of risk factors and life pathways that predispose young people to use cannabis and that they overlap with the life pathways that predispose young people to use other illicit drugs.[53]

In addition to these factors that predispose some young people to heavier use of psychoactive substances – including alcohol and tobacco first of all – the sociological conditions under which users can obtain cannabis are such that they are in contact with an environment that is at least marginal if not criminal. Dealers are often the same people who also sell heroin, crack, amphetamines, cocaine and ecstasy such that the probability that a young cannabis user, already more vulnerable due to the factors of his personal trajectory, would come into contact with these other substances more easily. We would also add that wholesalers and dealers "cut" or even mix their products; we were told at times that ecstasy, for example, could contain many things other than MDNA.

Furthermore, if it is true that use of substances such as heroin and cocaine develops almost necessarily out of prior use of marijuana, then it also develops out of the use of other substances, nicotine and alcohol in particular, which are more gateways to a trajectory of use than cannabis.

If we come back to trends in drug use in the population, while more than 30% have used cannabis, less than 4% have used cocaine and less than 1% heroin.

However, it is true that regular and heavy users are more likely than occasional users to use other substances. The study by Cohen and Kaal[54] discussed in the previous section shows for example that more than 90% of long-term cannabis users have also used tobacco and alcohol during their lifetime. Above all, it also shows that 48% in Amsterdam and 73% in San Francisco have used cocaine at least once in their life, and 37% in Amsterdam, 77% in San Francisco and 47% in Bremen have used hallucinogens at least once. Nevertheless, no regular cannabis users were regular users of other substances. The authors also show that the most common sequence is alcohol (around age 14), tobacco (around age 15), cannabis (around age 17), followed by other drugs in the early 20s.

We feel that the available data show that **it is not cannabis itself that leads to other drug use** but the combination of the following factors:

- Factors related to personal and family history that predispose to early entry on a trajectory of use of psychoactive substances starting with alcohol;
- Early introduction to cannabis, earlier than the average for experimenters, and more rapid progress towards a trajectory of regular use;
- Frequenting of a marginal or deviant environment;
- Availability of various substances from the same dealers.

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[53] *Ibid.*

[54] Cohen and Kaal, *op. cit.*, page 92-93.

CANNABIS, VIOLENCE AND CRIME

It is clear that there is some association between psychoactive substances and crime. It is just as clear that this link is much more complex than is sometimes thought, as Professor Brochu pointed out during his testimony before the Committee.

> *Just in my office at the Université de Montréal, I have 2,973 studies that attempt to show a link between psycho-active substances and crime. Most of these studies come from the United States or from English-speaking countries, which tends to colour their perspective somewhat, since we know that our neighbours to the south have very clearly opted for a punitive approach to illegal drugs. What comes out of all these studies is that the link between drugs and crime is very complex.*[55]

Since his testimony, Professor Brochu has released the study he mentioned to the Committee.[56]

We can examine the drug-crime relationship from at least three angles: the effects of the substance itself, the effects of the cost of the substance, and the drug's position in the criminal world.

A significant proportion of offenders have psychoactive substance abuse problems, predominantly with alcohol. In fact, the study concludes that alcohol is the substance most frequently associated with violent crime; in the case of crimes against property, illegal drugs predominate. Cannabis ranked third (3% to 6% according to the study), far behind alcohol (24%) and cocaine (8% to 11%).

With respect to the second approach, the authors establish that between 17% and 24% of inmates committed a crime to obtain the money needed to buy their substance of choice, most often cocaine.

Lastly, regarding the third approach, because illegal drugs are marginalized, users are exposed to a deviant environment. In the previous section we noted that, with regard to cannabis, the fact that dealers can offer heroin or crack as well as cannabis could promote a gateway trajectory towards these other drugs. Similarly, the fact that these substances are illegal could contribute to leading people to a trajectory of delinquency. Furthermore, the drug trafficking environment is a relatively violent environment where a whole series of crimes are committed. Lastly, the simple fact of selling cannabis is itself a criminal offence, and we know that a certain number of people are imprisoned for doing so.

All in all, cannabis itself does not lead to a trajectory of delinquency and it is more likely to be the other way around: someone who embarks on a trajectory of delinquency

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[55] Professor Serge Brochu, Université de Montréal, testimony before the Special Senate Committee on Illegal Drugs, Canadian Senate, First Session of the Thirty-Seventh Parliament, December 10, 2001, Issue 12, page 18.

[56] Brochu, K. et al., (2002) *Proportions of crimes associated with alcohol and other drugs in Canada.* Ottawa: Canadian Centre on Substance Abuse.

when young is exposed to illegal drugs more quickly and can experiment at a younger age and begin a career as a user when younger.

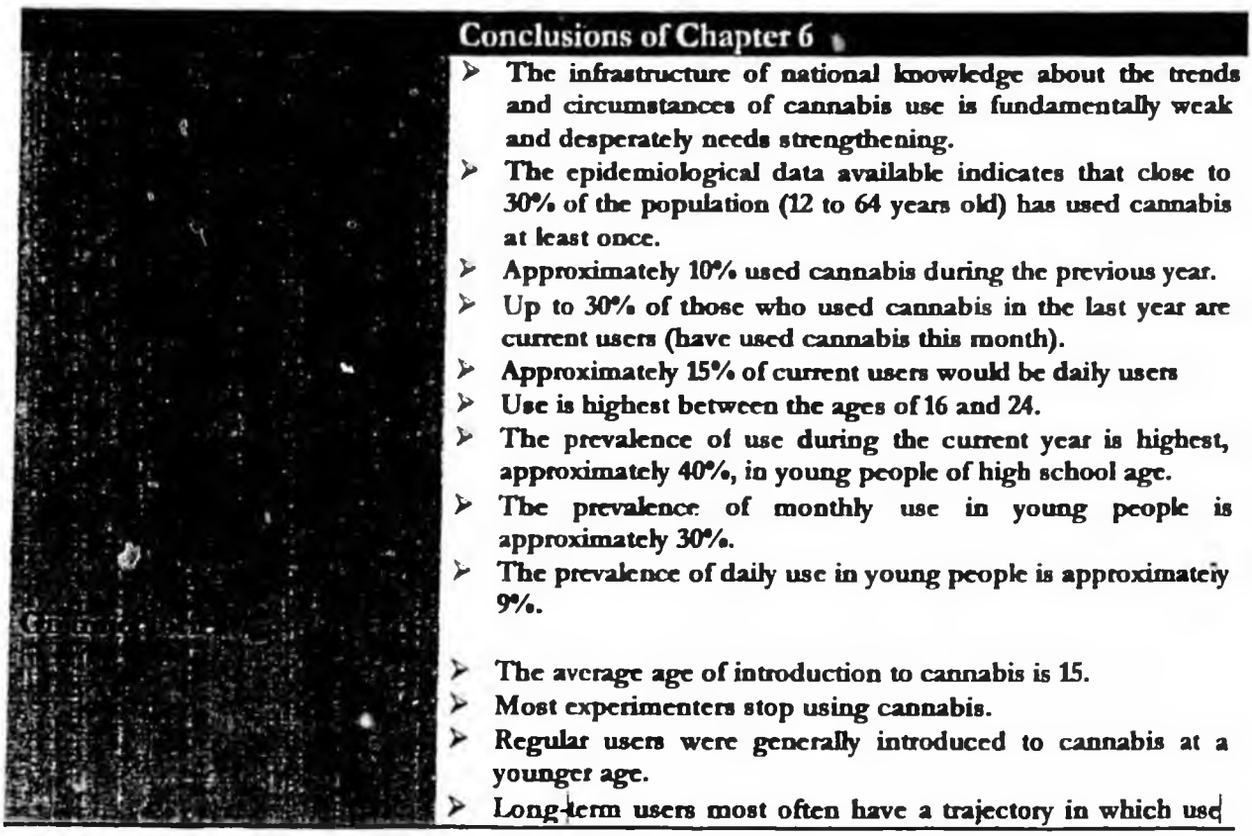
Furthermore, simply because of its relaxing and euphoristic psychoactive effects and its effect of relaxing muscle tone, cannabis is hardly likely to lead to acts of violence.

Data from studies on long-term users confirm this global picture of the relationship between cannabis and crime. Thus, Cohen and Kaal noted that less than 5% of their respondents had committed offences to obtain cannabis (pilfering, shoplifting, theft). The offence committed most frequently in order to obtain cannabis was selling it.

In short, the Committee has learned that cannabis is not a cause of violence or crime except in rare cases, and of course excluding driving while under the influence, which will be dealt with in Chapter 8.

CONCLUSIONS

We have learned the following from all the information on trends, patterns, circumstances, trajectories and social consequences of cannabis use:



rises and falls.

