ALASKA LEGISLATURE COMMITTEE FILES, 2005-2006 8672

11773 SENATE HEALTH,EDUCATION & SOCIAL SERVICES

(8). marijuana smoke contains more carcinogenic hydrocarbons than tobacco smoke and a person who smokes several marijuana cigarettes a week may be taking in as many cancer causing chemicals as someone who smokes a full pack of cigarettes every day.

- Tab "D", and Addendum. *BRITISH LUNG FOUNDATION, A SMOKING GUN*

(9). potency of marijuana …

- Tab "A" – Mississippi Monitoring Project Graphs and Charts

(10). treatment admissions …

- Tab "C" – Treatment admissions data from SAMHSA.

(11). Alaska ranks in top 10 for indoor growing states …

- Tab "A" – DEA Domestic Cannabis Eradication/Suppression Program Statistical Reports.

(12). A large percentage of persons arrested in this state, including adults and juveniles who commit violent offenses, have marijuana in their system at the time of arrest.

- Tab "A" and Tab "B"

(13). marijuana use by a parent has been, and will continue to be, a major contributing factor to children having easy access to and using marijuana.

- Tab "C" – State of Alaska Adolescent Health Survey, 1990 (Children in homes where parents used marijuana frequently were more likely than children in homes where parents did not use marijuana frequently to use themselves. (22.6% vs. 5%); Among youth perceiving parents would strongly disapprove of using marijuana only 5.4% had used marijuana in the past month vs. 28.7% in homes where the youth believed that their parents would only somewhat disapprove or neither approve or disapprove of their trying marijuana.

(14). criminal penalties for marijuana possession and education efforts are effective in reducing marijuana use and limiting its access by children.

- Addendum.

Reply Card
Safety City Brochure

Press Release
Proclamation
Key Messages
Strategy
Operation ABC:

Success Stories
Rx for Prevention
Strides for Safety
Youth Fatalities

Materials Catalog
State Coordinators
Regional Offices

Down the Road
Don't Get Towed
Booster Seats
Kids Aren't Cargo
Fast Lane

Zero Tolerance
Alcohol Poisoning

Air Bag Safety
Occupant Protection
Primary Safety Belt
Patterns for Life
Road to Danger?

Bus Safety Points
Wise Cycling
Ped. Safety Points
Prevent Ped. Crashes

Expect a Train

Drug Impaired Driving

Did you know?

- If you are in the 5- to 24-year-old age group, you have a much greater chance of dying in a motor vehicle crash than dying from homicide, suicide, a fall, cancer, or heart disease.
- The Bureau of the Census estimates that there were more than 22 million young people ages 15 to 20 in the United States in 1996. The number of licensed drivers in this age group was estimated at just under 12 million. By the year 2005 the youth population is expected to have increased by almost 14 percent.
- There is a Presidential Initiative that establishes zero tolerance for drugs when possessed, used, or abused by youth.
- Alcohol, marijuana, cocaine, and inhalants are drugs commonly abused by youth.
- Research shows that marijuana is harmful to the brain, heart, lungs, and immune system. It limits learning, memory, perception, judgment, and complex motor skills like those needed to drive a vehicle.
- People under the influence of cocaine become easily confused and lose the ability to concentrate or to think clearly for any length of time.
- Inhalants can cause damage to the heart, kidneys, liver, brain, and other organs, depending on the types of inhalants used.
- Alcohol and other drugs create a serious highway safety problem among the general driving population. The National Highway Traffic Safety Administration (NHTSA) estimates that drugs are used by approximately 10 to 22 percent of drivers involved in crashes, often in combination with alcohol.
- In a 1990-91 NHTSA study of 1,882 fatally injured

Women Aren't Attracted

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drivers from 7 states, alcohol was found in 51.5 percent of the drivers, and other drugs were found in 17.8 percent of the drivers.

- Studies of drivers injured in crashes or cited for traffic violations also show that many of those drivers have used drugs. In an ongoing NHTSA study of non-fatally injured drivers in Rochester, New York, 12 percent of all drivers tested positive for drugs other than alcohol (43 of 360 cases), and 23.5 percent of drivers less than 21 years old tested positive for drugs other than alcohol (4 of 17 cases). Studies of drivers taken for medical treatment have shown positive drug rates ranging from below 10 percent to as high as 30 to 40 percent. Studies of drug incidence among drivers arrested for motor vehicle offenses have found drugs in 15 to 50 percent of drivers. The higher rates typically are more prevalent among drivers who have been arrested for impaired or reckless driving but who were not impaired by alcohol, as shown by low blood alcohol concentration (BAC) levels.

Self-reported information confirms that teenagers use marijuana in driving situations. PRIDE's 9th Annual Survey of Students, an annual self-administered questionnaire given to students in grades 6 through 12, sampled 129,560 students in 26 states during the 1995-96 school year. Twelfth grade students who reported that they smoke marijuana in a car equaled 20 percent; 16.3 percent drink beer in a car; 12.5 percent drink liquor in a car; and 9.5 percent drink wine coolers in a car. When all senior high school students were asked if and where they use marijuana, they reported: 23.9 percent at a friend's house, 15.9 percent in a car, 11.6 percent at home, 6.5 percent at school, and 19.5 percent in other places.

The evidence from nationally recognized surveys clearly and consistently indicates that drug use by youth is well below the peak levels attained in the late 1970's, but it has risen steadily in the 1990's.

Have you thought about this?

It is illegal in all states to drive a motor vehicle under the influence of alcohol, drugs other than alcohol, or a combination of alcohol and other drugs.

The Drug Evaluation and Classification (DEC) Program trains

law enforcement officers in advanced impaired driving detection techniques to remove drug impaired drivers from the highway.

The DEC process is a systematic, standardized, post-arrest procedure used to determine whether a suspect is impaired by one or more categories of drugs. It is a systematic process because it is based on a variety of observable signs and symptoms proven to be reliable indicators of drug impairment.

Officers who have completed the extensive DEC training program are certified as Drug Recognition Experts (DRE's). DRE's learn to observe a suspect's appearance, behavior, performance of psychological tests, eye movements in different lighting conditions, and vital signs to ascertain what category or categories of drugs are causing the impairment.

Thirty-two states using the DEC Program have officers trained to remove drug impaired drivers. Information about drug impaired driving cases and training are available for prosecutors and judges.

Following are some examples of DRE's effectiveness in removing young drivers who were impaired by drugs:

- In 1995, 8 percent of the evaluations conducted in New Mexico were on arrestees under age 21 (the state does not routinely test for marijuana).
- In a study of 500 DRE cases in Arizona, 10.4 percent of arrestees were under age 21.
- In 1996, Maine reported 27.6 percent of the DRE evaluations conducted were on subjects under age 21.
- In the first 5 months of 1996, New York State Police data indicate that 29.8 percent of DRE evaluations were under age 21.
- In the first 9 months of 1996, Oregon State Police reported that 14.6 percent of the evaluations were conducted on subjects under age 21.

Take action:

- Evaluate the effectiveness of your state laws that prevent youth from possessing and using alcohol and other drugs.

- Provide materials that convey practical information about drugs, the health risks of drug use, how drugs impede safe driving, and the driving sanctions for drug impaired driving and other drug law violations
- Implement an intervention program for drug impaired drivers that incorporates assessment, drug education, counseling, and other treatment as needed.
- Contact your State Highway Safety Office or NHTSA to obtain additional information on drug impaired driving.

AN OPEN LETTER TO PARENTS:

HERE'S WHAT THE EXPERTS SAY ABOUT MARIJUANA AND TEENS.

■ "Marijuana is not a benign drug. Use impairs learning and judgment, and may lead to the development of mental health problems."
- *American Medical Association*

■ "Smoking marijuana can injure or destroy lung tissue. In fact, marijuana smoke contains 50 to 70 percent more of some cancer causing chemicals than does tobacco smoke."
- *American Lung Association*

■ "Teens who are high on marijuana are less able to make safe, smart decisions about sex - including saying no. Teens who have used marijuana are four times more likely to have been pregnant or gotten someone pregnant than teens who haven't."
- *National Campaign to Prevent Teen Pregnancy*

■ "Marijuana can impair perception and reaction time, putting young drivers, their passengers and others on the road in danger. Teens, the highest risk driving population, should avoid anything that might impair their ability to operate a vehicle safely."
- *American Automobile Association*

■ "Marijuana use may trigger panic attacks, paranoia, and even psychoses, especially if you are suffering from anxiety, depression or having thinking problems."
- *American Psychiatric Association*

■ "Marijuana can impair concentration and the ability to retain information during a teen's peak learning years."
- *National Education Association*

■ "Recent research has indicated that for some people there is a correlation between frequent marijuana use and aggressive or violent behavior. This should be a concern to parents, community leaders, and to all Americans."
- *The National Crime Prevention Council*

And, according to the National Institute on Drug Abuse, marijuana can be addictive. In fact, more teens are in treatment with a primary diagnosis of marijuana dependence than for all other illicit drugs combined.

