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Exposing Marijuana Myths: A Review of the Scientific Evidence¹

Introduction

Since the 1920s, supporters of marijuana prohibition have exaggerated the drug's dangers. In different eras, different claims have gained prominence, but few have ever been abandoned. Indeed, many of the "reefer madness" tales that were used to generate support for early anti-marijuana laws continue to appear in government and media reports today.

For a while in the 1970s, it seemed as if scientific inquiries were beginning to influence the government's marijuana policies. Following thorough reviews of the existing evidence by scholars¹ and official commissions,² criminal penalties for marijuana offenses were lessened and a number of states moved in the direction of decriminalization.³ However, in response to lingering concerns about marijuana's potential toxicity, the government expanded its funding of scientific research, mostly through the newly created National Instutite on Drug Abuse (NIDA).

Probably the most important studies of the 1970s were three large "field studies" in Greece, Costa Rica and Jamaica. These studies, which evaluated the impact of marijuana on users in their natural environments, were supplemented by clinical examinations and laboratory experiments oriented toward answering the questions about marijuana that continued to be debated in the scientific literature. The data from these studies, published in numerous books and scholarly journals, covered such matters as marijuana's effects on the brain, lungs, immune and reproductive systems, its impact on personality, development, and motivational states, and its addictive potential.

Although these studies did not answer all remaining questions about marijuana toxicity, they generally supported the idea that marijuana was a relatively safe drug -- not totally free from potential harm, but unlikely to create serious harm for most individual users or society. In the years since, thousands of additional studies have been conducted, many of them funded by NIDA, and together they reaffirm marijuana's substantial margin of safety. Our review of that body of work reveals an occasional study indicating greater toxicity than previously thought. But in nearly all such cases, the methodologies were seriously flawed and the findings could not be replicated by other researchers.

Especially since the 1980s, when the federal government's renewed war on cannabis began, both the funding of marijuana research and the dissemination of its findings have been highly politicized. Indeed, NIDA's role seems to have become one of service to the War on Drugs. Dozens of claims of toxicity appear in its documents, despite the

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existence of scores of scientific studies refuting their validity. At the same time, studies that fail to find serious toxicity are ignored.

In the following pages, we review the scientific evidence surrounding the most prominent of the anti-marijuana claims.

CLAIM #1: MARIJUANA USE IS INCREASING AT AN ALARMING RATE

Reports of a recent slight increase in marijuana use, especially among youth, are being used to convince Americans that a renewed campaign about the drug's dangers is necessary to avert an impending epidemic.

THE FACTS

According to government surveys of the general population, marijuana use began decreasing in 1980, after more than a decade of steady increase. By 1990, the downward trend showed signs of slowing, but use-rates remained substantially lower than those recorded in the 1970s.

For example, among 12-17 year olds, past year marijuana use was about 8 percent in 1992, compared to 24.1 percent in 1979. Among 18-25 year olds, past year use was 23 percent in 1992, compared to 46.9 percent in 1979.⁵

A separate survey of high school students shows similar trends, with use-rates in the 1990s well below those reported in the 1970s. However, after reaching an all-time low in 1992, they increased slightly during the next two years.

Lifetime Prevalence of Marijuana High School Seniors, 1976-1994⁶

1976	1978	1980	1982	1984	1986	1988	1990	1992	1994

52.8 59.2 60.3 58.7 54.9 50.9 47.2 40.7 32.6 38.2

The High School Survey was originally conceived by the National Institute on Drug Abuse (NIDA) as a measure of non-pathological drug use. This is still what it measures.

Adolescence is a time of experimentation, with drug use as well as other activities. Most adolescent drug users do not go on to become "drug abusers." Indeed, most adolescent drug users, after a few years of experimentation, cease using illegal drugs altogether.

We will probably never know why marijuana-use rates go up and down over time. However, it is worth noting that the recent increase occurred among the same population of young people who had been exposed to a decade-long anti-marijuana campaign in the schools and the media. That campaign, based on exaggerations of marijuana's harms and a "just say no" ideology, has clearly failed.⁷

Young people, and Americans generally, need to know the scientific evidence about marijuana if they are to make informed decisions about both their own drug use and the future of American drug policy.

CLAIM #2: MARIJUANA POTENCY HAS INCREASED SUBSTANTIALLY

The claim that there has been a 10-, 20- or 30-fold increase in marijuana potency since the 1970s is used to discredit previous studies that showed minimal harm caused by the drug and convince users from earler eras that today's marijuana is much more dangerous.

THE FACTS

For more than 20 years the government-funded Potency Monitoring Project (PMP) at the University of Mississippi has been analyzing samples of marijuana submited by U.S. law enforcement officials. At no time have police seizures reflected the marijuana generally available to users around the country and, in the 1970s, they were over-represented by large-volume low-potency Mexican kilobricks.⁸

During the 1970s, the PMP regularly reported potency averages of under 1%, with a low of 0.4% in 1974. Quite clearly, these averages under-estimate the THC content of marijuana smoked during this period.

Marijuana of under 0.5% potency has almost no psychoactivity. While it is possible that people sometimes obtained marijuana of such low potency, for the drug to have become popular in the 1960s and 1970s, most people must have regularly obtained marijuana with higher THC content.

Until the late 1970s, PMP samples included none of the traditionally higherpotency cannabis products, such as buds and sinsemilla, even though these products were available on the retail market. When changes in police practices resulted in their seizure, PMP potency averages increased.

Every independent analysis of potency in the 1970s found higher THC averages than the PMP. For example, the 59 samples submitted to PharmChem Laboratories in 1973 averaged 1.62%; only 16 (27%) contained less than 1% THC, more than half were over 2% and about one-fifth were over 4%. In 1975, PharmChem samples ranged from 2 to 5%, with some as high as 14% -- nearly *30 times* the .71 average reported by the PMP.⁹

After 1980, both the number and variety of official seizures increased dramatically, improving the validity of the PMP's reported averages, although they continue to be based on "convenience" rather than "representative" samples.

As shown below, average potency has remained essentially unchanged since the early 1980s:

<u>Mean Percentage THC of Seized Marijuana, 1981-1993</u> Mississippi Potency Monitoring Project

1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993
2.28	3.05	3.23	2.39	2.82	2.30	2.93	3.29	3.06	3.36	3.36	3.00	3.32

Even if potency had increased slightly since the 1970s, it would not mean that smoking marijuana had become more dangerous. In fact, since the primary health risk of marijuana comes from smoking, higher potency products can be less dangerous because they allow people to achieve the desired effect by inhaling less.

CLAIM #3: MARIJUANA IS A DRUG WITHOUT THERAPEUTIC VALUE

Proposals to make marijuana legally available as a medicine are countered with claims that safer, more effective drugs are available, including a synthetic version of delta-9-THC, marijuana's primary active ingredient.

THE FACTS

For thousands of years, throughout the world, people have used marijuana to treat a variety of medical conditions.¹⁰

Today, in the United States, such use is prohibited. Although thirty-six states have passed legislation to allow marijuana's use as a medicine, federal law preempts their making marijuana legally available to patients.

A number of studies have shown that marijuana is effective in reducing nausea and vomiting,¹¹ lowering intraocular pressure associated with glaucoma,¹² and decreasing muscle spasm and spasticity.¹³ Today, many people use marijuana for these and other medical purposes, despite its illegal status.¹⁴

People undergoing cancer chemotherapy have found smoked marijuana to be an effective anti-nauseant -- often more effective than available pharmaceutical medications.¹⁵ Indeed, 44 percent of oncologists responding to a questionnaire said they had recommended marijuana to their cancer patients; others said they would recommend it if it were legal.¹⁶

Marijuana is also smoked by thousands of AIDS patients to treat the nausea and vomiting associated with both the disease and AZT drug therapy. Because it stimulates appetite, marijuana also counters HIV-related "wasting," allowing AIDS patients to gain weight and prolong their lives.