- Long-term regular users experience a period of heavy use in their early 20s.
- Most long-term users integrate their use into their family, social and occupational activities.

- Cannabis itself is not a cause of other drug use.
- Cannabis use can be a gateway because it is illegal, which puts users in contact with other substances.

- Cannabis itself is not a cause of delinquency and crime.
- Young people with a trajectory of regular and heavy use are often already on a deviant if not delinquent trajectory.
- Cannabis is not a cause of violence.

9
Social Problems

The most heated debates about marijuana prohibition often concern the drug's role in social problems. Ideally, drug laws should minimize the negative impact of illicit substances. Arguments for marijuana's illegal status often rely on perceptions of the social problems it might cause, including decreased productivity, dangerous driving, and uncontrollable aggression. If cannabis created such adverse effects, strong penalties for its possession, sale, and use would seem warranted. Popular publications imply that marijuana's role in amotivation, reckless driving, and aggression is a proven fact (e.g., Drug Watch Oregon, 1996; Indiana Prevention Resource Center, 1998; National Institute of Drug Abuse [NIDA], 1998). Yet data reveal that cannabis plays little role in any of these social problems. Details of the relevant studies appear below.

Amotivational Syndrome

Overview

Proponents of marijuana prohibition express concern about the drug's long-term impact on motivation. Despite data to the contrary, stereotypes suggest that regular users of cannabis, particularly adolescents, transform into apathetic slugs uninterested in school, work, or any productive activity (Nahas, 1990). Researchers first identified a subset of lethargic, unmotivated cannabis smokers over 100 years ago (IHDC, 1894). Yet these data did not prove that marijuana actually altered motivation. By the late 1960s, investigators coined the expression "amoti-

vational syndrome" to describe indifferent, listless adolescents who smoked marijuana. Yet the tacit assumption that cannabis drained their motivation was never tested.

Educators and parents grew particularly worried that marijuana destroyed ambition in the young. Case studies suggested that amotivational syndrome included poor hygiene and depressed mood, as well as a loss of energy, productivity, and drive. Authors repeatedly emphasized that the syndrome included an absence of clear goals or focused effort. Researchers suggested that repeated exposure to the drug created this condition, perhaps through a negative effect on the central nervous system (McGlothlin & West, 1968; Smith, 1968). Despite evidence to the contrary, concern about marijuana's influence on motivation continues today (NIDA, 1998). The primary problems with research on amotivational syndrome concern defining its symptoms and proving marijuana actually causes them.

Defining Amotivation

Vague definitions and varied measurements of amotivational syndrome have led to compelling critiques of the idea. Some investigators have examined employment history and educational achievement; others look at performance on laboratory tasks. Yet all claim to measure motivation or amotivational syndrome. Nearly all measurement strategies reflect stereotypically Western values about productivity. Many researchers tacitly assume that motivated people perform well in school, work hard for their employers, and persevere on laboratory tasks. Yet some of the world's most famous achievers failed in these domains. People do not share the same goals or value the pursuit of objectives in the same way. Some cultures emphasize future plans over a focus on the present. Others clearly do not. In fact, the intense pursuit of future goals may minimize enjoyment of the present moment, leading to considerable distress (Burke, 1999).

The notion of amotivational syndrome can inadvertently pathologize behaviors that many people in other cultures find fulfilling (Morningstar, 1985). One culture's amotivational syndrome may be another culture's ideal lifestyle. For example, vacation time varies dramatically from country to country, reflecting different attitudes about leisure and productivity (Robinson, 1994). In addition, motivation and achievement do not

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necessarily lead to happiness or increased satisfaction in life. The idea of amotivational syndrome may present a false promise that accomplishments lead invariably to happiness.

Even within Western culture, the definitions of amotivational syndrome vary considerably. There is no formal diagnosis or established list of symptoms. Most researchers employ their own unique measures of motivation, making comparisons between studies difficult. Reports usually describe amotivation as a subtle shift in priorities. Achievement becomes less important; leisure becomes more important. Sufferers purportedly have few long-term goals or no concrete plans for attaining them. They may lose the ability to concentrate, endure frustration, and participate in life. If a marijuana-induced amotivational syndrome does exist, its symptoms do not sound similar to the obvious problems associated with the abuse of other drugs. Chronic cannabis users rarely report the drastic financial, social, and occupational difficulties typical of addiction to alcohol, opiates, or cocaine. Nevertheless, if marijuana created an absence of drive, it would clearly interfere with the steady achievement stereotypically associated with the American dream.

The purported symptoms of amotivational syndrome are hardly unique to cannabis use. Clinical depression often includes the fatigue, poor concentration, and apathy typical of amotivation. This overlap suggests that a subset of depressed people who use marijuana may account for clinical observations of amotivational syndrome. Sad, unmotivated people may happen to smoke cannabis, giving the impression that the drug has created the symptoms. In fact, the links among depression, amotivation, and marijuana consumption are not particularly straightforward.

Recent data reveal that cannabis consumption has no significant association with depression in adults. A subset of people who use marijuana to cope with problems show more depressive symptoms, but it is not clear that the cannabis caused their depression. People who first tried marijuana before age 16 showed more depression later in life. Yet this relationship disappeared when the use of other drugs was taken into account (Green & Ritter, 2000). A separate study revealed that measures of motivation correlated more with depression than with marijuana consumption, even among heavy users (Musty & Kaback, 1995). Thus, depression, rather than cannabis, may cause amotivational symptoms.

Cannabis as a Cause

The idea that marijuana diminishes motivation requires the same firm evidence of association, temporal antecedence, and isolation discussed in chapter 3 with the gateway effect. Marijuana must precede and correlate with amotivation to cause it. The symptoms also must not stem from some other contributor like personality, depression, or the use of another drug. Ensuring that amotivational syndrome arises from cannabis requires experiments. Researchers can randomly assign people to receive cannabis or placebo. This arrangement ensures that everyone is equally likely to end up in the group that smokes marijuana, assuring that any identified deficits arise from cannabis rather than personality, depression, or other drug use.

In an alternative approach, participants work after smoking a placebo and at other times after smoking cannabis. This strategy, known as a "within-subjects design," ensures that all participants work both intoxicated and sober. Investigators can then compare each person's intoxicated performance to his or her own work in the absence of the drug. Under these circumstances, any identified impairment must stem from cannabis. Thus, laboratory experiments can rule out alternative explanations for marijuana's impact on motivation. This type of research requires extensive time, effort, and funding. Cannabis use over many days should produce the lethargy and lack of ambition typical of the disorder. Only a few laboratory experiments provide enough data from repeated daily exposure to provide any meaningful conclusions.

Laboratory Performance

In one of the first studies of chronic cannabis administration, researchers employed 6 men to build chairs for 70 days. They earned $2 per chair initially, but went on strike twice and raised their fees. They had periods without cannabis, and weeks when they could purchase as much as they wanted, at $.50 per joint. For 28 days, the researchers required that they smoke at least 2 joints containing a total of 17 mg of THC. Generally, the men built fewer chairs and worked fewer hours when required to consume cannabis. They also built fewer chairs immediately after they went on strike and increased their wages. The men showed no other signs of amotivation.

This study clearly supports the idea that intoxication can decrease

productivity (Miles et al., 1974). Yet it is unclear if this would qualify as evidence for amotivational syndrome. Arranging for a strike to increase wages likely required motivation, organization, and drive. Making fewer chairs might reflect lower motivation, but it more likely offers further evidence that intoxication impairs performance.

In another study of chronic administration, researchers paid 30 men to stay in the hospital for 94 days. They ingested no drugs for the first 11 days, smoked cannabis for the next 64, took a break from the drug for a week, used cannabis daily for 9 more days, and then did not smoke the last 3 days. The men smoked an average of 5.2 joints per day when the researchers permitted consumption. They were paid for daily work on two different tasks. One required adding large numbers on a calculator. The other required answering textbook questions. Participants received $.10 for each correct answer on these two tasks. Acute intoxication and chronic exposure had no impact on any measure of performance. The men showed statistically comparable total responses, total correct responses, errors, and time worked throughout the 94-day period (Cohen, 1976). These data offer no support for amotivational syndrome.

These long-term studies offer little support for cannabis-induced losses of productivity. Thus, proponents of marijuana prohibition often cite other research that demonstrates decreased motivation during intoxication. One standard way to manipulate motivation in the laboratory requires offering extra cash for good performance on tasks. In one study of marijuana's effects, researchers attempted to increase motivation and performance on simple tasks by offering financial incentives. On a reaction-time task, intoxicated people did not respond to this incentive as dramatically as the people who had not smoked cannabis. Offering extra money did not motivate people to react more quickly while high, but it did speed reaction times for people who were not intoxicated. The authors emphasize that this result offers little support for amotivational syndrome. Instead, these data mean that intoxicated people do not react to standard techniques for enhancing motivation (Pihl & Sigal, 1978).