Teens say their parents are the single most important influence when it comes to drugs. Know their friends. Ask them where they are going and when they will be home. Take time to listen. Talk to your teens about marijuana.

PARENTS.
THE ANTI-DRUG.

Substance Abuse and Mental Health Services Administration
www.samhsa.gov

An Agency of the U.S. Department of Health and Human Services

SAMHSA
News Release

FOR IMMEDIATE RELEASE
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Utah Has Lowest Use of Illicit Drugs; Alaska the Highest
SAMHSA Unveils State Substance Abuse Data from 2003 National Survey on Drug Use and Health

Utah has the lowest rate of past month use of illicit drugs in the nation, as well as the lowest rate for binge drinking, the Substance Abuse and Mental Health Services Administration (SAMHSA) revealed today. Alaska has the highest rate of illegal drug use, while North Dakota has the highest rate for binging on alcohol.

The data are from "State Estimates of Substance Use from the 2002-2003 National Surveys on Drug Use and Health". SAMHSA combined two years of data from the annual National Survey on Drug Use and Health to enhance the precision of estimates for the less populous states. The report estimates state rates of use of illegal drugs, binge drinking, serious mental illness and tobacco use.

"State-by-state data is a powerful tool for policymakers at the federal, state and local levels to identify needs and target prevention and treatment resources. While we as a nation are making overall progress in reducing illicit drug use among youth, it is clear from the findings that illicit drug, alcohol and tobacco use vary substantially among states and regions," SAMHSA Administrator Charles Curie said. "To help continue to build on the gains we have made, SAMHSA announced last year a total of $230 million over five years to implement its Strategic Prevention Framework in 19 States and two territories to advance community-based programs for substance abuse prevention, mental health promotion and mental illness prevention. More awards are expected this year."

Estimates of past month use of any illicit drug ranged from a low of 6.3 percent in Utah to a high of 12.0 percent in Alaska for all persons ages 12 and older. Other states with high past month use of any illicit drug include Colorado, Montana, Oregon, Nevada, New Mexico, New Hampshire, Rhode Island, Vermont and the District of Columbia.

Utah had the lowest rate in the nation for binge alcohol use in the past month among all persons ages 12 or older, 15.9 percent. Binge alcohol use is defined as drinking five or more drinks on the same occasion on at least one day in the 30 days prior to the survey. North Dakota had the highest rate, 31.4. Colorado, Montana, South Dakota, Nebraska, Minnesota, Iowa, Wisconsin, Massachusetts and Rhode Island also had high rates of binge drinking.

The report estimates that Tennessee has the lowest rate of past year dependence on or abuse of alcohol, 6.0 percent. The highest rate of alcohol dependence or abuse is 10.8 percent in

North Dakota. The District of Columbia had the highest rate of past year illicit drug dependence or abuse, 4.0 percent. This compares with Kansas and Iowa, which had the lowest rate of dependence or abuse, 2.5 percent.

New Mexico had the highest percentage of persons ages 12 or older needing treatment for an illicit drug use problem, but not receiving it, 3.5 percent. Montana, Nebraska, New Mexico, and South Dakota all ranked in the top fifth of states for all three age groups for needing but not receiving treatment for an alcohol problem.

For specific drugs, Tennessee had the lowest rate, 7.4 percent, for marijuana use among those ages 12 and older in the past year, while Alaska had the highest rate, 16.7 percent. This compares to the national rate of 10.8 percent for marijuana use in the past year. For current use – use in the past month—Utah had the lowest rate, 4.0 percent of the population ages 12 and older, while New Hampshire had the highest rate, 10.2 percent. The national current use rate for marijuana was 6.2 percent.

The highest rate of past year cocaine use among persons ages 12 or older was found in Colorado, 3.9 percent. The lowest rate was found in Idaho, 1.6 percent. Arizona and Colorado were the only two states that ranked in the top fifth for all three age groups, 12-17, 18 to 25 and 26 or older.

Kentucky had the highest rate of past month tobacco use among persons ages 12 or older, 39.8 percent. Utah had the lowest rate, 19.7 percent.

Rhode Island had the highest rate of serious mental illness among persons ages 18 or older, 11 percent, while Hawaii had the lowest rate in the nation at 7.2 percent.

The report is available on the web at www.oas.samhsa.gov

AUTHOR: Jennifer M. Lincoln; Ron Perkins; Freddie Melton; George A. Conway
TITLE: Drowning in Alaskan Waters
SOURCE: Public Health Reports v111 p531-5 N/D '96

PUBLISHER ABSTRACT AB Objective. To enumerate drowning fatalities in Alaska in order to identify risk factors and areas for intervention. Methods. Information from death certificates, state troopers' reports, and medical examiner reports were abstracted and analyzed. Rates were calculated using 1990 census figures as denominator data. Results. There were 542 drowning fatalities in Alaska for the years 1988 to 1992. The 20-29 age group had the highest frequency and rate of drownings. The incidence rate for the state was 20 drownings per 100,000 population per year, almost 10 times higher than the overall U.S. rate of 2.11 per 100,000 per year. Incidence rates were highest among adolescent males (10-19), young adult males (20-29), Alaska Natives, and rural residents. Alaska Native males, ages 30-39 averaged 159 drownings per 100,000 per year, the highest drowning rates in the state. Conclusions. Drowning is a major public health concern in Alaska. People who fish commercially and young Native males are groups at nigh risk for drowning. Intervention efforts should be concentrated on these two populations.

Drowning is the second leading cause of injury death in Alaska.(FN1) In the United States as a whole, drowning ranks fourth among unintentional injury deaths after motor vehicle-related deaths, poisonings, and falls, based on data from the National Center for Health Statistics. Drowning is the second leading cause of injury death among children and young adults ages 1 to 24 years.(FN2)

Alaska has the highest drowning rate of any state. During the years 1984 to 1990, the United States had an age-adjusted drowning rate of 2.11 per 100,000 population per year, while Alaska experienced an age-adjusted rate of 11.03 per 100,000 per year, more than five times higher than the national rate.(FN2) A previous study of causes of death in Alaska for 1980–1989 found an average drowning rate of 22 per 100,000 per year.(FN1) For this same time period, drowning was the leading cause of injury death among Alaska Natives, with an age-adjusted drowning rate of 51.9 per 100,000 per year.(FN1) Additionally, for the years 1990 to 1992, drowning was the leading cause of occupational fatalities in Alaska, according to statistics maintained by the Alaska Field Station of the National Institute for Occupational Safety and Health.

Despite these high rates and the potential to learn about a serious national problem by studying the worst-case state, there has not been a comprehensive drowning study for the state of Alaska since 1979. In 1993, the Alaska Area Native Health Service of the Indian Health Service and the Alaska Field Station, Division of Safety Research, National Institute for Occupational Safety and Health, undertook a collaborative study to enumerate drowning fatalities in Alaska. The goal of the study was to identify potential risk factors and possible interventions.

Alaska encompasses 586,412 square miles, is twice the size of Texas, and has more coastline than the rest of the continental United States combined—6640 miles, including islands. However, the state ranks 47th among the 50 states in total road miles. Alaska has more than 3000 rivers and three million lakes, but only 13,485 miles of roads. Only five of the state's urban centers are connected by road.(FN3) Alaskans, especially in the outlying communities, use rivers and coastal waterways as highways both in the summer (by boat) and winter (as ice roads) for travel, commercial purposes, subsistence (including hunting, wood gathering, and berry picking), and recreation.

METHODS

Drowning fatalities were identified by reviewing death certificates for the state of Alaska for the years 1988 through 1992. Demographic data, cause of death (including

E-code when available), and circumstances surrounding the incident (including location, activity, use of safety equipment) were recorded from certificates that cited drowning, immersion injury, or hypothermia with immersion as a cause of death.