In 1986, a synthetic delta-9-THC capsule (Marinol) was marketed in the United States and labeled for use as an anti-emetic. Despite some utility, this product has serious drawbacks, including its cost. For example, a patient taking three 5 mgm capsules a day would spend over \$5,000 to use Marinol for one year. In comparison to the natural, smokeable product Marinol also has some pharmacological shortcomings.¹⁷

Because THC delivered in oral capsules enters the bloodstream slowly, it yields lower serum concentrations per dose.

Oral THC circulates in the body longer at effective concentrations, and more of it is metabolized to an active compound; thus, it more frequently yields unpleasant psychoactive effects.

In patients suffering from nausea, the swallowing of capsules may itself provoke vomiting.

In short, the smoking of crude marijuana is more efficient in delivering THC and, in some cases, it may be more effective.

The continuing illegality of medical marijuana is based more on political than scientific considerations. Although during the 1970s the government supported exploration into marijuana's therapeutic potential,¹⁸ its role has become one of blocking new research¹⁹ and opposing any change in marijuana's legal status.²⁰

CLAIM #4: MARIJUANA CAUSES LUNG DISEASE

It is frequently claimed that marijuana smoke contains such high concentrations of irritants that marijuana users' risk of developing lung disease is equal to or greater than that of tobacco users.

THE FACTS

Except for their psychoactive ingredients, marijuana and tobacco smoke are nearly identical.²¹ Because most marijuana smokers inhale more deeply and hold the smoke in their lungs, more dangerous material may be consumed *per cigarette*. However, it is the *total volume of irritant inhalation* -- not the amount in each cigarette -- that matters.

Most tobacco smokers consume more than 10 cigarettes per day and some consume 40 or more. Regular marijuana smokers seldom consume more than 3-

5 cigarettes per day and most consume far fewer. Thus, the amount of irritant material inhaled almost never approaches that of tobacco users.

Frequent marijuana smokers experience adverse respiratory symptoms from smoking, including chronic cough, chronic phlegm, and wheezing. However, the only prospective clinical study shows no increased risk of *crippling pulmonary disease (chronic bronchitis and emphysema)*.

Since 1982, UCLA researchers have evaluated pulmonary function and bronchial cell characteristics in marijuana-only smokers, tobacco-only smokers, smokers of both, and non-smokers. Although they have found changes in marijuana-only smokers, the changes are much less pronounced than those found in tobacco smokers.

The nature of the marijuana-induced changes were also different, occuring primarily in the lung's *large airways* -- not the *small peripheral airways* affected by tobacco smoke. Since it is small-airway inflamation that causes chronic bronchitis and emphysema, marijuana smokers may not develop these diseases.²²

In an epidemiological survey, approximately 1200 subjects gave information on smoking and pulmonary function at 2-year intervals. A large percentage of the subjects underwent pulmonary function testing. Although a small group who reported *previous marijuna smoking* had significant pulmonary abnormalities, *curent marijuana smokers* had no significant reduction in any pulmonary functions.²³

There are no epidemiological or aggregate clinical data suggesting that marijuana-only smokers develop *lung cancer*. However, since some bronchial cell changes appear to be *pre-cancerous*, an increased risk of cancer among frequent marijuana smokers is possible.²⁴

Since the pulmonary risks associated with marijuana are related to smoking, the danger is eliminated with other routes of administration. For committed smokers, pulmonary risk might be *reduced* with higher-potency products, which produce desired psychoactive effects with less inhalation of irritants. Smokers could also be encouraged to abandon deep inhalation and breath-holding, which increase drug delivery only slightly. Finally, pulmonary risk might be reduced if marijuana were smoked in water pipes rather than cigarettes.²⁵

CLAIM #5: MARIJUANA IMPAIRS IMMUNE SYSTEM FUNCTIONING

It has been widely claimed that marijuana substantially increases users' risk of contracting various infectious diseases. First emerging in the 1970s, this claim took on new significance in the 1980s, following reports of marijuana use by people suffering from AIDS.

THE FACTS

The principal study fueling the original claim of immune impairment involved preparations created with white blood cells that had been removed from marijuana smokers and controls. After exposing the cells to known immune activators, researchers reported a lower rate of "transformation" in those taken from marijuana smokers.²⁶

However, numerous groups of scientists, using similar techniques, have failed to confirm this original study.²⁷

In fact, a 1988 study demonstrated *an increase* in responsiveness when white blood cells from marijuana smokers were exposed to immunological activators.²⁸

Studies involving laboratory animals have shown immune impairment following administration of THC, but only with the use of extremely high doses. For example, one study demonstrated an increase in herpes infection in rodents given doses of 100 mg/kg/day -- a dose approximately *1000 times* the dose necessary to produce a psychoactive effect in humans.²⁹

There have been no clinical or epidemiological studies showing an increase in bacterial, viral, or parasitic infection among human marijuana users. In three large field studies conducted in the 1970s, in Jamaica, Costa Rica and Greece, researchers found no differences in disease susceptibility between marijuana users and matched controls.³⁰

Marijuana use does not increase the risk of HIV infection; nor does it increase the onset or intensity of symptoms among AIDS patients.³¹ In fact, the FDA decision to approve the use of Marinol (synthetic THC) for use in HIV-wasting syndrome relied upon the absence of any immunopathology due to THC.³²

Today, thousands of people with AIDS are smoking marijuana daily to combat nausea and increase appetite. There is no scientific basis for claims that this practice compromises their immune responses. Indeed, the recent discovery of a peripheral cannabinoid receptor asociated with lymphatic tissue should encourage aggressive exploration of THC's potential use as an immune-system stimulant.³³

CLAIM #6: MARIJUANA HARMS SEXUAL MATURATION AND REPRODUCTION

Marijuana has been said to interfere with the production of hormones associated with reproduction, causing possible infertility among adult users and delayed sexual development among adolescents.

THE FACTS

There is no evidence that marijuana impairs male reproductive functioning.

The Jamaican and Costa Rican field studies detected no differences in hormone levels between marijuana users and non-users.

In epidemiological surveys of marijuana users, no problems with fertility have emerged as important.

In 1974, researchers reported diminished testosterone, reduced sexual function and abnormal sperm cells in males identified as chronic marijuana users.³⁴ In a laboratory study, the same researchers reported an acute decrease in testosterone, but no chronic effect after nine weeks of smoking; they did not evaluate sperm volume or quality.³⁵ In other laboratory studies, researchers have been generally unable to replicate these findings³⁶ although by administering very high THC doses -- up to 20 cigarettes per day for 30 days -- one study found a slight decrease in sperm concentrations.³⁷ In all studies, test results remained within normal ranges and probably would not have affected actual fertility.

Severe adverse consequences have also been produced in male laboratory animals, although only with extremely high daily THC doses.³⁸

More importantly, in both the human and animal laboratory studies, all observed changes were reversed once THC adminstration was halted.

The claim that marijuana impairs female reproductive functioning in humans has no support in the scientific literature.

There have been no epidemiological studies indicating diminished fertility in female users of marijuana, and a recent survey found no impact of chronic marijuana use on female sex hormones.³⁹

Animal studies show hormonal changes and depressed ovulation following extremely high daily doses of THC. As occurs with males, these changes disappear once the experiment is completed.⁴⁰ In addition, when THC was administered to female monkeys for an entire year, they developed tolerance to its hormonal effects and normal cycles were reestablished.⁴¹

Almost immediately following publication of the few studies showing a marijuana impact on reproductive hormones, warnings about marijuana's potential impact on adolescent sexual development began to appear.

Other than one case report of a 16-year old marijuana smoker who had failed to progress to puberty,⁴² there has been nothing to indicate that such a potential exists. In whatever other ways one might consider marijuana to be bad for adolescents, *it does*

not retard their sexual development.

CLAIM #7: MARIJUANA USE DURING PREGNANCY HARMS THE FETUS

A powerful accusation in anti-drug campaigns is that children are permanently harmed by their mothers' use of drugs during pregnancy. Today, it is commonly claimed that marijuana is a cause of birth defects and development deficits.