Two other studies performed in a residential laboratory revealed that intoxicated men were less likely to perform tasks that they disliked (Foltin et al., 1989, 1990). After smoking marijuana, these men spent less time on work and chores and more time on recreational activities. Popular articles often refer to these studies as evidence for amotivational syndrome. Perhaps intoxication decreases a person's willingness to work

on unappealing projects, but this effect hardly parallels the directionless apathy typical of most definitions of amotivation. If these results qualify as evidence for amotivational syndrome, then most psychoactive drugs could serve as a cause. In fact, anything that might create procrastination, including watching television, could serve as a source of amotivation.

Because laboratory studies of humans offer little evidence for amotivational syndrome, critics point out that the duration of exposure was relatively brief. A couple of months of chronic smoking may not lead to any symptoms, but those who believe in amotivational syndrome charge that the disorder does not appear until later. Few people would participate in a study for a longer period, but animal research has examined the impact of 12 months of drug exposure. In this hallmark study, researchers randomly assigned 62 adolescent male monkeys to inhale cannabis smoke or a placebo. The dosage was similar to 4 or 5 marijuana cigarettes for a human. Some received smoke daily, others only on weekends, for a full year. This arrangement permitted an examination of the impact of long-term exposure to cannabis on motivation and performance.

After the year of exposure, all the monkeys performed two tasks daily for two months. The tasks were typical for primate research. One measure, conditioned position responding, provided the monkeys with a banana-flavored pellet for pressing different buttons in response to different colors. Marijuana had little impact on correct responses. The monkeys who smoked cannabis performed as well as the placebo smokers on the rate of responding and the percentage of correct responses. A subset of monkeys who had smoked every day did not complete as many trials on this task as the placebo smokers, but only during the last month of the experiment. Those who smoked cannabis only on weekends did not differ from the controls on any measure. Thus, conditioned position responding offers little evidence for amotivational syndrome, even after a year of cannabis exposure.

Proponents of marijuana prohibition might argue that conditioned position responding did not require much motivation in the first place. Essentially, the task may have been too easy to show any amotivational effects. Fortunately, the investigators also used a more difficult measure, the progressive ratio (PR) task. In the PR task, the monkeys must press a bar an increasing number of times to receive pellets. If the monkey presses the bar 3 times to get the first pellet, he has to press it 6 times

to get the second pellet, 9 times to get the third pellet, and so on. The number of required presses progresses upward after each reinforcer.

Among a subset of monkeys who practiced the tasks during their year of exposure to smoke, those who received cannabis did not perform as well as those who received placebo. This deficit even appeared for the monkeys who only smoked on weekends. In contrast, the group who did not practice showed the same performance whether they had smoked cannabis or not. That is, those who smoked cannabis but did not practice the task performed equally as well as those who smoked placebo and did not practice the task. Thus, the progressive ratio task showed marijuana-related deficits in monkeys who had practiced the task during the year, but not in the unpracticed monkeys. This result may mean that monkeys who practiced while intoxicated did not learn the task as well as the monkeys who practiced while sober. The responses returned to normal within 3 months of abstinence from cannabis, when all groups performed equally well (Slikker, Paule, Ali, Scallet, & Bailey, 1992).

This study of primates shows decreased performance on a difficult exercise after a year of marijuana use, but only in those who had practiced the task during their exposure to cannabis. The drug had no impact on the easier, conditioned position responding. The investigators did not report changes in hygiene, mood, or other symptoms of amotivational syndrome. This study and some of the human research certainly confirms that intoxication can impair performance on some tasks in some conditions. Nevertheless, this seems like rather slim evidence for full-blown amotivational syndrome. Yet many critics dismiss this laboratory evidence as irrelevant. The term often implies a failure to achieve in life, not simple deficits on laboratory tasks. To further test the role of cannabis in motivation, other investigators have examined marijuana's correlation with educational and work performance. Impairments on these life tasks appear more relevant to the idea of amotivational syndrome.

Correlations with Education and Work

Surveys of associations between drug use and job or school activities lack the experimental control found in the chronic administration studies. Investigators can only assume that marijuana use causes poor performance at work or school. Alternative explanations remain equally tenable. For example, poor adjustment in work or school might lead some

people to use cannabis. A third factor may account for the association, too. Depressed people might perform poorly and choose to use cannabis. People with certain personality characteristics might choose to use marijuana and make school or work a low priority. Thus, a simple association between cannabis consumption and education or work does not prove that amotivational syndrome exists. Nevertheless, the absence of an association between marijuana and achievement might undermine arguments for cannabis-induced amotivation. It is extremely unlikely that the drug causes amotivational syndrome if use and performance do not correlate. Therefore, these studies put the theory behind amotivational syndrome at risk for refutation.

School Performance

Parents and educators express understandable concern about marijuana, amotivational syndrome, and schoolwork. Research has focused on academic achievement in college and high school students. Contrary to popular belief, over half a dozen studies reveal that marijuana smokers and nonsmokers have comparable grades in college. One typical report surveyed 1,400 undergraduates, revealing no differences between users and nonusers on grades, changes in their majors, or number of colleges attended. Chronic users (those who smoked at least 3 times a week for 3 years) took more time off from their schooling but were also more likely to plan to earn a graduate degree (Hochman & Brill, 1973).

Surprisingly, at least two other studies found higher grades in the marijuana smokers than in nonsmokers (Gergen, Gergen, & Morse, 1972; Goode, 1971). Note that, despite these findings, no one has ever proposed that cannabis could help school performance. Users and nonusers also show no differences in their orientations toward achievement, their extracurricular activities, or their participation in sports. Thus, research on college students provides no support for the idea of amotivational syndrome (Zimmer & Morgan, 1997).

Although cannabis consumption in college has no link to school performance, high school students who use marijuana have lower grades and quit school more often. Cannabis smokers in high school also spend less time on their homework and miss more days of school (Kandel & Davies, 1996). At first glance, this association between cannabis and school performance seems consistent with the idea of amotivation. Perhaps cannabis destroys motivation in young teens. Yet data do not support this

restricted form of amotivational syndrome, either. Most heavy users had earned lower grades prior to their marijuana consumption, suggesting cannabis could not have caused the poorer performance (Shedler & Block, 1990). In addition, high school students who smoke cannabis heavily also tend to use alcohol and other illicit substances. Once these factors are taken into account, the link between cannabis and academic performance disappears. These results suggest that drugs other than marijuana might lower grades (Hall, Solowij, & Lennon, 1994).

Marijuana probably does not cause poor school performance. Instead, the regular consumption of cannabis in high school serves as part of a general pattern of deviance. Heavy users appear more unconventional in general. They are more critical of society, less involved in church and school, and more involved in delinquent acts. They often behaved this way before they ever discovered cannabis (Donovan, 1996). Because these young people showed these qualities before using marijuana, the drug seems an unlikely cause of amotivational syndrome in high school students. Thus, depressed, unmotivated, unconventional adolescents may choose to smoke marijuana, but the drug does not appear to create their deviance. Despite this evidence, concerns about drug use in adolescents inspired the National Organization for the Reform of Marijuana Laws to recommend that only adults consume cannabis (NORML, 1996a).

Employment

Two contradictory attitudes have developed about marijuana's impact on job performance. Many people believe the drug destroys motivation and detracts from efficiency, yet others use the drug to enhance their work. Both ideas may be true, depending on the type of job involved. People who perform repetitive, simple tasks may turn to cannabis to relieve the boredom. For example, laborers in India increased their ganja consumption 50% during the harvest season (Chopra & Chopra, 1957). In Jamaica, farm hands who smoked marijuana actually worked harder than those who did not (Comitas, 1976). Perhaps marijuana makes monotonous physical labor more bearable. In contrast, jobs that require complex or rapid decisions likely suffer during intoxication (Chait & Pierri, 1992). Thus, the acute effects of cannabis on performance may vary dramatically with different jobs.

The enduring lack of initiative that defines amotivational syndrome requires more than brief changes in work performance during intoxica-

tion. Wages, hours, and employment history may serve as better indices of motivation on the job. Research performed in countries where workers frequently smoke cannabis has shown little difference between heavy users, occasional users, and abstainers. These groups had comparable forms of employment in Costa Rica and Jamaica (Bowman & Pihl, 1973; Carter, 1980). In Costa Rica, users were unemployed more often than nonusers, probably because of imprisonment for marijuana offenses. Nevertheless, heavy users had better-paying, higher-status jobs than occasional users or abstainers. People with the most stable employment smoked 15.4 joints per day. Those who changed jobs more often smoked less than half that amount, 7.6 joints per day. The unemployed smoked even less- 6.2 joints per day (Page, 1983). Perhaps people with steady employment have enough experience on the job to function properly while intoxicated and enough money to afford marijuana.