To validate the resultant drowning database, we compared it with a computer listing obtained from the Alaska Bureau of Vital Statistics. The listing was sorted according to the following International Classification of Diseases (ICD) External Cause Codes (E-codes):

E 830 accident to watercraft causing submersion [for example, injury resulting from vessel overturning, sinking, or burning]

E 832 other accidental submersion or drowning in water transport accidents

E 910 accidental drowning and submersion

E 954 suicide by submersion [drowning]

E 964 assault by submersion [drowning]

E 984 submersion [drowning], undetermined whether accidentally or purposely inflicted.

We also compared our drowning database for 1988–1992 with the Alaska Occupational Injury Surveillance System (AOISS) database that records occupational fatalities occurring in the state. AOISS collects information from a variety of agencies, including the United States Coast Guard, the state of Alaska's Department of Epidemiology, and the Bureau of Vital Statistics. AOISS includes data from 1990 on, so only the years 1990, 1991, and 1992 could be validated. Forty-four percent (108) of the occupational fatalities recorded in AOISS for the years 1990 to 1992 were drownings or presumptive drownings.

After the data were abstracted, the fatalities were classified by geographic location, whether the injury occurred while working, occupation of those injured at work, and activity at time of incident. Fatality rates were calculated by age group, racial categories, and gender. Denominators for the incidence rates were based on the 1990 Alaska population.(FN4) Medical examiners' and public safety officers' reports were used to assess use of personal flotation devices (PFDs) and toxicologic evidence of alcohol or drug consumption.

RESULTS

A total of 542 fatal drownings occurred in Alaskan waters from 1988 through 1992. Of these, 60% occurred during the months of May through September, while 221 (40%) occurred from October through April. The frequency of drownings varied by age and sex (see Figure 1), with the highest frequency and the highest rate in the 20-29 age group. This group accounted for 168 (31%) of the total drownings, equivalent to a rate of 35.5 drownings per 100,000 per year. The 30-39 age group also had 168 drownings (31% of the total), representing a rate of 28.6 drownings per 100,000 per year. During the five-year period, there were 497 male drowning victims (92%) and 45 females (8%). The greatest number of victims, 148 (27%) were fishing commercially when they drowned; 124 (23%) were using boats for nonrecreational purposes, and 38 (7%) deaths were attributed to falls from docks (Figure 2).

For the five-year period, we calculated the average annual drowning rate for the state as 20 per 100,000 population per year. Of the victims, 335 (62%) were white, 186 (34%) were Alaskan Native, and 21 (4%) were neither white nor Alaska Native. The drowning rate for Alaska Natives was 43 per 100,000 per year. The combined rate for all non-Natives was 15 per 100,000 per year.

Alaska Native males (78 per 100,000 per year) and females (8 per 100,000 per year) had fatal drowning rates almost three times those for non-Native males (27 per 100,000 per year) and non-Native females (2 per 100,000 per year) (Figure 3). Alaska

Native males ages 10 to 19 had a drowning rate of 74 per 100,000 per year, those ages 20 to 29 had a rate of 140 per 100,000 per year, and those in the 30 to 39 age group had a rate of 159 per 100,000 per year.

Of the drowning victims, 326 (60%) drowned in salt water and 199 (37%) drowned in fresh inland waters. The other 3% drowned in bathtubs and hot tubs. Thirty-eight percent of the drownings were work-related, 72% of these among commercial fishing industry workers.

Information on PFD use was available on fewer than 10% of the death certificates. Medical examiner and state trooper reports were requested for all of the drownings. These reports contained information on PFD use and on alcohol and drug involvement. Of the 542 total drownings detected by our surveillance, 349 drownings were not presumptive; a body was recovered and a medical examiner's report should have been filed. Of these, 186 medical examiner reports were located, with all of these victims having been tested for alcohol: 113 (61%) had detectable blood alcohol. In 94 (51%) of these people the level was above 100 milligrams per deciliter (mg/dl). Alcohol testing was completed on 54% of the Alaska Native victims and on 53% of the non-Native victims. Of the 100 Alaska Native decedents tested, 49 (49%) had detectable blood alcohol levels of more than 100 mg/dl for the 86 non-Natives tested, 41 (52%) had a blood alcohol level of more than 100 mg/dl. In addition, 162 victims were tested for illicit drugs: 18 (11%) victims had detectable cannaboids, one victim had detectable cocaine metabolites, one had detectable amphetamine metabolites, and another had detectable opiate and cannaboid metabolites. Thirteen (8%) victims had both detectable alcohol and one of the drug metabolites.

We divided the drowning incidents into census boroughs and calculated the drowning rate for each borough. The geographic region with the highest drowning rate was the Lake-peninsula Borough in Southwest Alaska that includes the villages of Illiamna, Nondalton, and Chignik. The drowning rate for this area was 160 per 100,000 per year. The Aleutians East Borough had a rate of 110 per 100,000 per year, and the Dillingham Borough and the Aleutians West Borough each had a rate of 90 per 100,000 per year.

In validating the drowning database with the AOISS database, we detected seven (1%) additional presumptive drownings that were unaccounted for in the death certificate database. We notified the Alaska Bureau of Vital Statistics of these missing certificates, and the missing cases were added to our drowning database for analysis.

DISCUSSION

The incidence of drowning is extremely high among Alaska Natives residing in rural areas and among Alaskan commercial fishing workers, likely reflecting the very high exposure to water hazards in these populations.

For the study period, the Alaskan drowning rate was approximately ten times the national incidence rate of 2.11 drownings per 100,000 population per year, and the Alaska Native population had a rate 20 times higher than the national rate.(FN1) The drowning rate of Alaska Native males was three times as high as the combined rate for non-Native males. The rate ratio of Alaska Native males (78.1 per 100,000 per year) to non-Native males (26.7 per 100,000 per year) was 2.9:1. Alaska Native males of all ages had very high rates: the rate of the 30-39 age group was 35 times higher than the national average, for the 20-29 group it was 66 times higher, and for the 10-19 group it was 75 times higher.

To combat drownings in rural native villages, the Alaska Area Native Health Service has developed programs in which flotation coats are sold at or below cost. There are currently seven active floatcoat sales programs located in hub communities across the state. More than 3500 floatcoats were sold in the first three years (1991 to 1993) of the program. The Yukon/Kuskokwim Health Corporation in southwest Alaska has been the most active of the seven programs, accounting for more than 70% of the floatcoat sales. From October 1992 to September 1993, at least 16 people in this region attested that their floatcoats saved their lives.(FN5)

Blood alcohol concentration (BAC) studies in decedents must be regarded with some caution. Blood alcohol levels in corpses can be exaggerated by alcohol produced by the decomposition process. One study showed a difference of 18% in those testing positive for alcohol depending on length of time in the water 29% positive BACs among victims who had been submerged six hours or less and 47% positive BACs among victims who had been submerged up to 12 hours.(FN6) Whether parallel differences would be observed in arctic and subarctic conditions and cold waters is unknown because fermentation is a temperature-dependent process.

The problem of alcohol interacting with cold water hazards to cause drowning is by no means limited to Alaska. A review of 36 studies on drownings from 1947 to 1986 found that 21% to 47% of those who drowned had positive BACs.(FN7) There are several reasons to suspect alcohol as a contributing factor in drownings. Alcohol may hamper the ability to avoid dangerous circumstances. The warm sensation that alcohol creates may make some victims misjudge their heat loss, resulting in hypothermia. The risk for caloric labyrinthitis, an inner ear disturbance that disorients the swimmer, may be increased by intoxication.(FN6) Alcohol may also affect sober people since they may drown as a result of an intoxicated person's actions.