THE FACTS

A number of studies reported low birth weight and physical abnormalities among babies exposed to marijuana in utero.⁴³ However, when other factors known to affect pregnancy outcomes were controlled for -- for example, maternal age, socio-economic class, and alcohol and tobacco use -- the association between marijuana use and adverse fetal effects disappeared.⁴⁴

Numerous other studies have failed to find negative impacts from marijuana exposure.⁴⁵ However, when negative outcomes are found, they tend to be widely publicized, regardless of the quality of the study.

It is now often claimed that marijuana use during pregnancy causes childhood leukemia. The basis for this claim is one study, in which 5% of the mothers of leukemic children admitted to using marijuana prior to or during pregnancy. A "control group" of mothers with normal children was then created and questioned by telephone about previous drug use. Their reported .5 percent marijuana userate was used to calculate a 10-fold greater risk of leukemia for children born to marijuana users.⁴⁶ Given national surveys showing marijuana prevalence rates of at least 10%, these "control group" mothers almost certainly under-reported their drug use to strangers on the telephone.

Also used as evidence of marijuana-induced fetal harm are two longitudinal studies, in which the children of marijuana users were examined repeatedly. However, on closer examination, the effects of marijuana appear to be quite minimal, if existent at all.

After finding a slight deficit in visual responsiveness among marijuana-exposed newborns, no differences were found at 6 months, 12 months, 18 months, or 24 months.⁴⁷ At age 3, the only difference (after controlling for confounding variables) was that children of "moderate" smokers had *superior* psycho-motor skills. At age 4, children of "heavy" marijuana users (averaging 18.7 joints/week) had lower scores on *one subscale* of one standardized test of verbal development.⁴⁸ At age 6, these same children scored lower on one computerized task -- that measuring "vigilance." On dozens of others scales and subscales, no differences were ever found.⁴⁹

In another study, standardized IQ tests were administered to marijuana-exposed and unexposed 3 year-olds. Researchers found no differences in the overall scores. However, by dividing the sample by race, they found -- among African-American children only -- lower scores on one subscale for those exposed during the *first trimester* and lower scores on a different subscale for those exposed during the *second trimester*.⁵⁰

Although it is sensible to advise pregnant women to abstain from using most drugs -including marijuana -- the weight of scientific evidence indicates that marijuana has few adverse consequences for the developing human fetus.

CLAIM #8: MARIJUANA CAUSES BRAIN DAMAGE

Critics state that marijuana damages brain cells and that this damage, in turn, causes memory loss, cognitive impairment, and difficulties in learning.

THE FACTS

The original basis of this claim was a report that, upon post-mortem examination, structural changes in several brain regions were found in two rhesus monkeys exposed to THC.⁵¹ Because these changes primarily involved the hippocampus, a cortical brain region known to play an important role in learning and memory, this finding suggested possible negative consequences for human marijuana users.

Additional studies, employing rodents, reported similar brain changes.

However, to achieve these results, massive doses of THC -- up to 200 times the psychoactive dose in humans -- had to be given. In fact, studies employing *100 times* the human dose have failed to reveal any damage.⁵²

In the most recently published study, rhesus monkeys, through face-mask inhalation, were exposed to the equivalent of 4-5 joints per day for an entire year. When sacraficed seven months later, there was no observed alteration of hippocampal architecture, cell size, cell number, or synaptic configuration. The authors conclude that:

"while behavioral and neuroendocrinal effects were observed during marijuana smoke exposure in the monkey, residual neuropathological and neurochemical effects of marijuana exposure were not observed seven months after the year-long marijuana smoke regimen."⁵³

Thus, twenty years after the first report of brain-damage in two marijuana-exposed monkeys, the claim of physiological damage to brain cells has been effectively disproven.

No post-mortem examinations of the brains of human marijuana users have ever been conducted. However, numerous studies have explored marijuana's effect on brain-related cognitive functions. Many employ an experimental design -- in which subjects are given marijuana in a laboratory setting, and then compared to controls on a variety of measures involving attention, learning, and memory.

In a number of studies, no significant differences were detected.⁵⁴ In fact, there is substantial research demonstrating that marijuana intoxication *does not* impair the retrieval of information learned previously.⁵⁵ However, there is evidence that marijuana, particularly in high doses, may interfere with users' ability to *transfer new information into long-term memory*.⁵⁶

While there is general agreement that, while under the influence of marijuana, learning is less efficient,⁵⁷ there is no evidence that marijuana users -- even long-term users -- suffer permanent impairment. Indeed, numerous studies comparing chronic marijuana users with non-user controls have found no significant differences in learning, memory recall, or other cognitive functions.⁵⁸

CLAIM #9: MARIJUANA IS AN ADDICTIVE DRUG

It is now frequently stated that marijuana is profoundly addicting and that any increase in prevalence of use will lead inevitably to increases in addiction.

THE FACTS

Essentially all drugs are used in "an addictive fashion" by some people. However, for any drug to be identified as highly addictive, there should be evidence that substantial numbers of users repeatedly fail in their attempts to discontinue use and develop usepatterns that interfere with other life activities.

National epidemiological surveys show that the large majority of people who have had experience with marijuana do not become regular users.

In 1993, among Americans age 12 and over, about 34% had used marijuana sometime in their life, but only 9% had used it in the past year, 4.3% in the past month, and 2.8% in the past week.⁵⁹

A longitudinal study of young adults who had first been surveyed in high school also found a high "discontinuation rate" for marijuana. While 77% had used the drug, 74% of those had not used in the past year and 84% had not used in the past month.⁶⁰

Of course, even people who continue using marijuana for several years or more are not necessarily "addicted" to it. Many regular users -- including many *daily*

users -- consume marijuana in a way that does not interfere with other life activities, and may in some cases enhance them.

There is only scant evidence that marijuana produces physical dependence and withdrawal in humans.

When human subjects were administered daily oral doses of 180-210 mg THC -the equivalent of 15-20 joints per day -- abrupt cessation produced adverse symptoms, including disturbed sleep, restlessness, nausea, decreased appetite, and sweating. The authors interpreted these symptoms as evidence of physical dependence. However, they noted the syndrome's relatively mild nature and remained skeptical of its occurrence when marijuana is consumed in usual doses and situations.⁶¹ Indeed, when humans are allowed to control consumption, even high doses are not followed by adverse withdrawal symptoms.⁶²

Signs of withdrawal have been created in laboratory animals following the administration of very high doses.⁶³ Recently, at a NIDA-sponsored conference, a researcher described unpublished observations involving rats pre-treated with THC and then dosed with a cannabinoid receptor-blocker.⁶⁴ Not surprisingly, this provoked sudden withdrawal, by stripping receptors of the drug. This finding has no relevance to human users who, upon ceasing use, experience a very gradual removal of THC from receptors.

The most avid publicizers of marijuana's addictive nature are treatment providers who, in recent years, have increasingly admitted insured marijuana users to their programs.⁶⁵ The increasing use of drug-detection technologies in the workplace, schools and elsewhere has also produced a group of marijuana users who identify themselves as "addicts" in order to receive treatment instead of punishment.⁶⁶

CLAIM #10: MARIJUANA-RELATED MEDICAL EMERGENCIES ARE INCREASING

As evidence of its harmful effects, prohibition advocates point to dramatic increases in emergency-room episodes related to marijuana ingestion.

THE FACTS

Data gathered by the Drug Abuse Warning Network (DAWN) show a recent increase in "marijuana mentions" by people seeking treatment in hospital emergency rooms. Using a one-page form, emergency-room personnel record "drug abuse episodes," note the presence or absence of alcohol as a contributing factor, and list up to four other drugs recently consumed by the patient.

Although DAWN began compiling data in the 1970s, recent changes in recording procedures, the hospital selection, and methods of statistical estimation prevent

comparisons of data gathered prior to 1988 with those gathered recently. Thus, discussion of emergency-room trends is limited to the years 1988 to 1993.⁶⁷

The lowest number of marijuana-mentions, recorded in 1990, was 15,706 (7.1 mentions per 100,000 population). The highest was 29,166 (12.7 per 100,000 population), recorded in 1993.