In the United States, where cannabis consumption is less prevalent, the impact of the drug on wages, hours, and job turnover still does not support the idea of amotivational syndrome. Data actually suggest some positive links between marijuana consumption and work, but only for adults. One survey of more than 8,000 young adults who held a variety of jobs showed higher wages with increased use (Kaestner, 1994a). People who had smoked more marijuana in their lifetimes earned more money. Note that this correlation does not imply that cannabis consumption actually causes better pay. Perhaps people who earn more money can afford more marijuana. Another report from the same respondents revealed a negative correlation between consumption and work hours for men. Those who smoked more worked fewer hours. Yet given that their wages were higher, they may have become more efficient at work. Hours and consumption did not correlate significantly for women (Kaestner, 1994b).

Other studies of employment histories and drug use reveal that marijuana smokers do not appear to lose their jobs more often than nonsmokers, even though employers are more likely to fire users of other illicit drugs (Normand, Salyards, & Mahoney, 1990; Parish, 1989). One study of over 10,000 military personnel found that cannabis users were discharged more often (McDaniel, 1988). This result may not actually address amotivation because possession of cannabis can serve as a reason for discharge. Some of these recruits may have performed perfectly but lost their jobs because of possession. This effect did not replicate in a survey of navy recruits, which revealed that cannabis users were dis-

charged at the same rate as others (Blank & Fenton, 1991). The respondents in these studies were all over age 18, so these data do not address amotivation in adolescents. Nevertheless, cannabis consumption does not appear to have a dramatic negative impact on wages, hours, or job turnover in adults.

Self-Perceptions of Motivation

A few studies have used the direct and intuitive approach of asking users their perceptions of marijuana's impact on their motivation. This research did not assess the many hypothesized facets of amotivational syndrome, such as lethargy, poor hygiene, and impaired social functioning. Yet these studies do reveal that a percentage of heavy users think that the drug saps their ambition or drive. Interpreting these results requires caution. Many of these participants used illicit drugs besides marijuana. They also could have suffered from unassessed conditions that undermined their energy or motivation. Nevertheless, members of every sample believe that the drug makes them less ambitious or dynamic. In one of the first studies of this kind, researchers interviewed 99 New Yorkers by phone. These people had used marijuana an average of 27 out of the previous 30 days. Eleven of these people (11%) reported reduced energy and motivation. Yet alcohol consumption was not reported, and almost half of the sample used an illicit drug other than marijuana. The report does not reveal if these 11 people who reported less energy also used other illicit drugs, but they clearly attributed their lack of motivation to cannabis (Rainone, Deren, Kleinman, & Wish, 1987).

In another study, investigators interviewed 37 Americans who claimed to have smoked marijuana at least 5,000 times. Three of these heavy users (8%) said that cannabis had a negative impact on their work because it attenuated motivation. In contrast, 7 people (19%) said that the drug enhanced their creativity and improved their work (Gruber, Pope, & Oliva, 1997). The investigators purposely excluded people who used other drugs extensively, which may explain why they found the lowest rate of reported problems with motivation.

Other researchers interviewed 268 Australians who had smoked marijuana at least 3 times per week for the previous 10 years. Over one-fifth of them (21%) felt that cannabis made them tired, unmotivated, or listless. It is unclear if they experienced these symptoms during intoxication

or afterward. The use of other drugs in this sample was quite high, which may have contributed to perceptions of decreased motivation. Almost one-third (30%) of the respondents reported problematic consumption of alcohol, a sedative that lowers motivation and energy. Almost one-fourth (24%) had used an illicit drug besides cannabis in the previous month (Reilly, Didcott, Swift, & Hall, 1998). Thus, despite heavy drinking and the use of other drugs, these heavy cannabis users still reported marijuana altered their motivation. Yet the consumption of these other drugs may have sapped their drive instead.

Note that none of the studies above had a control group that could reveal if people who do not smoke cannabis also struggle with their productivity, enthusiasm, or drive. Many people feel tired, unmotivated, and low in energy without using any drugs at all. Perhaps marijuana smokers misattribute these symptoms to the drug. They may experience the natural ebb and flow of energy that all people feel, but consider this variation a result of marijuana. For example, a study of 237 students found that roughly 5% showed amotivational symptoms whether they used cannabis or not (Duncan, 1987). These results cast doubt on the idea that marijuana attenuates motivation. Instead, a percentage of people at any given time report motivational problems regardless of their drug use. Some of these people smoke cannabis and therefore attribute their lack of motivation to the drug. Yet tenable alternative sources of these problems may get overlooked because of expectancies about marijuana.

In another study, occasional marijuana users served as the control group for a sample of heavy smokers. The 44 occasional users never smoked more than 10 times in a month. The 45 heavy smokers used cannabis daily for at least 2 years. Heavy smokers also consumed more illicit drugs than occasional users, which may account for some differences between the groups. The groups did not differ in mental health, anxiety, depression, emotional control, or happiness. Yet the heavy smokers reported that marijuana was more likely to impair their motivation. The result was statistically significant, but the investigators did not correct for the large number of variables that they examined. Thus, this finding may have appeared by chance. If it did not appear by chance, then heavy users think that marijuana impairs their motivation more than occasional users. Oddly, despite the potential deficit in motivation, heavy users reported a trend toward greater life satisfaction. Again, the investigators did not correct for the large number of comparisons, so this find-

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ing may also stem from chance. Nevertheless, these results suggest that heavy users are less motivated but more satisfied with their lives (Kouri, Pope, Yurgelun-Todd, & Gruber, 1995). Perhaps they have rejected the conventional notion that motivation and productivity are essential for fulfillment.

Because the data from all these studies are correlational rather than longitudinal, they do not reveal if the heavy smokers reported poor motivation prior to ever using cannabis. Perhaps people who do not make productivity a priority subsequently choose to use marijuana. It is also unclear if the consumption of other drugs undermined productivity. Users may attribute their decreased ambition to marijuana when other drugs may have created the effect. The fact that the one study that specifically excluded users of other drugs found one of the smallest rates of motivation problems (8%) (Gruber et al., 1997) supports this idea. Mental or physical illnesses may have contributed to these symptoms, too. Nevertheless, it is clear that a percentage of people who use cannabis at high rates feel that the drug impairs their motivation.

Summary

Laboratory studies of humans and primates offer little support for amotivational syndrome. School performance does not vary with cannabis consumption in college students. High school students who smoke marijuana do worse in school. Nevertheless, most performed poorly before they used cannabis, and many used other drugs that probably contributed to their lower grades. Employment data show no links between cannabis use and lower wages, poor work performance, or job turnover. Self-reports in heavy users show that a percentage of people think cannabis impairs their drive, but consumption of other drugs or the presence of physical and emotional problems may serve as the true cause of their lack of motivation.

No studies show the pervasive lethargy, dysphoria, and apathy that initial reports suggested should appear in all heavy users. Thus, the evidence for a cannabis-induced amotivational syndrome is weak. Yet a subset of depressed users may show the symptoms of amotivational syndrome (Musty & Kaback, 1995). These people would likely benefit from cognitive-behavioral treatments for depression, which can improve mood, motivation, and achievement.

SB

74

TESTIMONY AGAINST (FILE 2)

Testimony to Senate Health and Social Services Committee
April 1, 2005

My name is Bill Parker. I am a former member of the Alaska House of Representatives, and I retired from state service as Deputy Commissioner of Corrections. Today I speak for Alaskans for Marijuana Regulation and Control.

S.B.74 attempts to recriminalize marijuana for adults in Alaska, in violation of the Alaska Constitution.

In 1975, in a landmark case known as Ravin, the Alaska Supreme Court ruled the privacy clause of the Alaska Constitution protects possession of a small amount of marijuana by adult Alaskans in their own homes for their own use. In 1975, the Alaska Legislature changed the statutes to decriminalize marijuana in Alaska.

Various attempts have been made in the 30 years since to attack this de-criminalization, both legally and politically.

An initiative in 1990 attempted to re-criminalize marijuana in Alaska, but initiatives change statutes, not the Constitution, and the initiative had no effect.

Many legal attempts have been made to test the constitutionality in the courts, all unsuccessful. The latest was last fall when the Alaska Supreme Court declined to take up the Appellate Court's latest ruling upholding Ravin.

S.B.74 is another attempt to attack the constitutional issue in a legal and political manner.

If S.B.74 passes with these findings, they will be admissible in court, and the administration will have new arguments that marijuana is much more potent and dangerous than in 1975, so much so that it is almost a different substance.