Commercial fishing contributes significantly to the national and regional economies. In 1989, the United States harvested one-fifth the world's total of fish. Alaska accounts for nearly 50% by volume and almost 40% by value of the total U.S. harvest. In the 1980s, Alaska's largest private employer was the fishing industry.(FN8)

U.S. Coast Guard casualty data show that the west coast of the United States accounted for the greatest share of total vessel losses and fatalities. Alaska ranked second in the nation in total vessel losses and fatalities.(FN8)

Strategies are currently being developed to reduce commercial fishing fatalities in Alaska by correcting instability problems, such as overloading, that cause vessels to sink or capsize and by using PFDs and "man overboard" alarms to prevent workers from drowning when falling overboard. It has been previously demonstrated that when fishers who drowned or were presumed to have drowned were compared with those who survived incidents in which at least one other fisher drowned, 63% of those wearing PFDs survived but only 12% of those not wearing PFDs survived.(FN9)

Nationally, the hazards of commercial fishing have also captured the attention of Congress, which enacted the Commercial Fishing Industry Vessel Safety Act (CFIVSA, P.L. 100-424) of 1988. These safety measures were implemented between 1990 and 1993. Two of the present authors analyzed U.S. Coast Guard statistics for Alaskan commercial fishing vessel casualties from 1991 to 1994.(FN10)

The number of vessel casualties (vessels lost) has remained relatively constant, as has the number of people on board (number at risk), while remarkable progress has been made in the case-fatality rate in these vessel casualties, which has dropped from 24% in 1991 to 2% in 1994. This impressive progress in reducing mortality has occurred primarily by keeping seamen who have evacuated capsized or sinking vessels afloat and warm (using immersion suits and life rafts) and being able to locate them readily, via emergency position indicating radio beacons (EPIRB's) all of which are required by the CFIVSA.(FN10)

Some possible sources of error and limitations of our data are worth noting. Out-of-state deaths of Alaskan residents were not included in this study. However, residents of other states who drowned in Alaska, usually commercial fishing industry workers from out of state, were included. Since the denominators used to calculate rates were based on Alaskan 1990 census data, this inclusion could have resulted in artificially inflating drowning rates in the West Aleutian and East Aleutian districts, where most of these workers died. Death certificates reveal very little about circumstances surrounding the drownings.

Of the 542 people who died by drowning, 148 (27%) were commercially fishing at the time they drowned. Because most commercial fishing activities occur on salt water, more people drowned in salt water than in fresh water. Alaska Natives were most often using boats for transportation or subsistence or other non-recreational activities when they drowned. The National Institute for Occupational Safety and Health Alaska Field Station is focusing drowning prevention efforts on the commercial fishing industry—specifically on the acceptance and wearing of PFDs and the possible utility of man-overboard alarms. An ongoing surveillance system has been set up by the Alaska Field Station in collaboration with the Alaska Area Native Health Service and the Alaska Bureau of Vital Statistics to collect information on all drownings in Alaska regardless of occupational status. This database is linked to the AOISS database mentioned above.

Drowning continues to be a major public health problem in the United States and particularly in Alaska. In Alaska, further surveillance and detailed investigation of fatal and nonfatal immersion events via hospital records and medical examiners' and state troopers' reports are essential to increase information on risk factors such as alcohol and PFD usage. Studies focusing on specific geographic regions would permit more detailed analysis of the problem. Such information is critical for developing and targeting intervention efforts to reduce drownings.

Added material

Ms. Lincoln and Dr. Conway are with the Alaska Field Station, Division of Safety Research, National Institute for Occupational Safety and Health. Ms. Lincoln is an Occupational Safety and Health Specialist, and Dr. Conway is Chief of the Field Station. Mr. Perkins is Director of the Community Injury Prevention Program, Alaska Area Native Health Service. Mr. Melton, a student at East Central Oklahoma University, was a summer intern with the U.S. Public Health Service at the time of this study.

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John Tomaro and Al Zangri of the Alaska Bureau of Vital Statistics, Dr. Anne Lanier of the Indian Health Service, and Helen Peters and Anna Douglas of Arctic Investigations Program provided invaluable assistance.

Figure 1. Drownings by age and gender, Alaska, 1988–1992 (N=542)

Figure 2. Drownings by activity, Alaska, 1988–1992 (N=542)

Figure 3. Drowning rates for Alaska Natives and non-Natives by gender, Alaska, 1988–1992

FOOTNOTES

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3. Trends in Marijuana Incidence

3.1 Introduction

Estimates of marijuana incidence, or the number of new marijuana users during a given year, provide an important measure of the Nation's marijuana use problem. They can suggest emerging patterns of use, give clues about the changes in the prevalence of use, identify at-risk subgroups for targeting prevention programs, and suggest substance abuse treatment needs for the Nation.

This chapter presents incidence estimates of marijuana use based on data from the 1999 and 2000 National Household Surveys on Drug Abuse (NHSDAs). These incidence estimates are based on the NHSDA questions on age at first use, year and month of first use for recent initiates, the respondent's date of birth, and the interview date. Using this information, along with editing and imputation when necessary, an exact year, month, and day of first use was determined for each substance used by each respondent. Because these data were collected on a retrospective basis, incidence estimates were always 1 year behind the data on current use. For age-specific incidence rates, the period of exposure was defined for each respondent and age group for the time that a respondent was in an age group during a calendar year.

The average age of new users in each year also was estimated. These rates are presented in this report as the number of new marijuana users per 1,000 potential new users because they indicate the rate of new use among persons who had not yet used the drug (i.e., potential new users). More precisely, the rates are actually the number of new users per 1,000 person-years of exposure. The numerator of each rate is the number of persons in the age group who first used the drug in the year. The denominator is the person time exposure measured in thousands of years. Each person's exposure time ends on the date of first use. For age-specific estimates, exposure is limited to the time during the year that the person was in that age group. Persons who first used the drug in a prior year had zero exposure to first use in the current year, and persons who still had never used the drug by the end of the current year had 1 full year of exposure to the risk.

Because these incidence estimates were based on retrospective reports, they were subject to several biases, as discussed in Chapter 2. It is possible that some of these biases, particularly telescoping and underreporting because of fear of disclosure, may affect estimates for the most recent years more significantly. However, further analysis is needed to understand the magnitude of these biases. In addition, the estimates in this report were based on the new CAI data, and the estimation methodology for these estimates was different from that used in NHSDAs prior to 1999 (i.e., based on paper-and-pencil interviewing [PAPI] methodology). The revised methodology had an impact on age-specific rates (Gfroerer et al., in press). Thus, comparisons with prior NHSDA estimates should not be made.

Estimated trends and incidence rates of marijuana use reported in this chapter were based on the combined sample of 1999 and 2000 CAI data. These estimates are presented by the overall sample, combined age groups and gender (e.g., 12 to 14 male, 12 to 14 female, 15 to 17 male, 15 to 17 female, 18 to 20 male, 18 to 20 female, 21 or older male, and 21 or older female), and race/ethnicity (e.g., white, black, Hispanic, Asian/Pacific Islander/Native Hawaiian, American Indian/Alaska Native, and persons reporting more than one race).

3.2 Trends in Marijuana Incidence

Table 3.1 summarizes the estimated number of new marijuana users, mean age of first use, and annual incidence rates from 1999 and 2000 NHSDA data. An estimated 2.0 million Americans aged 12 or older used marijuana for the first time in 1999, which was fewer than the estimated number of new users in 1998 (approximately 2.5 million Americans), but still above the 1989 and 1990 levels (1.4 million each year). Figure 3.1 shows that the rate of marijuana initiation increased during the late 1960s and early 1970s, with a peak in 1976 and 1977 (21.0 per 1,000 potential new users). After that period, the rate of new marijuana use decreased to 8.5 in 1990, followed by an increase to 16.8 in 1996, then a decrease to 13.6 in 1999. The mean age at first use was 19 years in the early 1970s and decreased to 17 years in the 1990s.