Using these figures, an increase of 86% has been reported. However, if 1988 is used as the "base year" instead -- a year in which there were 19,962 marijuana mentions -- the increase is reduced immediately by more than half, to 42%.

Despite marijuana being the *most frequently used* illicit drug, in emergency rooms, it remains the *least often mentioned* illicit drug.

In 1993, marijuana accounted for 6.25% of mentions, compared to 15.3% for cocaine and 9.8% for heroin. Even over-the-counter pain medications were mentioned more often than marijuana -- comprising 9% of the total.

For youth aged 6 to 17, there were more mentions of marijuana than of heroin and cocaine -- not because marijuana is more harmful to them but because these latter drugs are used so infrequently by young people. In this age group, mentions of over-the-counter pain medications were substantially higher than those for marijuana. While marijuana accounted for 6.48% of drug mentions by youth, over-the-counter pain medications accounted for 47%.

For the total population, not only is marijuana mentioned less frequently than other recreational drugs, it is seldom mentioned alone. In 1992, in more than 80% of the drug-abuse episodes involving marijunana, at least one other drug was mentioned; and, in more than 40%, two or more additional drugs were mentioned.

Of 24,000 marijuana mentions in 1992, more than 13,000 involved alcohol and nearly 10,000 involved cocaine.

Despite recent increases in marijuana mentions, hospital emergency rooms are not flooded with marijuana users seeking medical attention. In 1992, of 433,493 total drug mentions, only 4,464 -- about 1% -- involved the use of marijuana alone.

CLAIM #11: MARIJUANA PRODUCES AN AMOTIVATIONAL SYNDROME

Marijuana is said to have a deliterious effect on society by making users passive, apathetic, unproductive, and unable (or unwilling) to fulfill their responsibilities.

THE FACTS

The concept of an amotivational syndrome first appeared in the late 1960s,⁶⁸ as marijuana use was increasing among American youth. In the years since, despite the absence of an agreed-upon definition of the concept, numerous researchers have attempted to verify its occurrence.

Large-scale studies of high school students have generally found no difference in grade-point averages between marijuana users and non-users.⁶⁹ One study found lower grades among students reported to be *daily users of marijuana*, but the authors failed to identify a causal relationship and concluded that both phenomena were part of a complex of inter-related social and emotional problems.⁷⁰

In one longitudinal study of college students, after controlling for other factors, marijuana users were found to have *higher grades* than non-users⁷¹ and to be equally as likely to successfully complete their educations.⁷² Another study found that marijuana users in college scored higher than non-users on standardized "achievement values" scales.⁷³

Field studies conducted in Jamaica, Costa Rica and Greece also found no evidence of an amotivational syndrome among marijuana-using populations.

In these samples of working-class males, the educational and employment records of marijuana users were, for the most part, similar to those of non-users. In fact, in Jamaica, marijuana was often smoked during working hours as an aid to productivity.

The results of laboratory studies have been nearly as consistent.

In one study lasting 94 days, marijuana had no significant impact on learning, performance or motivation.⁷⁴

In another 31-day study, subjects given marijuana worked *more hours* than controls and turned in an equal number of tokens for cash at the study's completion.⁷⁵

However, in a Canadian study that *required* subjects in the marijuana group to consume unusually high doses, some reduction in work efficency was noted in the days following intoxication.⁷⁶

Undoubtedly, when marijuana is used in a way that produces near-constant intoxication, other activities and responsibilities are likely to be neglected.

However, the weight of scientific evidence suggests that there is nothing in the pharmacological properties of cannabis to alter people's attitudes, values, or abilities regarding work.

CLAIM #12: MARIJUANA IS A MAJOR CAUSE OF HIGHWAY ACCIDENTS

The detrimental impact of alcohol on highway safety has been well documented. Marijuana's opponents claim that it, too, causes significant impairment and that any increase in use will lead to increased highway accidents and fatalities.

THE FACTS

In high doses, marijuana probably produces driving impairment in most people. However, there is no evidence that marijuana, in current consumption patterns, contributes substantially to the rate of vehicular accidents in America.

A number of studies have looked for evidence of drugs in the blood or urine of drivers involved in fatal crashes. All have found alcohol present in 50 percent or more. Marijuana has been found much less often. Furthermore, in the majority of cases where marijuana has been detected, alcohol has been detected as well.⁷⁷

For example, a recent study sponsored by the U.S. National Highway Traffic Safety Administration (NHTSA) involving analysis of nearly 2000 fatal accident cases, found 6.7 percent of drivers positive for marijuana. In more than two-thirds of those, alcohol was present and may have been the primary contributor to the fatal outcome.⁷⁸

To accurately assess marijuana's contribution to fatal crashes, the positive rate among deceased drivers would have to be compared to the positive rate from a random sample of drivers not involved in fatal accidents. Since the rate of past-month marijuana use for Americans above the legal driving age is about 12 percent, on any given day a substantial proportion of all drivers would test positive, particulary since marijuana's metabolites remain in blood and urine long after its psychoactive effects are finished.

A recent study found that one-third of those stopped for "bad driving" between the hours of 7 p.m. and 2 a.m. -- mostly young males -- tested positive for marijuana only.⁷⁹ To be meaningful, these test results would have to be compared to those from a matched control group of drivers.

A number of driving simulator studies have shown that marijuana does not produce the kind of psychomotor impairment evident with modest doses of alcohol.⁸⁰ In fact, in a recent NHTSA study, the only statistically significant outcome associated with marijuana was *speed reduction*.⁸¹

A recent study of *actual driving ability* under the influence of cannabis -- employing the same protocol used to test the impairment-potential of medicinal drugs -- evaluated the impact of placebo and three active THC doses in three driving trials, including one in high-density urban traffic.

Dose-related impairment was observed in drivers' ability to maintain steady lateral position. However, even with the highest dose of THC, impairment was relatively minor -- similar to that observed with blood-alcohol concentrations between .03 and .07 percent and many legal medications. Drivers under the influence of marijuana also tended to drive more slowly and approach other cars more cautiously.

While recognizing some limitations of this study, the authors conclude that "THC is not a profoundly impairing drug." $^{\!\!82}$

CLAIM #13: MARIJUANA IS A "GATEWAY" TO THE USE OF OTHER DRUGS

Advocates of marijuana prohibition claim that even if marijuana itself causes minimal harm, it is a dangerous substance because it leads to the use of "harder drugs" such as heroin, LSD, and cocaine.

THE FACTS

Most users of heroin, LSD and cocaine have used marijuana. However, most marijuana users never use another illegal drug.

Over time, there has been no consistent relationship between the use patterns of various drugs.⁸³

As marijuana use increased in the 1960s and 1970s, heroin use declined. And, when marijuana use declined in the 1980s, heroin use remained fairly stable.

For the past 20 years, as marijuana use-rates fluxuated, the use of LSD hardly changed at all.

Cocaine use increased in the early 1980s as marijuana use was declining. During the late 1980s, both marijuana and cocaine declined. During the last few years, cocaine use has continued to decline as marijuana use has increased slightly.

In 1994, less than 16 percent of high school seniors who had ever tried marijuana had ever tried cocaine -- *the lowest percentage ever recorded*. In fact, as shown below, the proportion of marijuana users trying cocaine has declined steadily since 1986, when a high of more than 33 percent was recorded.