That is why the findings section of this bill is important. The findings are flawed. Expert witnesses from Alaska and Outside are going to explain those flaws today.

They will discuss the complex medical and sociological issues that other government panels have studied at length.

- The Shafer Commission's report to President Nixon in 1972, "Marijuana: Signal of Misunderstanding."
- The National Research Council's 1982 report, "An Analysis of Marijuana Policy."
- The Institute of Medicine's 1999 report, "Marijuana and Medicine: Assessing the Scientific Base."
- The 2002 report of the British Advisory Council on the misuse of drugs, "The Classification of Cannabis."
- The House of Commons Home Affairs Committee 2002 study, "The Government's Drug Policy: Is It Working?"
- Jamaica's 2001 National Commission on Ganja came to the same conclusion;

  Marijuana is not so harmful that the penalties for possession need to be increased.

S.B.74 would take Alaska in the opposite and wrong direction.

The State's witnesses could not be called objective observers. Those directly involved in implementing an administration's policies cannot evaluate those policies impartially. Evaluators should be independent academics.

The testimony today will show that the time schedule alone for S.B.74 is inadequate to evaluate marijuana in Alaska. We have submitted, in writing, the findings of experts in their fields who determine marijuana to be relatively harmless compared to alcohol. Each finding must be examined individually as the other commissions and committees have done with scientific integrity.

Here is a quick review of the evidence you will hear today:

Experts will point out the differences between scientific research and pseudo-science, the confusion between correlation and causation.

The administration's assertions about increased potency of marijuana are inaccurate and misleading in several respects:

- There are serious questions about the actual potency of marijuana today and yesterday. There is no reliable way to measure potency.
- There is no proof that marijuana is more addictive or dangerous than previously.
- In fact, more potent marijuana would result in people using less, because of the effect of autotitration.

The administration's treatment of statistics is misleading because most of their conclusions are court-ordered, not a clinical diagnosis of marijuana addiction or even a self-referral. Most had to choose between treatment or incarceration. Most chose treatment.

The rate of marijuana use among minors in Alaska is no higher today than it was in 1975. In fact, according to the government's own statistics, overall use in grades 6 through 12 in Alaska schools is lower now after 30 years of decriminalization.

Marijuana use by minors has not been shown to cause psychosis later in life.

Marijuana use does not induce violent behavior, rape, or child abuse.

The emergency room data used to show that marijuana is more dangerous today is not conclusive. The relation to marijuana in patients is so widely construed as to be meaningless. And the administration has overstated and misinterpreted the evidence of marijuana's link to lung cancer, juvenile crime, and the possibility of addiction and dependence.

The weight of scientific evidence available today discredits the old 'gateway drug' theory.

There are laws already in place to prohibit driving while impaired by alcohol or marijuana. These laws will remain in effect.

S.B.74 would have a bad effect on medical marijuana patients by jeopardizing their ability to possess marijuana if adult use of marijuana in the home is criminalized.

If the administration's aim is to promote the public health and welfare, re-criminalizing personal, adult use of marijuana in the home won't do it. Re-criminalization will only feed the black market and increase the social costs that flow from it.

Science shows marijuana causes far less harm to the public health and welfare than alcohol or tobacco. And that's as true today as it was in 1975.

Shafer Commission, 1972

(National Commission on Marihuana and Drug Abuse), Commissioned by President Richard M. Nixon

- Commission members included 4 MD's, 2 PhD's, 1 theologian, and 5 elected officials
- 26 staff professionals, including additional MD's and PhD's, assisted the commission
- 49 support staff, analysts, and researchers were involved
- More than 1 year was spent in its investigations and preparation of its report

National Research Council of the National Academy of Science, 1982

"An Analysis of Marijuana Policy"

- 4 years of effort
- Included a review of the 1972 National Commission on Marijuana and Drug Abuse report
- Conclusion: "On balance, we believe that a policy of partial prohibition is clearly preferable to a policy of complete prohibition."

Institute of Medicine, 1999

"Marijuana and Medicine: Assessing the Science Base"

- Division of Neuroscience and Behavioral Health, IOM
- Took testimony from hundreds of doctors, scientists, treatment professionals, and many others
- Worked from 1997 to 1999
- Conclusions included:

"Compared to most other drugs...dependence among marijuana users is relatively rare."

"There is no conclusive evidence that marijuana causes cancer in humans, including cancers usually related to tobacco use"

"It does not appear to be a gateway drug"

"Earlier studies purporting to show structural changes in the brains of heavy marijuana users have not been replicated with more sophisticated techniques."

Report of the National Commission on Ganja, Jamaica, 2001

- "nine months of consultation and reflection,
- "visits to every parish and hearings amounting to 3776 pages of transcriptions"
- "The Commission is persuaded that the criminalisation of thousands of people for simple possession for consumption does more harm to the society than could be done by the use of ganja itself."

Advisory Council on the Misuse of Drugs, Great Britain, 2002

"The Classification of Cannabis under the Misuse of Drugs Act 1971"

- "…based on a detailed scrutiny of the relevant scientific literature, including four reviews commissioned by the Department of Health in 1998, as well as an update commissioned by the Home Office and completed in November 2001"
- "The Council … recommends the reclassification of all cannabis preparations to Class C [least harmful]."

House of Commons Home Affairs Committee, 2002

"The government's drugs policy: Is it working?"

- "We have taken oral evidence from 45 witnesses over a total of 11 evidence sessions."
- "…more than 200 people and organisations…provided written submissions…"
- "We support, therefore, the Home Secretary's proposal to reclassify cannabis from Class B to Class C."

Report of the Canadian Senate Special Committee on Illegal Drugs, 2002

"Cannabis: Position for Canadian Public Policy"

- "product of a team effort over a period of 2 years"
- The Parliamentary Research Branch synthesized and analyzed literature on legal studies and socio-criminological studies. In all, the Committee received 23 reports.
- Heard testimony from expert sociologists and lawyers, psychologists and physicians, police officers and criminologists
- The Committee held more than 40 days of public hearings…more than 100 persons from all backgrounds.

Even the most recent commission reports echo the findings of many earlier commissions, including the LaGuardia Commission in 1944, and going back to the British Indian Hemp Drugs Commission in 1894.

The independent reports have been astonishingly consistent for well over a century:

"...continued criminalization of cannabis remains unjustified based on scientific data on the danger it poses."

--Final Report of the Canadian Senate Special Committee, 2002

Alaska Civil Liberties Union

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Testimony of Michael W. Macleod-Ball, Executive Director, Alaska Civil Liberties Union
Senate Health, Education and Social Services Committee
Friday, April 1, 2005

RE: SB 74 – *An Act making findings relating to marijuana use and possession...*

Chairman Dyson, members of the committee:

Thank you for providing us this opportunity to present evidence countering the administration's misleading characterization of marijuana as a substance that has grown dangerous over the last 30 years.

As the administration has acknowledged, both before this committee and in public statements, the purpose of this bill is to provide a framework for overturning the Alaska Supreme Court's decision that our constitutional right to privacy includes the right to possess and use small amounts of marijuana in the home for purely personal purposes. The bill also significantly steps up the penalties in a manner to bring the bill directly into conflict with existing case law. The administration hopes that a legislative finding concerning marijuana's risks will be sufficient for the court to reverse itself if and when this bill is enacted and then challenged in court. Because this legislation directly impacts a fundamental right – the right to privacy – the ACLU believes that any legislative finding not reflective of the weight of evidence available for review will be set aside by the courts. Alternatively, if this committee takes on the heavy burden of weighing the available science fully and completely, and produces findings that are reflective of the weight of the evidence, we believe the court would consider such balanced findings an insufficient basis for justifying a restriction on the constitutional right to privacy. The ACLU believes that drug policy based on imposing criminal sentences on mere users is counterproductive – and this bill tries to do that. But our disagreement with this bill on a policy level won't defeat the legislation – that will be accomplished by the very nature of this proposal – enactment of a restriction on a fundamental constitutional right based on incomplete hearings and biased or inadequate findings.