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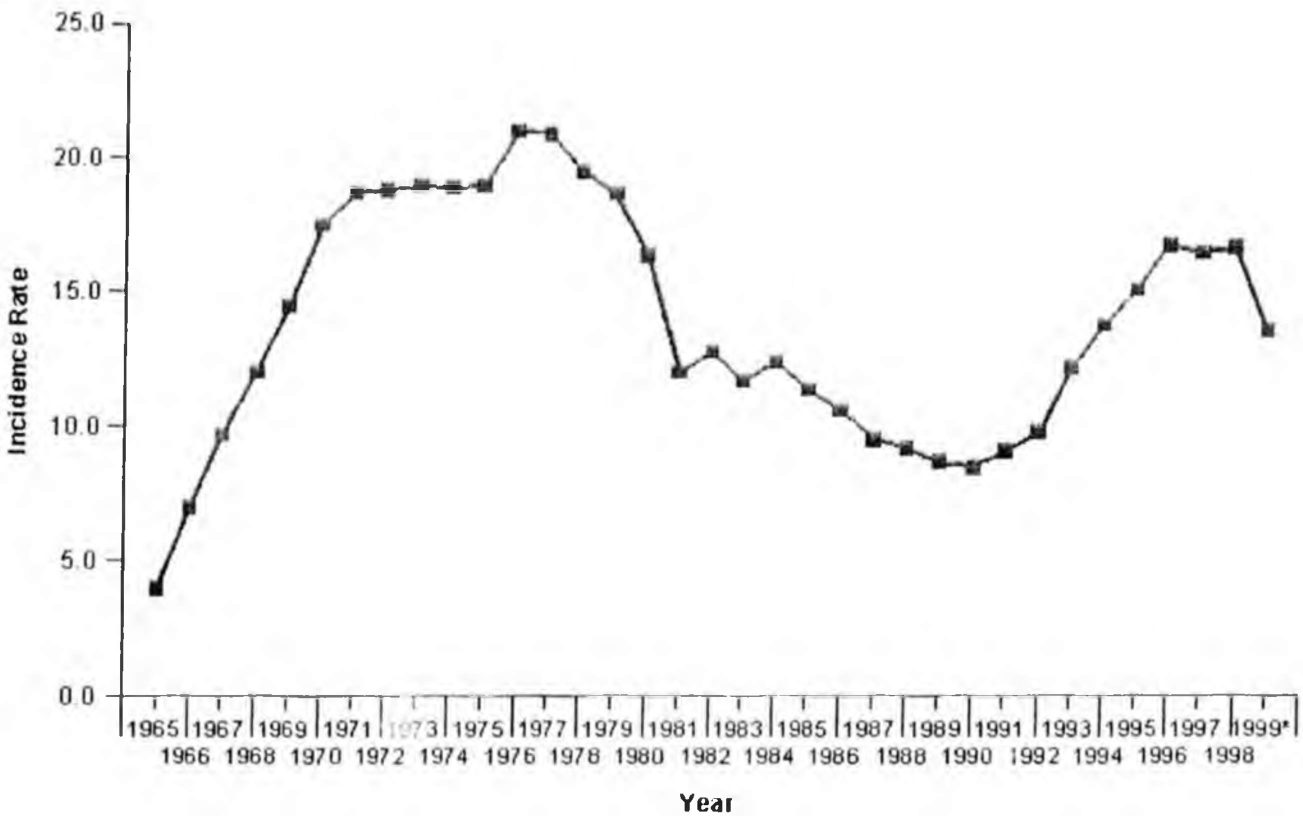
Figure 3.1 Marijuana Incidence Rates, by Year



Note: The numerator of each rate is the number of persons who first used marijuana in the year, while the denominator is the person-time exposure measured in thousands of years for persons aged 12 or older.

* Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

3.3 Trends, by Age and Gender

Over the years, rates of marijuana incidence were generally higher among males than among females (Tables 3.2 and 3.3, Figure 3.2). Among males, the rate increased dramatically from 4.9 in 1965 to 22.9 in 1971. The highest peak was noted in 1976-1977 (close to 24). After the late 1970s, incidence rates for males declined to around 10 in the late 1980s, followed by a period of increase during the 1990s to 19.3 in 1997. For females, the incidence rate increased steadily from 3.3 in 1965 to 18.9 in 1976. Similar to the pattern of males, the rate was lower during the 1980s, followed by an increase during the early 1990s. The most recent peak for females was in 1996 (15.5). For both genders, the rate in 1999 (15.5 and 12.1, respectively, for males and females) was lower than the rate in 1996-1998.

The estimated mean age at first marijuana use generally has been slightly younger in males than in females. For males, the mean age at first marijuana use ranged from 18-19 years during late 1960s to 16-17 years in recent years. For females, the mean age at first marijuana use decreased from 20 years during late 1960s to around 17 years in recent years. The average age of new marijuana users in 1999 was 16.4 years for males and 17.6 years for females.

Detailed data on age- and gender-specific incidence rates are summarized in Table 3.4. The data indicate that trends of incidence rates peaked at different periods for youths and adults. Among youths aged 12 to 17, annual incidence rates reached peaks during the late 1970s and late 1990s, and the pattern was similar for both genders. Among adults, particularly males, a peak rate of initiation was reached during the late 1960s, with rates remaining high throughout the 1970s, before dropping significantly in the 1980s. In addition, among adults aged 21 or older, the data did not show a peak in new use during the late 1990s, while persons aged 18 to 20 did.

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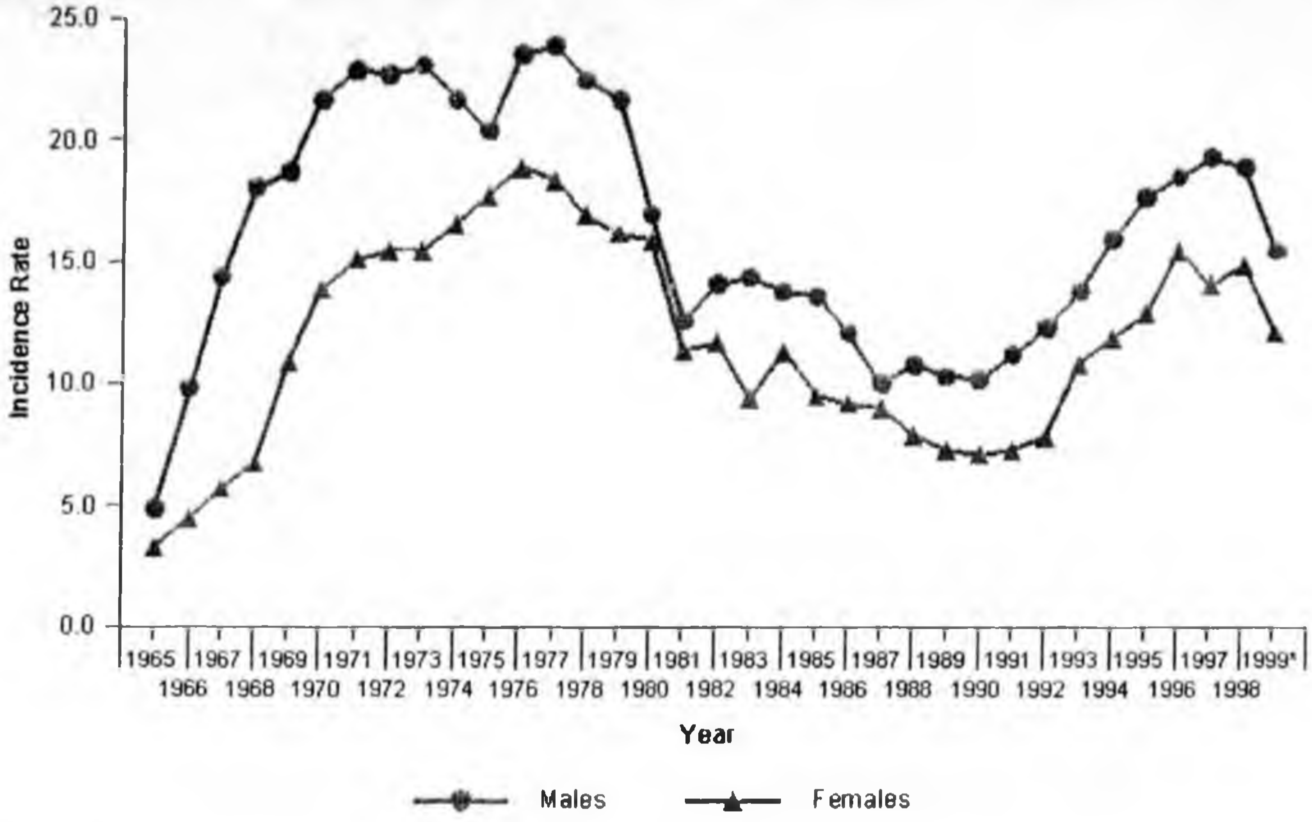
Figure 3.2 Marijuana Incidence Rates, by Gender and Year



Note: The numerator of each rate is the number of persons who first used marijuana in the year, while the denominator is the person-time exposure measured in thousands of years for persons aged 12 or older.

* Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

3.4 Trends, by Race/Ethnicity

The trends of marijuana incidence also varied across racial/ethnic groups (Tables 3.5 to 3.7). In 1999, an estimated 1.4 million new marijuana u ers were white; there were 0.25 million black initiates, 0.25 million Hispanic initiates, 0.04 million Asian initiates (including other Pacific Islanders and Native Hawaiians), 0.03 million American Indian/Alaska Native initiates, and 0.03 million initiates who reported more than one race. Except for American Indians/Alaska Natives, the estimated numbers of new users were lower in 1999 than in 1998. In recent years, American Indians/Alaska Natives appeared to have a younger mean age of first marijuana use (14.1 years in 1999) than members of other racial/ethnic groups. In 1999, the mean age of marijuana initiation was 17.2 years for whites, about 16.4 years for blacks and Hispanics, 18.8 for Asians (including other Pacific Islanders and Native Hawaiians), and 15.8 years for persons reporting more than one race.