Proportion of Marijuana Users Ever Trying Cocaine High School Seniors, 1975-1994⁸⁴

1975:	19%	1980: 27%	1985: 31%	1990: 22%
1976:	19%	1981: 28%	1986: 33%	1991: 22%
1977:	20%	1982: 27%	1987: 30%	1992: 18%
1978:	22%	1983: 28%	1988: 26%	1993: 17%
1979:	25%	1984: 29%	1989: 23%	1994: 16%

In short, there is no inevitable relationship between the use of marijuana and other drugs. This fact is supported by data from other countries. In Holland, for example, although marijuana prevalence among young people increased during the past decade, cocaine use decreased -- and remains considerably lower than in the United States. Whereas approximately <u>16 percent</u> of youthful marijuana users in the U.S. have tried cocaine, the comparable figure for Dutch youth is <u>1.8 percent</u>.⁸⁵ Indeed, Holland's policy of allowing marijuana to be purchased openly in government-regulated "coffee shops" was designed specifically to separate young marijuana users from illegal markets where heroin and cocaine are sold.⁸⁶

CLAIM #14: DUTCH MARIJUANA POLICY HAS BEEN A FAILURE

While American critics of marijuana prohibition often point to Holland as a model for an alternative policy, prohibition's supporters claim that Holland's permissiveness has had disasterous consequences, including escalating rates of drug use among youth.

THE FACTS

In 1976, following the recommendations of two national commissions, the Dutch government revised many aspects of its drug policy. While not legalizing marijuana, it adopted an "expediency principle," which directed police and prosecutors to ignore retail sale to adults as long as the circumstances of the sale do not constitute a public nuisance.

This change in policy was based on several factors, including:

- --a principle of tolerance toward alternative lifestyles
- --a finding that, compared to other illegal drugs, marijuana poses little risk to users
- --a desire to protect marijuana users from the marginalization that accompanies arrest and prosecution
- --a belief that separating the retail markets for "soft" and "hard" drugs decreases the likelihood that marijuana users will experiment with cocaine or heroin

Following the policy change, marijuana sales emerged openly in coffee shops, which were required to follow a set of regulations, including a ban on advertising, sale of no more than 30 grams at a time, and a minimum purchase age of 18. The sale of other drugs on the premises is strictly prohibited, and constitutes grounds for immediate closure by the police. Local officials were also authorized to create additional regulations to protect the interests of the community -- for example, limiting the number of coffee shops concentrated in any one area.⁸⁷

Since liberalization, marijuana use has increased in the Netherlands, although rates remain similar to those in neighboring European countries, and are generally lower than those in the United States.

MARIJUANA DUTCH YOU (ages 12-18)	A USE AMONG JTH) ⁸⁸	USE AMONO AMERICAN (ages 12-17)	G YOUTH ⁸⁹	USE AMONG AMERICAN YOUTH (high school seniors) ⁹⁰		
ever used p	ast month	ever used p	ast month	ever used p	ast month	
1984 4.8%	2.3%	1985 23.2%	11.2%	1985 54.2%	25.7%	
1988 8.0	3.1	1988 17.4	6.4	1988 47.2	18.0	
1992 13.6	6.5	1993 11.7	4.9	1993 35.3	15.5	

While marijuana use-rates have increased in Holland, cocaine use-rates have not -indicating that separation of the "hard" and "soft" drug markets has prevented a "gateway effect" from developing. In 1992, about 1.5% of 12 to 18 year-olds had *ever tried cocaine* and only .3% had *used it in the past month*.⁹¹

Although there are some Dutch critics of Holland's liberalized marijuana policy, the government's official position remains steadfastly supportive of the 1976 initiative that decriminalized possession and retail sale.⁹²

⁵ <u>Preliminary Estimates From the 1993 National Household Survey on Drug Abuse</u>, Rockville, MD: U.S. Department of Health and Human Services (1994).

¹ Grinspoon, L., <u>Marijuana Reconsidered</u>, Cambridge: University of Harvard Press (1971); Kaplan, J., <u>Marijuana: The new Prohibition</u>, New York: World Publishing Company (1970); Brecher, E.M., <u>Licit and Illicit Drugs</u>, Boston: Little, Brown and Company (1972).

² U.S. National Commission on Marijuana and Drug Abuse, <u>Marijuana: A Signal of Misunderstanding</u>, Washington, DC: U.S. Government Printing Office (1972); Canadian Government's Commission of Inquiry, <u>The Non-Medical Use of Drugs: Interim Report</u>, Ottowa (1970).

³ Himmelstein, J., <u>The Strange Career of Marijuana: Politics and Ideology of Decriminalization in America</u>, Westport, CT: Greenwood Press (1983); Single, E.W., "The Impact of Marijuana Decriminalization: An Update," <u>Journal of Public health Policy</u> 10:456-66 (1989).

⁴ Carter, W.E. (ed), <u>Cannabis in Costa Rica: A Study of Chronic Marijuana Use</u>, Philadelphia: Institute for Study of Human Issues (1980); Rubin, V. and Comitas, L., <u>Ganja in Jamaica</u>, The Hague: Mouton (1975); Stefanis, C. et al, <u>Hashish: Studies of Long Term Use</u>, New York: Raven Press (1977).

⁶ Johnston, L.D. et al, Monitoring the Future, Ann Arbor: University of Michigan Institute for Social Research (1994).

- ⁷ Ennett, S.T, et al, "How Effective is Drug Abuse Resistance Education? A Meta-Analysis of Project DARE Outcome Evaluations," <u>American Journal of Public Health</u> 84:1394-1401 (1994).
- ⁸ ElSohly, M.A. et al, "Constituents of Cannabis Sativa L XXIV: The Potency of Confiscated Marijuana, Hashish, and Hash Oil Over a Ten-year Period," Journal of Forensic Sciences 29:500-14 (1984).

⁹ Perry, D., "Street Drug Analysis and Drug Use Trends, Part II, 1969-1976," <u>PharmChem Newsletter</u> 6 (1977).

- ¹⁰ Rubin, V., "Cross-Cultural Perspectives on Therapeutic Uses of Cannabis," pp 1-18 in S. Cohen and R.C. Stillman (eds), <u>The Therapeutic Potential of Marijuana</u>, New York: Plenum Medical Book Company (1976).
- ¹¹ Chang, A.E. et al, "Delta-Nine-Tetrahydrocannabinol as an Antiemetic in Cancer Patients Receiving High-Dose Methotrexate: A Prospective Randomized Evaluation, <u>Annals of Internal Medicine</u> 91: 819-24 (1979).
- ¹² Hepler, R.S. and Frank, I.R., "Marijuana Smoking and Intracular Pressure," Journal of the American Medical Association 217: 1392 (1971).

- ¹⁴ Grinspoon, L. and Bakalar, J.B, <u>Marijuana: The Forbidden Medicine</u>, New Haven: Yale University Press (1993).
- ¹⁵ Vinciguerra, V. et al, "Inhalation Marijuana as an Antiemetic for Cancer Chemotherapy," <u>New York State Journal of Medicine</u> 85:525-27 (1988); Dansac, D., "In the Matter of Marijuana Rescheduling Petition," Affidafit filed in Drug Enforcement Adminstration Hearings, Docket 86-22 (1987).
- ¹⁶ Doblin, R. and Kleiman, M.A.R., "Marijuana as an Anti-Emetic Medicine: A Survey of Oncologists' Attitudes and Experiences," <u>Journal of Clinical Oncology</u> 19: 1275-1290 (1991).
- ¹⁷ Agurell, S. et al, "Pharmacokinetics and Metabolism of Delta-1-Tetrahydrocannabinol and Other Cannabinoids with Emphasis on Man," <u>Pharmacological Reviews</u> 38: 21-43 (1986).
- ¹⁸ Cohen, S., "Therapeutic Aspects," pp 194-225 in R.C. Petersen (ed) <u>Marijuana Research Findings: 1976</u>, Rockville, MD: National Institute on Drug Abuse (1977); Cohen, S. and Stillman, R.C. (eds), <u>The Therapeutic Potential of Marijuana</u>, New York: Plenum Medical Book Company (1976); National Institute on Drug Abuse, <u>Marijuana and Health</u>, Report to Congress (1980).
- ¹⁹ "Members of Congress Voice Support for Marijuana/AIDS Research," <u>Newsletter of the Multidisciplinary Association for Psychedelic Studies</u> 5,3 (Winter 1995).
- ²⁰ Drug Enforcement Administration, Drug Legalization: Myths and Misconceptions, Washington, DC: U.S. Department of Justice (1994).
- ²¹ Huber, G.L. et al, "The Effects of Marihuana on the Respiratory and Cardiovascular Systems," pp 3-18 in G. Chesher et al (eds), <u>Marijuana: an International Research Report</u>, Canberra: Australian Government Publishing Service (1988).
- ²² Tashkin, D.P. et al, "Longitudinal Changes in Respiratory Symptoms and Lung Function in Non-smokers, Tobacco Smokers, and Heavy, Habitual Smokers of Marijuana With or Without Tobacco," pp 25-36 in G. Chesher et al (eds), <u>Marijuana: an International</u> <u>Research Report</u>, Canberra: Australian Government Publishing Service (1988).
- ²³ Sherrill, D.L. et al, "Respiratory Effects of Non-Tobacco Cigarettes: A Longitudinal Study in General Population," <u>International Journal of Epidemiology</u> 20: 132-37 (1991).
- ²⁴ Fligiel, S.E.G. et al, "Bronchial Pathology in Chronic Marijuana Smokers: A Light Electron Microscope Study," <u>Journal of Psychoactive</u> <u>Drugs</u> 20:33-42 (1988).
- ²⁵ Doblin, R., "The MAPS/California NORML/Marijuana Waterpipe/Vaporizer Study," <u>Newsletter of the Mulpidisciplinary Association for</u> <u>Psychedelic Studies</u> 5,1 (Summer 1994).