It cannot seriously be argued that in order for this committee to properly evaluate the risks of marijuana use and possession in the privacy of the home, it must consider far more evidence than it has been able to take in over the course of three one or two hour sessions. We believe the weight of the evidence clearly shows that marijuana is not significantly more dangerous than it was in the 1970s, when Alaska law legalized use and possession in the home. However, we acknowledge the availability of dozens, perhaps hundreds, of studies and papers on the subject and that all of those papers present detailed, scientific analysis and conclusions. This five person committee, with all of your various backgrounds and experiences, and even supported as you are by an insightful and dedicated staff, cannot possibly come to a well-rounded decision based on a fair evaluation of the available evidence on such an abbreviated schedule. National and international panels have tackled this subject over the years – and they typically take months, if

not years, to do a thorough evaluation of all the evidence and opinions. Uniformly, those panels conclude that marijuana is significantly less dangerous than alcohol and uniformly, the findings of those panels cannot be read to show any substantial increase in the risks associated with marijuana over the last 30 years. The administration's proposed bill offers you a pre-set list of findings – obviously and not unexpectedly slanted far in favor of the proposition that marijuana is dangerous. We understand that the administration would want you to believe those proposed findings – but if you examine all the information to be presented to you today, together with all the evidence presented to you by the administration, you should be unable in good conscience to vote in support of a bill including those findings. The findings associated with this bill are clearly one-sided, ignore the weight of evidence on the subject, and in some cases are simply wrong.

We believe the administration is offering misleading information to you. Others with more scientific expertise may speak to this, but I will offer one small example. I might offer more, but the committee declined to provide us with a copy of the studies submitted by the government. I was limited to a review of no more than five or ten minutes of one copy of the package. In that short amount of time, I happened across one of the administration's assertions – that 15% of rape suspects were under the influence of marijuana. This assertion presumably supports finding #5 – that a high percentage of domestic violence arrestees are under the influence of marijuana – and finding #12 – that a large percentage of violent offenders have marijuana in their systems upon arrest . If you examine the report in question, you will see that 70 % - 70% - of the suspects were under the influence of alcohol. You will also see that there is no information about whether the marijuana users had also used alcohol. You would also see that the authors of the report were so concerned about the correlation between alcohol and violence that they parsed out the alcohol subject for further detail. You would also see that the authors deemed the marijuana correlation too slight to warrant further evaluation. But the administration didn't tell you that in their bullet pointed highlights – they simply said that 15% of rape suspect had marijuana in their systems – hoping that no one would take attention to the narrative of the report. And the findings proposed in this bill are – each and every one of them – as the experts to follow will tell you – misleading assertions based on similar kinds of half truths and omissions. We believe strongly that findings such as these, if left to stand, will be the downfall of this bill in court.

In most cases, legislative findings really wouldn't matter all that much, because the courts generally defer to the Legislature's findings on a subject. However, when a bill restricts a suspect class or, as in this case, a fundamental right, a different standard applies. In such cases, the court will examine the Legislature's consideration of the facts – or will conduct its own fact-finding. The courts will not blindly accept legislative findings when a law has distinctions based on religion or ethnicity, when a law restricts free speech – especially political speech, restricts freedom of worship, or – as here – restricts an individual's right of privacy.

Privacy is a fundamental right in Alaska – stronger than the privacy right inferred to exist under the US Constitution. Legislators, acting on a 2/3 vote in the 1970's and backed by referendum of the voters, amended the Alaska Constitution to write in an express right of privacy. Because the right is express, not merely inferred as the federal right is, it is generally considered to be stronger than the federal right. But the Constitution doesn't say how far the right of privacy

extends – and so the courts have been asked to do the job of defining the meaning of our right of privacy.

Shortly after the amendment adding the privacy right, the Ravin case came along. At its core, Ravin has less to do with marijuana possession than with attempting to define the scope of the privacy right. Ravin stands for the proposition that activities in the home are entitled to a higher degree of privacy protection than other kinds of activities. Also, activities that are purely personal and don't pose a significant risk to others are entitled to some degree of privacy protection. The court was rather eloquent in describing its reasoning:

> "...the authority of the state to exert control over the individual extends only to activities of the individual which affect others or the public at large as it relates to matters of public health or safety, or to provide for the general welfare. We believe this to be a tenet of a free society. The state cannot impose its own notions of morality, propriety, or fashion on individuals when the public has no legitimate interest in the affairs of those individuals."

Having come to these conclusions about the scope of the privacy right, the court had to determine whether marijuana use and possession fell within its protective ambit.

In order to do so, the court considered voluminous materials and heard from numerous experts. The Supreme Court concurred with lower court rulings that marijuana was not without risk. However, it said the health effects were not so severe as to warrant a government intrusion on a basic human right. So, the administration's argument in support of this bill will now stand or fall on whether it can demonstrate that marijuana is so much more dangerous now than in the 1970's, when Ravin was decided, that justification now exists to restrict individual privacy rights where none existed before. Be clear on this point: it's not enough to find that marijuana has risks – the court already decided in the 1970's that there was some risk in the use of marijuana – albeit far less than in the use of alcohol. The court heard evidence from some that marijuana was dangerous, offset by others who said that it had some risks, but not many. The court decided that balance of evidence was insufficient to justify a restriction on privacy rights. In order to make this legislation fly, you need to determine that marijuana is far more dangerous than it was in 1975. Unfortunately, the evidence won't tell you that marijuana spurs violence. It won't tell you there has been a significant increase in young users. It won't tell you there is any significant increase in potency that isn't offset by decreased doses and reduced health impacts. We don't think the case can be made that there is any increased danger in marijuana use today and, if you consider all evidence available to you, we are certain you'll have to agree.

Why should you be concerned about the available evidence, making sure you've fully evaluated everything, and made accurate findings? Because the legislation will stand or fall on whether you do the job fully and fairly. As noted before, courts will generally defer to fact-finding done by the legislature – but not if a fundamental right is restricted. In that case, the court will look for a substantial relationship between the proposed restriction and a compelling state interest. And it will be far less likely to defer to the legislature's findings....for lack of a better term, the court will take a "hard look" at what the legislature did in adopting the legislation.

There isn't a lot of Alaska precedent on this issue, but there is plenty on the federal level – and it's likely the reasoning would be the same among the Alaska judiciary. Even when fundamental rights are not implicated, the courts will not just blindly accept legislative reasoning. As Justice Clarence Thomas said, when he was an appellate justice, with respect to federal legislation, if Congress "could make a statute constitutional simply by 'finding' that black is white or freedom, slavery, judicial review would be an elaborate farce." Lambrecht v. FCC, 958 F.2d 382, 392 n. 2 (D.C. Cir. 1992). When a fundamental right is restricted, though, the court will not merely look at the legislative record, it can go beyond that to find facts relevant to the nature of the law. So, for example, in a 1994 case, Turner Broadcasting v. FCC, the US Supreme Court said that when First Amendment rights are implicated, deference afforded to legislative findings does not foreclose a court's independent judgment of the facts bearing on an issue of constitutional law. It should be noted that in several cases, the courts have looked disparagingly on findings that were the result of pre-ordained decisions, insufficient hearings, or hasty deliberations. Even in Alaska, the few precedents suggest that the ordinary deference to legislative action disappears when constitutionally protected rights are at issue.

Examining all precedents on this issue, if the court is at all likely inclined to defer to the legislature in this context, it will only be if the legislature has comprehensively reviewed the available evidence and arrives at conclusions – "findings", if you will – that accurately reflect that evidence. The courts will not tolerate proceedings that are merely a show to make it seem as if the legislature has taken a comprehensive look at the available evidence. They will not stand for a paper record of several hundred pages if the result is directly contrary to the evidence submitted. Based on our experience before this committee, it is our view that the attention given to the issues falls far short of what the court would expect.

I do not mean to belittle the effort this committee has made. Far from it. We have been treated cordially and have been helped at every turn by committee members and staff. We have provided documentary evidence, which has been accepted willingly. But to understand the complexity of the issue involved here requires commitment of far more time and expertise. By rights, this panel should convene something akin to a Blue Ribbon Commission – as has been done elsewhere – to advise and inform the committee on this issue. But there seems to be no interest in conducting such a thorough review of the issues. It's understandable – this committee and this body has many important issues to address and there is a natural urge to push things along – get things done. But when a restriction on a fundamental right is involved, you need to do more.

Even in the absence of such an obligation, other panels have done more – much, much more. Starting with the commission that examined the marijuana issue in the 1970's and continuing through recent panels in Canada and elsewhere, experts have testified for days and deliberations have continued for months. This is very complicated information and no one can be expected to fully comprehend all of the findings in a short review lasting a few hours. And we believe strongly that the court will not let stand the restrictions you are contemplating adopting based merely on the record that will be before you as of the day's end.

I will let others speak to the findings relating to scientific issues, but I would like to address two findings in particular.

Finding #19 purports to "reconfirm that it is illegal to possess any amount of marijuana anywhere in this state...." In fact, by adopting such a finding, the Legislature wholly discounts the judiciary's role in our three-branch system of government. Pursuant to judicial decision, it in fact has NOT been illegal to possess small amounts of marijuana in the home for personal use. To adopt a finding that Reconfirms illegality, this body asserts, in effect, that the judiciary has no role in determining the constitutionality of legislative enactments.