Among whites, the trend pattern was generally consistent with the overall trend seen in Table 3.1. Probably because of small samples, more variation was noted for non-Hispanic minority groups (American Indians/Alaska Natives, Asians/Pacific Islanders/Native Hawaiians, and persons reporting more than one race) and for years before 1990. Incidence rates between 1990 and 1999 for the racial/ethnic groupings are displayed in Figure 3.3.

Among blacks, the annual incidence rate (per 1,000 potential new users) increased from 8.0 in 1966 to 16.7 in 1968, reached a peak at about the same time as whites (19.4 in 1976), then remained high throughout the late 1970s. Following the low rates in the 1980s, rates among blacks rose again in the early 1990s, reached a peak in 1997 and 1998 (19.2 and 19.1, respectively), then dropped to 14.0 in 1999. Similar to the general pattern for whites and blacks, Hispanics' annual incidence rate rose during late 1970s and 1990s, with a peak in 1998 (17.8).

Asians (including other Pacific Islanders and Native Hawaiians) typically had lower annual incidence rates than the other racial/ethnic groups. However, the sample size did not allow for the generation of reliable estimates for trend data prior to 1985. Among recent initiates, rates of first marijuana use by racial/ethnic groups were generally lower in 1999 than in 1998, with the exception of American Indians/Alaska Natives. Estimates from Table 3.7 suggest a higher rate of new marijuana use in recent years among American Indians/Alaska Natives and among persons reporting more than one race. The annual incidence rate among American Indians/Alaska Natives was 21.2 (per 1,000) in 1995 and had risen over these years to a rate of 46.5 in 1999. Similar to the rates for American Indians/Alaska Natives, incidence rates among persons reporting more than one race were higher than among other racial/ethnic groups during the 1990s. Their incidence rate ranged from 26.2 to 35.4 between 1995 and 1999 compared with a rate below 20.0 among whites, blacks, Hispanics, and Asians/Pacific Islanders/Native Hawaiians.

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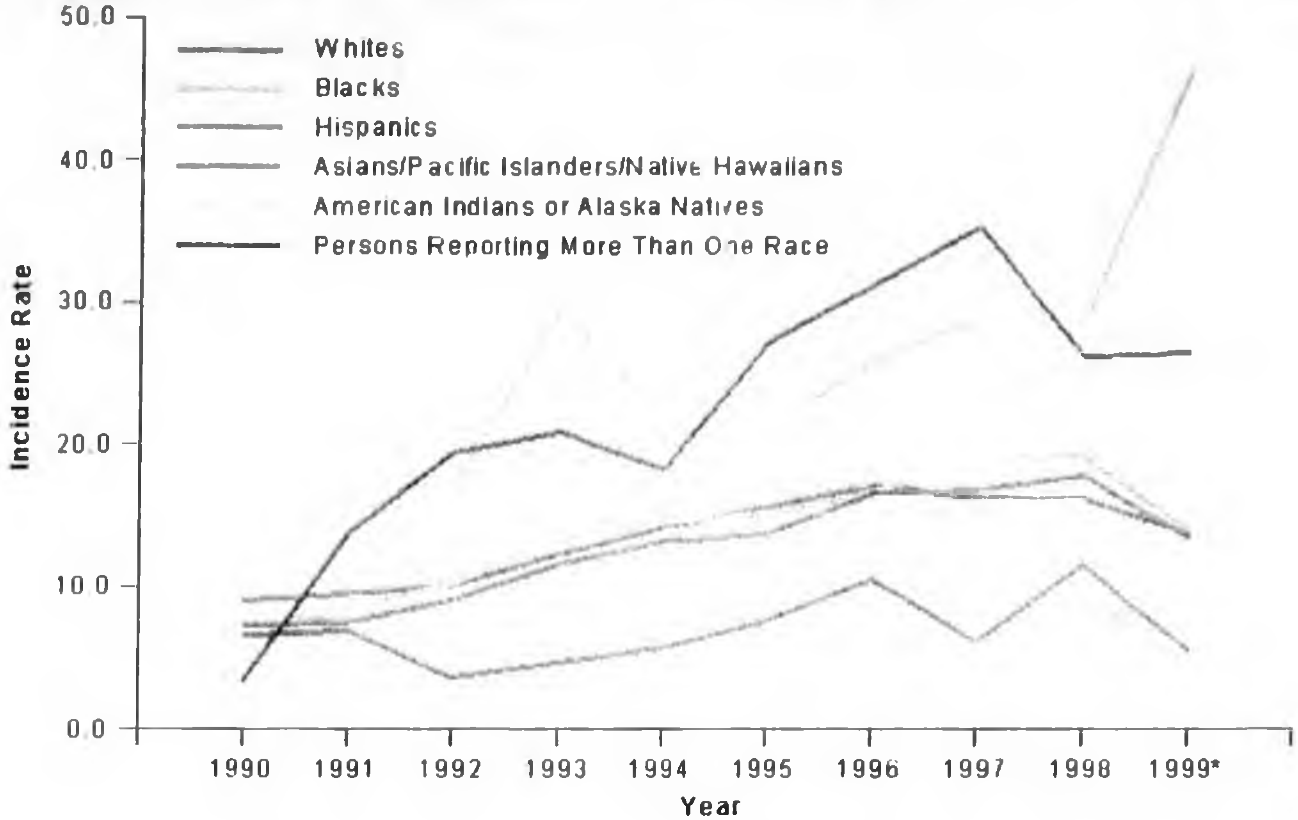
Figure 3.3 Marijuana Incidence Rates, by Race/Ethnicity and Year



Note: The numerator of each rate is the number of persons who first used marijuana in the year, while the denominator is the person-time exposure measured in thousands of years for persons aged 12 or older.

* Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

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Table 3.1 Estimated Numbers (in Thousands) of Persons Who First Used Marijuana During the Years 1965 to 1999, Their Mean Age at First Use, and the Annual Incidence Rates of First Use (Per 1,000 Person-Years of Exposure), for All Ages			
Year	Number of Initiates (1,000s)	Mean Age at First Use	Incidence Rates[1]

1965	553	20.4	4.0
1966	975	19.2	7.0
1967	1,385	19.5	9.7
1968	1,738	19.4	12.0
1969	2,123	19.0	14.5
1970	2,552	18.7	17.5
1971	2,789	18.7	18.7
1972	2,819	18.8	18.8
1973	2,854	18.6	19.0
1974	2,853	17.9	18.9
1975	2,874	18.3	19.0
1976	3,184	18.5	21.0
1977	3,163	18.3	20.9
1978	2,967	18.1	19.5
1979	2,859	18.1	18.7
1980	2,522	19.2	16.4
1981	1,867	17.9	12.0
1982	2,021	18.8	12.8
1983	1,865	18.2	11.7
1984	2,012	18.3	12.4
1985	1,865	18.1	11.4
1986	1,753	17.6	10.6
1987	1,588	17.6	9.5
1988	1,550	17.4	9.2
1989	1,447	17.7	8.7
1990	1,407	18.3	8.5
1991	1,485	18.0	9.1
1992	1,599	16.7	9.8
1993	1,954	17.2	12.2
1994	2,187	16.7	13.8
1995	2,357	16.5	15.1
1996	2,590	17.1	16.8
1997	2,494	17.0	16.5
1998	2,488	17.4	16.7
1999[2]	2,028	17.0	13.6

[1] The numerator of each rate is the number of persons who first used marijuana in the year, while the denominator is the person-time exposure measured in thousands of years for persons aged 12 or older.
[2] Estimated using 2000 data only.
Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

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Table 3.2 Estimated Age-Gender Numbers (in Thousands) of Persons Who First Used Marijuana During the Years 1965 to 1999