²⁷ Lau, R.J. et al, "Phytohemagglutinin-Induced Lymphocte Transformation in Humans Receiving Delta-9-Tetrahydrocannabinol," <u>Science</u> 192: 805-07 (1976); White, S.C. et al, "Mitogen-Induced Blastogenetic Responses to Lymphocytes from Marijuana Smokers," <u>Science</u> 188: 71-72 (1975).

¹³ Petro, D.J., "Marijuana as a Therapeutic Agent for Muscle Spasm or Spasticity," <u>Psychosomatics</u> 21: 81-85 (1980).

²⁶ Nahas, G.G. et al, "Inhibition of Cellular Mediated Immunity in Marijuana Smokers," <u>Science</u> 183:419-20 (1974).

- ²⁸ Wallace, J.M. et al, "Peripheral Blood Lymphocyte Subpopulations and Mitogen Responsiveness in Tobacco and Marijuana Smokers," Journal of Psychoactive Drugs 20:9-14 (1988).
- ²⁹ Mishkin, E.M. and Cabral, G.A., "Delta-9-Tetrahydrocannabinol Decreases Host Resistance to Herpes Simplex Virus Type 2 Vaginal Infection in the BGC3F1 Mouse," Journal of General Virology 66:2539-49 (1985).
- ³⁰ Carter, W.E. (ed), <u>Cannabis in Costa Rica: A Study of Chronic Marijuana Use</u>, Philadelphia: Institute for Study of Human Issues (1980); Rubin, V. and Comitas, L., <u>Ganja in Jamaica</u>, The Hague: Mouton (1975); Stefanis, C. et al, <u>Hashish: Studies of Long Term</u> <u>Use</u>, New York: Raven Press (1977).
- ³¹ Coates, R.A. et al, "Cofactors of Progression to Acquired Immunodeficiency Syndrome in a Cohort of Male Sexual Contacts of Men with Immunodeficiency Virus Disease," <u>American Journal of Epidemiology</u> 132: 717-22 (1990).
- ³² Plasse, T.F. et al, "Recent Clinical Experience with Dronabinol," <u>Pharmacology Biochemistry and Behavior</u> 40:695-700 (1991).
- ³³ Lynn, A.B. and Herkenham, M., "Localization of Cannabinoid Receptors and Nonsaturable High Density Cannabinoid Binding Sites in Peripheral Tissues of the Rat: Implications for Receptor-Mediated Immune Modulation by Cannabinoids," <u>Journal of Pharmacology</u> <u>and Experimental Therapeutics</u> 268: 1612-23 (1994).
- ³⁴ Kolodny, R.C. et al, "Depression of Plasma Testosterone Levels After Chronic Intensive Marijuana Use," <u>New England Journal of Medicine</u> 290:872-74 (1974).
- ³⁵ Kolodny, R.C. et al, "Depression of Plasma Testosterone with Acute Marijuana Administration," pp 217-25 in M.C. Braude and S. Szara (eds), <u>Pharmacology of Marijuana</u>, New York: Raven Press (1976).
- ³⁶ Mendelson, G.D. et al, "Plasma Testosterone Levels Before, During, and After Chronic Marijuana Smoking," <u>New England Journal of Medicine</u> 291:1051-55 (1975); Schaefer, C.F. et al, "Normal Plasma Testosterone Concentrations After Marijuana Smoking," <u>New England Journal of Medicine</u> 292:867-68 (1975).
- ³⁷ Hembree, W.C. et al, "Changes in Human Spermatozoa Associated with High Dose Marijuana Smoking," pp 429-39 in G.G. Nahas and W.D.M. Paton (eds), <u>Marijuana: Biological Effects</u>, Oxford: Pergamon Press (1979).
- ³⁸ Fijimoto, G.I. et al, "Effect of Marijuana Extract Given Orally on Male Rat Reproduction and Gonads," <u>Proceedings of Sixth Annual Meetings of Endocrinology Society</u> (1978); Okey, A.B. and Truant, G.S., "Cannabis Demasculinizes Rats But Is Not Estrogenic," <u>Life Sciences</u> 17:1113-18 (1975).
- ³⁹ Block, R.I. et al, "Effects of Chronic Marijuana Use on Testosterone, Luteinizing Hormone, Follicle Stimulating Hormone, Prolactin and Cortisol in Men and Women," <u>Drug and Alcohol Dependence</u> 28:121-8 (1991).
- ⁴⁰ Smith, C.G. and Asch, R.H., "Acute, Short-Term, and Chronic Effects of marijuana on the Female Primate Reproductive Function," pp 82-96 in M.C. Braude and J.P. Ludford (eds), <u>Marijuana Effects on the Endocrine and Reproductive Systems</u>, Rockville, MD: Department of Health and Human Services (1984).
- ⁴¹ Smith, C.G. et al, "Tolerance Develops to the Disruptive Effects of Delta 9 Tetrahydrocannabinol on the Primate Menstrual Cycle," <u>Science</u> 219:1453-55 (1983).
- ⁴² Copeland, K.C. et al, "Marijuana Smoking and Pubertal Arrest," Journal of Pediatrics 96:1079-80 (1980).
- ⁴³ Tennes, A., "Effects of Marijuana on Pregnancy and Fetal Development in the Human," pp 115-23 in M.C. Braude and J. P, Ludford (eds), <u>Marijuana Effects on the Endocrine and Reproductive Systems</u>, Rockville, MD: Department of Health and Human Services (1984).
- ⁴⁴ Astley, S., "Analysis of Facial Shape in Children Gestationally Exposed to Marijuana, Alcohol, and/or Cocaine," <u>Pediatrics</u> 89:67-77 (1992); Day, N. et al, "Prenatal Marijuana Use and Neonatal Outcome," <u>Neurotoxicology and Teratology</u> 13:329-34 (1992); Linn, S. et al, "The Association of Marijuana Use with Outcome of Pregnancy," <u>American Journal of Public Health</u> 73:1161-64 (1983).
- ⁴⁵ Hayes, J. et al, "Newborn Outcomes with Maternal Marijuana Use in Jamaican Women," <u>Pediatric Nursing</u> 14(2):107-10 (1988); Streissguth, A.P. et al, "IQ at Age 4 in Relation to Maternal Alcohol Use and Smoking During Pregnancy," <u>Developmental Psychology</u> 25: 3-11 (1989); Richardson, G.A. et al, "The Effect of Prenatal Alcohol, Marijuana and Tobacco Exposure on Neonatal Behavior," <u>Infant Behavioral Development</u> 12: 199-209 (1989); O'Connell, C.M. and Fried, P.A., "Prenatal Exposure to Cannabis: A Preliminary Report of Postnatal Consequences in School-Age Children," <u>Neurotoxicology and Teratology</u> 13: 631-39 (1991); Fried, P.A. et al, "60- and 72-Month Follow-Up of Children Prenatally Exposed to Marijuana, Cigarettes and Alcohol," <u>Journal of Developmental Behavior and Pediatrics</u> 13: 383-91 (1992); Dreher, M.C. et al, "Prenatal Exposure and Neonatal Outcomes in Jamaica: An Ethnographic Study," <u>Pediatrics</u> 93: 254-60 (1994).