Finding #18 asserts that the holding in Crocker v. State imposes "unnecessary and unreasonable requirements for search warrants to investigate marijuana growing...that inhibit law enforcement efforts to reduce the amount of marijuana illegally grown indoors and illegally sold or exported." This is simply inaccurate. Crocker merely held that "a judicial officer should not issue a warrant to search a person's home for evidence of marijuana possession unless the State's warrant application establishes probable cause to believe that the person's possession of marijuana exceeds the scope of the possession that is constitutionally protected under Ravin.... Before a search warrant can lawfully issue, the government must establish probably cause to believe that the evidence being sought is connected to a crime. This same rule governs search warrants for all controlled substances, not just marijuana." This is neither unnecessary nor unreasonable – it's the law. Probable cause means probable cause to suspect a violation of the law, not probable cause to suspect the occurrence of constitutionally protected activity. Could you get a warrant to enter a home if you suspected someone was exercising his or her right to freedom of speech? It's a silly point – but you understand my point. The finding in question is blatantly slanted and there is no need for it.

I'd also like to point out that much of the administration's claims and assertions are irrelevant to the consideration of this bill. For example, you heard about marijuana being a huge cash crop in Alaska – about huge discoveries of marijuana in the state. You have heard about crime and driving issues. Your ability to legislate with respect to those issues is now and has always been unhindered. Those things are illegal now and this bill does nothing to change those laws – therefore, those comments are simply irrelevant – again, designed to appeal to your gut instincts to legislate against this substance. The court has simply said that the right to privacy is fundamental – and therefore there must be a direct connection between some compelling state interest and the invasion of privacy that goes with restricting use and possession of small amounts in the home.

We would strongly urge this committee to remove or drastically alter the findings in this bill. As they stand, they do not reflect the state of the science on the subject. They will serve as the basis for a challenge to this bill. We strongly urge this committee undertake a far more comprehensive look at the science – or work for the creation of a truly independent panel to examine the science. The Alaska Civil Liberties Union would be please to help establish and/or inform such a panel. We believe that is the only way this Legislature can successfully adopt a restriction to the basic right of privacy without subjecting itself to challenge. We also believe the adoption of findings more fairly reflective of the science will lead to a judicial conclusion that the risks associated with marijuana use and possession do not justify a restriction on the right of privacy.

We understand the basic reflex to criminalize marijuana possession is based on a fear that our kids are being harmed. But there are better ways to deal with this than to make marijuana users criminals. There have been success stories – with tobacco and with other drugs at other times and places. Prison is not the best place to put someone who needs to stop using marijuana....and invading someone's privacy is not the best means to find the people who need to stop using marijuana. And another judicial challenge to another legislative enactment will do nothing to advance either the cause of personal privacy or the goal of reducing drug use. We urge you please not to pass Senate Bill 74.

Thank you Mr. Chairman:

My name is Dr. Lester Grinspoon. I believe that you have copies of my curriculum vitae and two of my books: Marihuana Reconsidered (Harvard University press, 1971, 1977 and Marihuana, the Forbidden Medicine (Yale University press1993, 1997).

To be very brief, I am an associate professor of psychiatry, emeritus at the Harvard Medical School. Shortly after graduating from the Harvard Medical School in 1955, I joined the faculty and over the ensuing 45 years combined research and teaching with some clinical practice. In 1967, I had finished writing my part of a book on our seven-year study of schizophrenia. As the senior author I had to wait to finally put it together for what two junior co-authors estimated would be two to three months before they completed their parts. Because I was at that time very m[illegible] concerned about the great danger young people were exposing themselves to as they recklessly ignored government warnings about its dangers, I decided to devote this unanticipated free time to studying marihuana in the Countway library with the object of producing a scientifically sound paper on the subject, one which I hoped to publish in a journal or periodical accessible to college-age people. Perhaps some would pay more attention to such a review than they apparently were to the material produced by the US Public Health Service. (A shortened version of the paper was published in Scientific American). As I delved into the medical, scientific and other literature I soon discovered, to my great surprise and consternation, that, despite my training in medicine and science, I had been brainwashed like most other American citizens about the dangers of this drug. After I finished the book on schizophrenia, I began work on Marihuana Reconsidered which was published by Harvard University press in 1971, a second edition in 1977 and republished as a classic in 1997. Briefly, after documenting that most of what we believe about the dangers of cannabis were mythical, I concluded that marihuana was far less harmful than either alcohol or tobacco and that its greatest harmfulness arose from the way we as a society were dealing with it. (At that time we were arresting about 300,000 mostly young people on marihuana charges; today the figure is about 750,000.).

Let me say at the outset that marihuana is no more harmful today than it was in 1975 when I testified in the Ravin case. Street marihuana is arguably more potent than it was at that time but this does not mean increased risk because both medicinal and recreational users very quickly learn how to titrate the dose to achieve the desired effect. A user who smokes (or vaporizes) marihuana has to inhale less of a more potent sample and, conversely, more of one that is less potent. It follows that to the extent that inhaling the smoke is considered a risk factor for pulmonary disease, the more potent sample provides a healthier choice. However, I should hasten to add that the pulmonary risk from smoking marihuana has been greatly exaggerated. There is not a single case of lung cancer or emphysema attributable to smoking marihuana to be found in the

medical literature. I believe that the lungs of marihuana smokers are at greater risk from the air of cities like Los Angeles or Houston (or any other city with poor air quality). However, for those, particularly in today's anti-smoking atmosphere, who wish to avoid smoke for any reason, there is now available a device called a vaporizer which holds the temperature of the marihuana to be consumed in a temperature window which vaporizes the cannabinoids at a temperature which is below the ignition point; thus no smoke.

I wish I had time to address the other 18 "Findings" because so many are erroneous and none is supported by documentation. Let me briefly consider another "Finding", the one that states... "marihuana use by children is associated with an increased risk of attempting suicide." I believe that the intention here is to suggest that because there is an association cannabis must be causal in this increment of risk. Psychiatry is becoming increasingly aware that children suffer from depression and some of them commit suicide. Like other children, some of the depressed children will use marihuana. In fact, because marihuana is an effective antidepressant, some of them may have discovered it as a self-medication. However, there is no credible evidence that I know of that establishes marihuana as a causal.

I am struck by the fact that so many of these "Findings" are the same claims of marihuana toxicity that have been made and discredited more than once in the history of this substance. For example, schizophrenics and patients suffering from other psychoses were thought to comprise a significant number of the patients admitted to Indian Insane Asylums in the second half of the 19th-century and the use of ganja was thought to be causative. It was for this reason that the British organized the Indian Hemp Drug Commission Study which was published in 1894. The commission examined 800 doctors, superintendents of insane asylums, and so forth. In a 3000 page, seven volume report the commission concluded that "There is no evidence of any weight regarding mental and moral injuries from the moderate use of these drugs." This report put to rest the belief that cannabis led to schizophrenia and other psychoses for a while, but it has recurred periodically most recently during the last year are so. For example, a report from New Zealand with a study group of only 759 subjects, claims to have established that those who smoked marijuana three or more times by the age of 15 had a 10% higher chance of developing schizophrenia. Similarly, other studies from Great Britain and the Netherlands would predict greater numbers of schizophrenic patients. If these predictions were correct, given the number of young people who are or who have used cannabis, we would expect an increment in the incidence of schizophrenia. However, no such increment exists.

In "Finding" number two the assertion is made that "... [marihuana] has addictive properties similar to heroin and other similar illegal controlled substances .." Most of those who are sophisticated about cannabis would question whether the word addiction is even appropriate to this drug, and all would agree that the withdrawal syndrome seen with "heroin and other illegal controlled substances" are not

observed upon cessation of marihuana use. The assertion made in "Finding" number four, that marihuana use "... makes it more likely that the person will go on to use more potent illegal controlled substances..." is simply a restatement of what was known as the "stepping stone hypothesis"; a belief which has long since been thoroughly discredited.

As I participate in these hearings, I am reminded of those which preceded the passage of the Federal Marihuana Tax Act of 1937 the first of the draconian legislation aimed at marihuana. A reading of the hearings before the House Ways and Means Committee that preceded the passage of the legislation demonstrates quite clearly how little empirical data was found to support the Act. Indeed, the enactment reflected far more the mass hysteria surrounding the subject than any concrete evidence of the drug's harmfulness. The hearings were characterized by brevity and lack of information. Little expert medical, sociological, or of the scientific evidence was produced or listened to. I would urge this committee to acquaint itself with some of the excellent comprehensive special reports and commission reports which have been developed over the past half-century. They would include:

The La Guardia Report (1944)
The Wooten Report (1968)
The Shafer Report (1972)
The Le Dain Commission (1973)
The Canadian Senate Special Committee on Illegal Drugs Report (2002)

I think that if this legislative body is as meticulous and comprehensive in collecting and assessing the data as these Commissions were, it will have a better chance of arriving at a sound judgment about whether the "harmfulness" of marihuana is sufficient to enact such a restrictive bill.