Year	Number of Initiates (1,000s)							
	Males 12-14	Females 12-14	Males 15-17	Females 15-17	Males 18-20	Females 18-20	Males 21+	Females 21+
1965	41	*	114	36	72	84	70	98
1966	64	*	159	86	271	129	142	102
1967	113	54	200	98	312	149	314	132
1968	98	38	248	109	552	195	293	184
1969	115	78	372	216	488	261	266	291
1970	197	187	435	328	496	279	333	303
1971	266	210	405	283	486	320	358	395
1972	264	148	496	453	414	308	385	326
1973	261	225	565	385	365	320	353	306
1974	245	271	584	468	329	301	253	307
1975	309	275	469	493	339	270	235	404
1976	213	208	665	603	414	317	303	420
1977	292	272	633	559	396	309	291	354
1978	263	221	691	542	317	341	230	296
1979	287	237	627	522	362	300	176	274
1980	184	165	486	531	215	297	249	312
1981	156	144	357	383	212	203	120	221
1982	189	132	385	391	254	215	154	258
1983	182	152	394	329	197	172	241	128
1984	237	176	382	385	209	207	160	2.5
1985	184	155	370	371	232	194	204	118
1986	155	134	361	382	212	183	159	118
1987	85	109	340	386	250	189	75	124

1988	132	80	348	327	210	164	112	136
1989	122	96	326	280	175	175	116	99
1990	130	94	309	240	197	135	103	148
1991	154	96	302	265	180	171	160	101
1992	185	159	347	258	222	173	104	82
1993	244	222	364	355	229	210	124	136
1994	276	261	450	394	242	234	123	121
1995	336	274	510	401	226	256	137	141
1996	350	294	523	523	235	268	138	202
1997	329	313	547	478	266	227	145	139
1998	334	313	519	467	236	250	154	175
1999[1]	291	255	446	399	151	175	124	159

* Low precision; no estimate reported.

[1] Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

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Table 3.3 Estimated Numbers (in Thousands) of Persons Who First Used Marijuana During the Years 1965 to 1999, Their Mean Age at First Use, and the Annual Incidence Rates of First Use (Per 1,000 Person-Years of Exposure), by Gender

Year	Number of Initiates (1,000s)		Mean Age at First Use		Incidence Rates[1]	
	Males	Females	Males	Females	Males	Females
1965	315	239	18.1	23.4	4.9	3.3
1966	642	333	18.8	19.9	9.8	4.5
1967	952	433	19.1	20.4	14.4	5.7
1968	1,212	527	19.0	20.1	18.1	6.8
1969	1,264	859	18.6	19.5	18.7	10.9
1970	1,479	1,112	18.6	19.0	21.7	13.9
1971	1,570	1,218	18.4	19.0	22.9	15.1
1972	1,560	1,258	19.2	18.3	22.7	15.5
1973	1,587	1,267	18.6	18.6	23.1	15.5

1974	1,493	1,360	17.7	18.1	21.7	16.6
1975	1,405	1,469	17.7	18.9	20.4	17.8
1976	1,625	1,559	18.2	18.8	23.6	18.9
1977	1,647	1,517	18.0	18.5	23.9	18.4
1978	1,556	1,411	17.6	18.7	22.5	17.0
1979	1,507	1,352	17.5	18.7	21.7	16.2
1980	1,187	1,335	19.0	19.4	17.0	15.9
1981	896	971	17.2	18.6	12.6	11.4
1982	1,014	1,007	17.9	19.7	14.1	11.7
1983	1,049	815	18.9	17.4	14.4	9.4
1984	1,020	992	18.3	18.2	13.8	11.3
1985	1,021	844	18.2	17.9	13.6	9.5
1986	925	828	17.8	17.4	12.1	9.2
1987	773	815	17.3	17.9	10.0	9.0
1988	834	716	17.1	17.9	10.8	7.9
1989	787	660	17.5	17.8	10.3	7.3
1990	774	633	17.5	19.4	10.2	7.1
1991	837	648	18.1	17.8	11.2	7.3
1992	909	690	16.6	16.8	12.3	7.8
1993	1,009	945	16.8	17.6	13.8	10.8
1994	1,152	1,035	16.7	16.8	16.0	11.9
1995	1,254	1,103	16.4	16.7	17.7	12.9
1996	1,284	1,306	16.4	17.7	18.5	15.5
1997	1,318	1,176	17.0	16.9	19.3	14.1
1998	1,268	1,220	17.6	17.2	18.9	14.9
1999[2]	1,034	993	16.4	17.6	15.5	12.1

[1] The numerator of each rate is the number of persons who first used marijuana in the year, while the denominator is the person-time exposure measured in thousands of years.

[2] Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

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Table 3.4 Estimated Annual Age-Gender Specific Incidence Rates of First Use (Per 1,000 Person-Years of Exposure) of Persons Who First Used Marijuana During the Years 1965 to 1999

Year	Age-Gender Specific Incidence Rates[1]							
	Males 12-14	Females 12-14	Males 15-17	Females 15-17	Males 18-20	Females 18-20	Males 21+	Females 21+
1965	7.2	*	21.5	5.9	18.2	16.0	3.0	3.4
1966	11.1	*	30.5	14.7	60.9	22.3	5.8	3.3
1967	19.7	8.8	37.7	17.2	68.0	24.0	12.4	4.1
1968	17.1	6.1	46.9	18.8	125.6	34.0	11.0	5.4
1969	19.1	11.9	71.2	37.4	121.7	49.1	9.6	8.2
1970	30.9	28.6	86.4	57.5	124.6	54.8	11.6	8.2
1971	41.3	32.3	83.0	48.9	125.5	64.1	12.2	10.3
1972	43.6	22.5	99.5	77.0	108.3	64.5	12.8	8.2
1973	43.9	33.5	110.6	68.0	98.2	68.3	11.4	7.5
1974	40.0	42.0	118.6	84.6	89.7	64.1	8.0	7.3
1975	48.3	44.4	99.4	88.5	93.2	58.2	7.2	9.4
1976	34.1	34.0	142.5	109.6	114.5	71.3	9.1	9.5
1977	49.9	44.5	131.6	108.3	117.5	72.6	8.5	7.8
1978	48.4	37.6	141.2	110.5	100.0	81.3	6.6	6.4
1979	57.0	41.6	133.2	106.3	114.3	73.7	4.9	5.8
1980	37.2	29.9	110.3	109.1	66.0	78.0	6.8	6.5
1981	30.9	26.2	82.9	79.7	60.3	54.4	3.2	4.5
1982	36.3	23.7	93.4	83.0	72.3	57.6	4.0	5.2
1983	34.5	26.9	96.2	69.6	58.4	44.9	6.2	2.5
1984	46.5	31.4	90.7	82.2	63.5	53.3	4.0	4.1
1985	38.1	28.8	85.8	79.2	73.7	50.5	5.0	2.2
1986	33.4	26.5	83.6	80.6	67.2	47.5	3.8	2.2
1987	18.5	22.0	80.8	81.6	76.0	50.0	1.8	2.2
1988	29.0	16.7	85.9	71.4	62.2	43.0	2.6	2.4
1989	26.3	19.6	82.9	64.3	51.7	45.3	2.6	1.7
1990	27.2	19.1	79.7	55.3	58.6	34.5	2.3	2.5
1991	31.5	18.6	77.3	62.2	56.5	44.3	3.5	1.7
1992	36.1	29.3	88.3	59.6	70.9	46.6	2.2	1.4
1993	45.6	39.6	90.5	82.5	75.2	57.4	2.6	2.2
1994	49.6	46.8	112.7	89.1	80.5	67.3	2.5	1.9
1995	61.2	49.9	126.8	90.3	76.9	74.6	2.8	2.2

1996	65.1	55.0	127.0	117.8	80.5	80.4	2.8	3.1
1997	60.2	59.2	130.5	110.3	94.0	67.7	2.8	2.1
1998	59.9	58.8	127.4	111.5	83.9	75.2	3.0	2.6
1999[2]	51.8	48.1	112.2	97.9	53.0	52.1	2.3	2.4

* Low precision; no estimate reported.

[1] The numerator of each rate is the number of persons who first used marijuana in the year, while the denominator is the person-time exposure measured in thousands of years.