- ⁴⁶ Buckley, J.D. et al, "Occupational Exposure of Parents of Children with Acute Nonlymphocytic Leukemia: A Report from the Children's Cancer Study Group," <u>Cancer Research</u> 49:4030-37 (1989).
- ⁴⁷ Fried, P.A., "Postnatal Consequences of Maternal Marijuana Use," pp 61-72 in T.M. Pinkert (ed), <u>Current Research on the Consequences of maternal Drug Abuse</u>, Rockville, MD: National Institute on Drug Abuse; Fried, P.A. and B. Watkinson, "12- and 24-Month Neurobehavioral Follow-Up of Children Prenatally Exposed to Marijuana, Cigarettes and Alcohol," <u>Neurotoxicology and Teratology</u> 10: 305-13 (1988).
- ⁴⁸ Fried, P.A. and B. Watkinson, "36- and 48-Month Neurobehavioral Follow-Up of Children Prenatally Exposed to Marijuana, Cigarettes, and Alcohol," <u>Developmental and Behavioral Pediatrics</u>11: 49-58 (1990).
- ⁴⁹ Fried, P.A. et al, "A Follow-Up Study of Attentional Behavior in 6-Year-Old Children Exposed Prenatally to Marijuana, Cigarettes, and Alcohol," <u>Neurotoxicology and Teratology</u> 14: 299-311 (1992).
- ⁵⁰ Day, N.L. et al, "Effect of Prenatal Marijuana Exposure on the Cognitive Development of Offspring at Age Three," <u>Neurotoxicology and</u> <u>Teratology</u> 16: 169-75 (1994).
- ⁵¹ Heath, B.C. et al, "Cannabis Sativa: Effects on Brain Function and Ultrastructure in Rhesus Monkeys," <u>Biological Psychiatry</u> 15:657 (1980).
- ⁵² Scallet, A.C., "Neurotoxicology of Cannabis and THC: A Review of Chronic Exposure Studies in Animals," <u>Pharmacology Biochemistry</u> <u>and Behavior</u> 40:671-82 (1991).
- ⁵³ Slikker, W. et al, "Behavioral, Neurochemical, and Neurohistological Effects of Chronic Marijuana Smoke Exposure in the Nonhuman Primate," pp 219-74 in L. Murphy and A. Bartke (eds), <u>Marijuana/Cannabinoids Neurobiology and Neurophysiology</u>, Boca Raton: CRC Press (1992).
- ⁵⁴ Weckowicz, T.E. et al, "Effect of Marijuana on Divergent and Convergent Production Cognitive Tests," Journal of Abnormal Psychology 84:386-98 (1975); Hooker, W.D., and Jones, R.T., "Increased Susceptibility to Memory Intrusions and the Stroop Interference Effect During Acute Marijuana Intoxication," <u>Psychopharmacology</u> 91: 20-24 (1987); Waskow, I.E. et el, "Psychological Effects of Tetrahydrocannabinol," <u>Archives of General Psychiatry</u> 22: 97-107 (1970); Dornbush, R.L. and Kokkevi, A., "Acute Effects of Cannabis on Cognitive, Perceptual, and Motor Performance in Chronic Hashish Users," <u>Annals of the New York Academy of Sciences</u> 282: 213-22 (1976).
- ⁵⁵ Darley, C.F. et al, "Marijuana Effects on Long-Term Memory Assessment and Retrieval," <u>Psychopharmacology</u> 52:239-41 (1977); Abel, E.L., "Retrieval of Information After Use of Marijuana," pp 121-24 in E.L. Abel (ed) <u>The Scientific Study of Marijuana</u>, Chicago: Nelson-Hall Publishers (1976); Abel, E.L., "Marijuana and Memory: Acquisition or Retrieval?" pp 125-32 in E.L. Abel (ed) <u>The Scientific Study of Marijuana</u>, Chicago: Nelson-Hall Publishers (1976).
- ⁵⁶ Abel, E.L., "Marijuana and Memory: Acquisition or Retrieval?" pp 125-32 in E.L. Abel (ed) <u>The Scientific Study of Marijuana</u>, Chicago: Nelson-Hall Publishers (1976); Miller, L. et al, "Effects of Marijuana on Recall of Narrative Material and Stroop Colour-Word Performance," pp 117-20 in E.L. Abel (ed) <u>The Scientific Study of Marijuana</u>, Chicago: Nelson-Hall Publishers (1976); Dornbush, R.L. et al, "Marijuana, Memory, and Perception," pp 133-40 in E.L. Abel (ed) <u>The Scientific Study of Marijuana</u>, Chicago: Nelson-Hall Publishers (1976); Dornbush, R.L. et al, "Marijuana, Memory, and Perception," pp 133-40 in E.L. Abel (ed) <u>The Scientific Study of Marijuana</u>, Chicago: Nelson-Hall Publishers (1976).
- ⁵⁷ Ferraro, D.P., "Acute Effects of Marijuana on Human Memory and Cognition," pp 98-119 in R.C. Petersen (ed) <u>Marijuana Research Findings: 1980</u>, Rockville, MD: National Institute on Drug Abuse (1980).
- ⁵⁸ Satz, P. et al, "Neuropsychologic, Intellectual, and Personality Correlates of Chronic Marijuana Use in Native Costa Ricans," <u>Annals of the New York Academy of Sciences</u> 282: 266-306 (1976); Grant, I. et al, "A Neuropsychological Assessment of the Effects of Moderate Marijuana Use," <u>Journal of Nervous and Mental Disease</u> 156: 278-80 (1973); Knights, R., "Psychological Test Results," pp 111-20 in V. Rubin and L. Comitas (eds), <u>Ganja in Jamaica</u>, The Hague: Mouton (1975); Page, J.B., "Psychosociocultural Perspectives on Chronic Cannabis Use: The Costa Rican Follow-Up," <u>Journal of Psychoactive Drugs</u> 20: 57-65 (1988); Carlin, A.S. and Trupin, E.W., "The Effect of Long-Term Chronic Marijuana Use on Neuropsychological Functioning," <u>International Journal of the Addictions</u> 12:617-24 (1977).
- ⁵⁹ <u>Preliminary Estimates from the 1993 National Household Survey on Drug Abuse</u>, Rockville, MD: U.S. Department of Health and Human Services (1994).
- ⁶⁰ Johnston, L.D. et al, <u>Drug Use Among American High School Seniors, College Students and Young Adults, 1975-1990.Vol II</u>, Rockville, MD: U.S. Department of Health and Human Services (1991), p 31.
- ⁶¹ Jones, R.T. et al, "Clinical Studies of Cannabis Tolerance and Dependence," <u>Annals of the New York Academy of Sciences</u> 282:221-39 (1976).