In conclusion, I must tell you that I have much more to say on this topic than I will be able to now, but given the extremely short time allotted for these hearings, there is no way I can thoroughly respond to the erroneous findings proposed in this bill. It would take days, if not weeks, to carefully review each of the studies and reports submitted by the government in order to fully explain to you the fallacies and inaccuracies. In short, this committee cannot possibly hope to seriously consider in the amount of time allocated the relevant evidence necessary to reach conclusions about the public health effects of marijuana. I would urge the committee to postpone action on this legislation until such time as a full and fair review of all the evidence has occurred. If the legislature does adopt "findings" I would urge you to fully annotate those findings with specific references to specific evidence in the legislative record.

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Marital status: married, three children

EDUCATION:

1951 B.S., Tufts College, Medford, Massachusetts, magna cum laude.

1955 M.D., Harvard Medical School, Boston, cum laude.

POSTGRADUATE TRAINING AND EXPERIENCE:

1955-1956 Intern in Medicine, Beth Israel Hospital, Boston, Massachusetts.

1956-1958 Field Investigator for the National Cancer Institute, Los Angeles, California.

1958-1961 Resident in Psychiatry, Massachusetts Mental Health Center, (Chief of Drug Unit 1959-1960; Chief of Service 1960-1961).

RESEARCH AND TEACHING APPOINTMENTS:

1950-1951 Olmstead Fellow in Biology, Tufts College, Medford, Massachusetts

1956-1958 Assistant in Medicine, University of Southern California School of Medicine, Los Angeles, California

1958-1959 Teaching Fellow in Psychiatry, Harvard Medical School, Boston, Massachusetts

1961-1962 Assistant in Psychiatry, Harvard Medical School, Boston, Massachusetts

1961-1963 Lecturer on Social Relations, Harvard University, Cambridge, Massachusetts

1962-1964 Instructor in Psychiatry, Harvard Medical School,
1962-1965 Boston, Massachusetts

1961-1991 Senior Psychiatrist, Massachusetts Mental Health Center, Boston, Massachusetts

1964-1965 Clinical Associate in Psychiatry, Harvard Medical School, Boston, Massachusetts

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1965-1968	Assistant Clinical Professor of Psychiatry, Harvard Medical School, Boston, Massachusetts
1968-1973	Associate Clinical Professor of Psychiatry, Harvard Medical School, Boston, Massachusetts
1973-1998	Associate Professor of Psychiatry, Harvard Medical School, Boston, Massachusetts
1998-2000	Associate Clinical Professor of Psychiatry, Harvard Medical School, Boston, Massachusetts
2000-	Associate Professor of Psychiatry Emeritus, Harvard Medical School, Boston, Massachusetts

OTHER APPOINTMENTS:

1961-1968	Director, Clinical Research Center, Massachusetts Mental Health Center
1962	Director, Summer Institute on Alternative Ways of Handling Conflict: Behavioral Science Research Toward Peace, Sponsored by the American Academy of Arts and Sciences
1963-1970	Consultant in Psychiatry and Research, Boston State Hospital
1969-	Examiner, American Board of Psychiatry and Neurology
1972-1988	Advisory Board, National Organization for the Reform of Marijuana Laws
1973-1974	Budget Committee, American Psychiatric Association
1973-	Executive Director, Massachusetts Mental Health Research Corporation
1974-1979	Consultant, Task Force on Interface between Psychiatry and Industry, American Psychiatric Association
1974-1979	Council on Research, American Psychiatric Association
1975-1977	Vice-Chairperson, Council on Research, American Psychiatric Association
1976-1981	Advisory Board, The Center for the Study of Non-Medical Drug Use

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1977-1979	American Psychiatric Association Representative to the American Association for the Advancement of Science
1977-1979	Chairperson, Council on Research, American Psychiatric Association
1979-1980	Vice-Chairperson, Scientific Program Committee, American Psychiatric Association
1979-1980	Chairperson, Subcommittee on Awards for Scientific Exhibits, American Psychiatric Association
1979-	Council on Marihuana and Health, National Organization for the Reform of Marijuana Laws
1980-1984	Chairperson, Scientific Program Committee, American Psychiatric Association
1980-1984	Scientific Advisory Board, Beneficial Plant Research Association
1984-1985	Chairperson, Task Force on Soviet/American Relations, American Psychiatric Association
1986-1990	Founding Board of Directors, Physicians for Human Rights
1987-	Advisory Board, The Drug Policy Foundation
1987-	Board of Advisors, The Albert Hofmann Foundation
1989-	Vice President, International Antiprohibitionist League
1989-1991	Advisory Board, Civil Liberties Union of Massachusetts/ACLU
1989-1991	Board of Directors, Center for Psychological Studies in the Nuclear Age
1990-	Advisory Board, Physicians for Human Rights
1990-1992	Board of Directors, Drug Policy Foundation
1991-1993	Board of Directors, Civil Liberties Union of Massachusetts
1993-1996	Faculty Member, Zinberg Center for Addiction Studies, Cambridge, Massachusetts
1994-1995	Chairperson, Board of Directors, National Organization for the Reform of Marihuana Laws

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1995-	Advisory Board, The Drug Research Group
1997-	Board of Scientific and Policy Advisors of the American Council on Science and Health
1997-	Honorary Member, Arbeitsgemeinschaft Cannabis als Medizin (Alliance for Cannabis as Medicine), Germany
1997-	International Advisory Committee, Physicians for Human Rights
1999	Reviewer of the draft report on the usefulness of marijuana as a medicine by the Institute of Medicine, subsequently published (Marijuana and Medicine: Assessing the Science Base) by National Academy Press, Washington, D.C., 1999
2000-	National Advisory Council, Center for Cannabis Research, University of California

EDITORIAL BOARDS:

1982-1984	Editor, Psychiatry Update: The American Psychiatric Association Annual Review; Volumes I-III
1982-1993	Journal of Psychiatric Research
1984-2000	Editor, The Harvard Mental Health Letter
1985-	Journal of Social Pharmacology
1985-	The Harvard Health Letter
1991-	Addiction Research
1998-	Journal of Cognitive Liberty and Ethics
2001	Journal of Cannabis Therapeutics

OTHER PROFESSIONAL ACTIVITIES:

Testified before legislative committees in the states of Massachusetts, Colorado, New Jersey, Washington, Vermont, and New York. Also testified before the National Marijuana Commission (1972), the House Armed Services Committee (1962), the Monopoly Subcommittee of the Senate Small Business Committee (1976), the House Select Committee on Narcotics (1977, 1979, 1989), the Controlled Substances Advisory Committee, the Drug Abuse Research Advisory Committee (1978), and the Senate Judiciary Committee (1980), etc.

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HONORARY SOCIETIES:

Phi Beta Kappa
Alpha Omega Alpha
Boylston Society, Harvard Medical School
Columbia University Seminar Associate

PROFESSIONAL ORGANIZATIONS:

Massachusetts Medical Society
American Psychiatric Association (Fellow)
American Association for the Advancement of Science (Fellow)
Group for the Advancement of Psychiatry
Society of Biological Psychiatry
World Federation of Mental Health

MEDICAL LICENSING AND CERTIFICATION:

Diplomate, National Board of Medical Examiners
Licensed, State of Massachusetts
Diplomate, American Board of Psychiatry

PSYCHOANALYTIC TRAINING:

Graduate, Boston Psychoanalytic Institute, Boston, Massachusetts, April 1967

Member, Boston Psychoanalytic Society, Boston, Massachusetts, 1967-1985

AWARDS:

Mencken Award: Honorable Mention Winner for contribution to Dealing with Drugs, 1988

Alfred R. Lindesmith Award for Achievement in the Field of Scholarship, a $10,000 award of the Drug Policy Foundation, Washington, D.C., 1990*

Norman E. Zinberg Award for Marihuana Research, an award of The National Organization for the Reform of Marijuana Laws, Washington, D.C., 1990

National Organization for the Reform of Marijuana Laws, First Annual Lester Grinspoon Award For Outstanding Achievement in Marijuana Law Reform: In grateful recognition of a lifetime dedicated to reforming unjust marijuana laws, selfless devotion to healing the sick with medical marijuana, and willingness to champion an unpopular cause, regardless of professional consequences, November 14, 1998

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Alliance of Reform Organizations, The ARO Lifetime Achievement Award for Distinguished Service in Drug Policy Reform, 1998

*see citation, page 25

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