- ⁶² Stefanis. C. et al, "Experimental Observations of a 3-Day Hashish Abstinence Period and Reintroduction of Use," <u>Annals of the New</u> <u>York Academy of Sciences</u> 282:113-20 (1976); Cohen, S. et al, "The 94-Day Study," pp 621-26 in M.C. Braude and S. Szara (eds), <u>The Pharmacology of Marijuana</u>, New York: Raven Press (1976).
- ⁶³ Deneau, G.A. and Kaymakcalan, S., "Physiological and Psychological Dependence to Synthetic Delta-9-Tetrahydrocannabinol (THC) in Rhesus Monkeys," <u>Pharmacologist</u> 13:246 (1971).
- ⁶⁴ Martin, B.R., "Marijuana: What It Is and What It Does," Presentation at NIDA's National Conference on Marijuana Use Prevention, Treatment and Research," Arlington, VA (July 1995).
- ⁶⁵ Gold, M.S., <u>The Good News About Drugs and Alcohol</u>, New York: Villard Books (1991).
- ⁶⁶ Jacobs, J.B. and Zimmer, L., "Drug Treatment and Workplace Drug Testing: Politics, Symbolism and Organizational Dilemmas," <u>Behavioral Sciences and the Law</u> 9:345-60 (1991).
- ⁶⁷ <u>Annual Emergency Room Data, 1990</u>, National Institute on Drug Abuse Statistical Series, Series I, Number 10-A, Rockville, MD: National Institute on Drug Abuse (1991); <u>Annual Emergency Room Data, 1992</u>, National Institute on Drug Abuse Statistical Series, Series I, Number 12-A, Rockville, MD: National Institute on Drug Abuse (1993); <u>Preliminary Estimates from the Drug Abuse Warning Network</u>, Advance Report Number 8, Rockville, MD: National Institute on Drug Abuse (1994).
- ⁶⁸ McGlothlin, H.W. and West, L.J., "The Marijuana Problem: An Overview," <u>American Journal of Psychiatry</u> 125:1126-34 (1968); Smith, D.E., "The Acute and Chronic Toxicity of Marijuana," <u>Journal of Psychedelic Drugs</u> 2:37-48 (1968).
- ⁶⁹ Brill, N.O. and Christie, R.L., "Marijuana Use and Psychosocial Adaptation," <u>Archives of General Psychiatry</u> 31:713-19 (1974); Kupfer, D.J. et al, "A Comment on the Amotivational Syndrome in Marijuana Smokers," <u>American Journal of Psychiatry</u> 130:1319-22 (1973).
- ⁷⁰ Kleinman, P.H. et al, "Daily Marijuana Use and Problem Behaviors Among Adolescents," <u>International Journal of Addictions</u> 23:87-107 (1988).
- ⁷¹ Mellinger, G.D. et al, "Drug Use, Academic Performance, and Career Indecision: Longitudinal Data in Search of a Model," pp 157-77 in D.B. Kandel (ed), <u>Longitudinal Research on Drug Use: Empirical Findings and Methodological Issues</u>, Washington, DC: Hemisphere (1978).
- ⁷² Mellinger, G.D. et al, "The Amotivational Syndrome and the College Student," <u>Annals of the New York Academy of Sciences</u> 282:37-55 (1976).
- ⁷³ Miranne. H.C., "Marijuana Use and Achievement Orientations of College Students," <u>Journal of Health and Social Behavior</u> 20:194-99 (1979).
- ⁷⁴ Cohen, S., "The 94-Day Cannabis Study," Annals of the New York Academy of Sciences 282:211-20 (1976).
- ⁷⁵ Mendelson, J.H. et al, "The Effects of Marijuana Use on Human Operant Behavior: Individual Data," pp 643-53 in M.C. Braude and S. Szara (eds), <u>The Pharmacology of Marijuana, Vol 2</u>, New York: Raven Press (1976).
- ⁷⁶ Campbell, I., "The Amotivational Syndrome and Cannabis use with Emphasis on the Canadian Scene," <u>Annals of the New York</u> <u>Academy of Sciences</u> 282:33-36.
- ⁷⁷ McBay, A.J. and Owens, S.M., "Marijuana and Driving," pp 257-63 in L.S. Harris (ed) <u>Problems of Drug Dependence 1980</u>, Washington, DC: U.S. Government Printing Office (1981); Teale, J.D. et al, "The Incidence of Cannabinoids in Fatally Impaired Drivers: An Investigation by Radioimmunoassay and High Pressure Liquid Chromatography," <u>Journal of the Forensic Science</u> <u>Society</u> 17:177-83 (1978).
- ⁷⁸ Terhune, K.W. et al, <u>The Incidence and Role of Drugs in Fatally Injured Drivers</u>, Washington, DC: Department of Transportation (1994).
- ⁷⁹ Brookoff, D. et al, "Testing Reckless Drivers for Cocaine and Marijuana," <u>New England Journal of Medicine</u> 331:518-22 (1994).
- ⁸⁰ Kv'alseth, T.O., "Effects of Marijuana on Human Reaction Time and Motor Control," <u>Perceptual and Motor Skills</u> 45:935-39 (1977); Hansteen, R.W. et al, "Effects of Cannabis and Alcohol on Automobile Driving and Psychomotor Tracking," <u>Annals of New York</u> <u>Academy of Sciences</u> 282:240-56 (1976); Moskowitz, H. et al, "Marijuana: Effects on Simulated Driving Performance," <u>Accident</u> <u>Analysis and Prevention</u> 8:45-50 (1976); Moskowitz, H. et al, "Visual Search Behavior While Viewing Driving Scenes Under the Influence of Alcohol and Marijuana," <u>Human Factors</u> 18:417-31 (1976).

- ⁸¹ Stein, A.C. et al, <u>A Simulator Study of the Combined Effects of Alcohol and Marijuana on Driving Behavior -- Phase II</u>, Washington, DC: U.S. Department of Transportation (1983).
- ⁸² Robbe, H. and O'Hanlon, J., Marijuana and Actual Driving Performance, Washington, DC: Department of Transportation (1993).
- ⁸³ U.S. Department of Health and Human Services, <u>National Household Survey on Drug Abuse: Main Findings 1990</u>; U.S. Department of Health and Human Services, <u>Preliminary Estimates from the 1993 National Household Survey on Drug Abuse</u>.
- ⁸⁴ Johnston, L.D. et al, <u>Monitoring the Future</u>, Ann Arbor: University of Michigan Institute for Social Research (1994).
- ⁸⁵ Cohen, P.D.A., <u>Cannabisgebruikers in Amsterdam</u>, Jaarbeurs Congrescentrum Utrecht (1995).
- ⁸⁶ Leuw, E. and Marshall, I.H. (eds)., <u>Between Prohibition and Legalization: The Dutch Experiment in Drug Policy</u>, Amsterdam: Kugler Publications (1994).
- ⁸⁷ Netherlands Institute for Alcohol and Drugs, "Cannabis Policy Fact Sheet," <u>Netherlands Alcohol and Drug Report</u> 1 (1995); Leuw, E., "Initial Construction and Development of the Official Dutch Drug Policy," pp 23-40 in E. Leuw and I.H. Marshall (eds), <u>Between</u> <u>Prohibition and Legalization: The Dutch Experiment in Drug Policy</u>, Amsterdam: Kugler Publications (1994).
- ⁸⁸ de Zwart, W.M. et al, <u>Key Data: Smoking, Drinking, Drug Use and Gambling Among Pupils Aged 10 Years and Older</u>, Utrecht: Netherlands Institute on Alcohol and Drugs (1994).
- ⁸⁹ National Household Survey on Drug Abuse: Main Findings 1990, Rockville, MD: National Institute on Drug Abuse (1991); Preliminary Estimates from the 1993 Household Survey on Drug Abuse. Advance Report Number 7, Rockville, MD: National Institute on Drug Abuse (1994).
- ⁹⁰ National Survey Results on Drug Use, from the Monitoring the Future Study, 1975-1993, Volume I, Rockville, MD: National Institute on Drug Abuse (1994).
- ⁹¹ de Zwart, W.M. et al, <u>Key Data: Smoking, Drinking, Drug Use and Gambling Among Pupils Aged 10 Years and Older</u>, Utrecht: Netherlands Institute on Alcohol and Drugs (1994).
- ⁹² "The Drug Policy in the Netherlands," Joint Report of the Ministry of Welfare, Health and Cultural Affairs and the Ministry of Justice (1994).