[2] Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

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Table 3.5 Estimated Numbers (in Thousands) of Persons Who First Used Marijuana During the Years 1965 to 1999, by Racial/Ethnic Subgroups

Year	Number of Initiates (1,000s)					
	White	Black	Hispanic	Asian / Pacific Islander / Native Hawaiian	American Indian / Alaska Native	More Than One Race
1965	427	*	*	*	*	*
1966	804	113	*	*	*	*
1967	1,180	128	49	*	*	*
1968	1,417	246	62	*	*	*
1969	1,834	175	63	*	*	*
1970	2,264	180	73	38	*	21
1971	2,313	228	177	*	14	*
1972	2,413	244	111	12	22	17
1973	2,442	260	91	*	*	*
1974	2,343	256	213	12	*	19
1975	2,377	296	171	12	*	*
1976	2,615	317	172	*	31	*
1977	2,608	277	163	74	*	28
1978	2,370	297	206	77	*	12
1979	2,388	275	127	*	8	23
1980	2,067	235	168	*	*	*

1981	1,518	195	120	*	6	*
1982	1,640	164	165	*	16	7
1983	1,459	149	187	39	11	*
1984	1,633	232	98	*	8	7
1985	1,437	179	165	65	7	12
1986	1,375	186	130	25	24	13
1987	1,242	140	134	52	10	12
1988	1,222	137	132	20	24	15
1989	1,074	137	183	21	*	16
1990	1,057	144	144	49	8	5
1991	1,092	164	146	50	14	20
1992	1,154	199	178	27	13	27
1993	1,388	256	225	34	23	28
1994	1,582	273	250	41	16	24
1995	1,711	282	259	54	16	35
1996	1,848	303	307	75	19	39
1997	1,733	345	308	44	20	43
1998	1,702	336	320	80	19	31
1999[1]	1,436	248	246	39	30	28

* Low precision; no estimate reported.

[1] Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

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Table 3.6 Estimated Mean Ages at First Use of Persons Who First Used Marijuana During the Years 1965 to 1999, by Racial/Ethnic Subgroups

Year	Mean Age at First Use					
	White	Black	Hispanic	Asian / Pacific Islander / Native Hawaiian	American Indian / Alaska Native	More Than One Race
1965	21.3	*	*	*	*	*
1966	19.3	19.2	*	*	*	*
1967	19.7	18.5	22.0	*	*	*

1968	19.3	19.6	20.2	*	*	*
1969	19.0	19.0	18.6	*	*	*
1970	18.9	17.1	17.1	18.0	*	*
1971	18.8	18.4	17.9	*	*	*
1972	18.8	18.0	21.2	*	19.5	*
1973	18.7	18.4	18.0	*	*	*
1974	17.9	18.4	17.1	*	*	16.6
1975	18.5	17.3	18.1	*	*	*
1976	18.4	19.8	17.7	*	16.8	16.4
1977	18.4	18.0	18.1	17.7	16.6	*
1978	17.9	18.5	20.1	17.9	*	*
1979	18.2	17.7	16.7	*	14.5	15.2
1980	19.4	18.8	17.9	17.1	*	*
1981	18.0	18.6	15.9	*	*	*
1982	18.8	20.8	17.0	*	18.1	*
1983	18.2	18.2	18.8	18.5	16.2	14.0
1984	18.0	20.3	14.7	29.5	10.8	13.5
1985	18.2	17.6	17.4	17.1	14.8	19.1
1986	17.8	17.4	16.9	16.1	14.5	14.9
1987	17.7	16.6	17.3	19.2	19.6	18.3
1988	17.4	16.5	18.0	17.9	21.8	15.1
1989	17.9	17.2	17.0	17.3	16.5	15.5
1990	18.6	17.8	17.7	17.5	19.3	14.9
1991	18.3	17.5	16.0	17.7	22.2	16.5
1992	16.8	17.0	15.7	17.5	15.4	16.0
1993	17.1	17.9	16.7	18.1	22.7	14.4
1994	16.8	16.9	16.2	16.5	15.8	15.2
1995	16.5	16.8	16.0	18.3	15.1	14.9
1996	17.1	17.6	16.7	17.3	14.5	16.0
1997	17.0	17.7	16.0	16.6	15.8	15.9
1998	17.8	17.2	16.0	17.6	14.6	17.0
1999[1]	17.2	16.5	16.4	18.8	14.1	15.8

* Low precision; no estimate reported.

[1] Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

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Table 3.7 Estimated Annual Incidence Rates of First Use (Per 1,000 Person-Years of Exposure) of Persons Who First Used Marijuana During the Years 1965 to 1999, by Racial/Ethnic Subgroups

Year	Racial/Ethnic Specific Incidence Rates[1]					
	White	Black	Hispanic	Asian / Pacific Islander / Native Hawaiian	American Indian / Alaska Native	More Than One Race
1965	4.0	*	*	*	*	*
1966	7.5	8.0	*	*	*	*
1967	10.8	8.9	4.1	*	*	*
1968	12.8	16.7	4.9	*	*	*
1969	16.4	11.7	4.9	*	*	*
1970	20.2	11.8	5.4	7.3	*	21.3
1971	20.6	14.7	12.7	*	20.0	*
1972	21.5	15.5	7.7	2.2	31.6	17.7
1973	21.8	16.3	6.2	*	*	*
1974	21.0	15.9	14.1	2.0	*	18.8
1975	21.3	18.2	11.1	2.0	*	*
1976	23.5	19.4	10.9	*	41.5	34.0
1977	23.5	16.8	10.1	11.7	*	27.0
1978	21.4	17.8	12.5	12.2	*	11.6
1979	21.6	16.3	7.5	*	10.3	21.3
1980	18.6	13.7	9.7	*	*	*
1981	13.5	11.1	6.8	*	*	*
1982	14.5	9.1	9.2	*	19.7	5.8
1983	12.8	8.1	10.1	5.7	14.1	*
1984	14.2	12.4	5.2	*	9.3	5.5
1985	12.4	9.4	8.6	9.1	8.0	9.2
1986	11.7	9.6	6.6	3.4	28.5	9.2
1987	10.5	7.1	6.7	7.0	11.4	8.2
1988	10.3	6.9	6.5	2.7	28.8	10.3
1989	9.2	6.9	9.1	2.9	*	10.9
1990	9.1	7.3	7.3	6.6	10.2	3.4

1991	9.5	8.4	7.4	6.9	16.9	13.9
1992	10.1	10.3	9.1	3.7	17.1	19.5
1993	12.3	13.4	11.6	4.7	29.3	20.9
1994	14.2	14.5	13.1	5.7	21.8	18.3
1995	15.6	15.2	13.7	7.6	21.2	27.1
1996	17.1	16.6	16.5	10.5	25.8	31.2
1997	16.3	19.2	16.8	6.2	28.8	35.4
1998	16.3	19.1	17.8	11.6	28.6	26.2
1999[2]	13.8	14.0	13.5	5.6	46.5	26.4

* Low precision; no estimate reported.

[1] The numerator of each rate is the number of persons who first used marijuana in the year, while the denominator is the person-time exposure measured in thousands of years.

[2] Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

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The joint demand for cigarettes and marijuana: evidence from the National Household Surveys on Drug Abuse

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Abstract

Recent studies have shown that efforts to curb youths' alcohol use, such as increasing the price of alcohol or limiting youths' access, have succeeded but may have had the unintended consequence of increasing marijuana use. This possibility is troubling in light of the doubling of teen marijuana use from 1990 to 1997. What impact will recent increases in cigarette prices have on the demand for other substances, such as marijuana? To better understand how the demand for marijuana and tobacco responds to changes in the policies and prices that affect their use, we explore the National Household Survey on Drug Abuse (NHSDA) from 1990 to 1996. We find evidence that both higher fines for marijuana possession and increased probability of arrest decrease the probability that a young adult will use marijuana. We also find that higher cigarette taxes appear to decrease the intensity of marijuana use and may have a modest negative effect on the probability of use among males.

JEL classification: I1

Keywords: Marijuana; Cigarettes; Price; Complements; Elasticity; Demand

1. Introduction

Marijuana use among youths continues to rise despite community, state, and national efforts to educate and inform individuals of the harmful effects of drug use and abuse. For example, between 1990 and 1997, marijuana use among 12–17-year-old more than doubled,

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increasing from a prevalence of 4.4–9.7% (SAMHSA, 1998). The dramatic increase in marijuana use poses new challenges to decision makers developing policies to curb drug use among youths. During this same time period, current cigarette use has steadily increased among 8th, 10th, and 12th graders (MTF, 2000). Epidemiological studies indicate that licit drug use (e.g. tobacco and alcohol) may serve as a gateway to illicit drug use (Kandel, 1975; Kandel and Faust, 1975; Kandel and Yamaguchi, 1993; Duncan et al., 1998). If cigarette use is a gateway to marijuana use, recent policies directed at curbing current youth smoking may also lead to declines in marijuana use. Other studies have demonstrated interdependence between policies directed at curbing alcohol and marijuana use (DiNardo and Lemieux, 1992; Model, 1993; Chaloupka and Laixuthai, 1997; Pacula, 1998a,b).

All of these studies emphasize the importance of understanding the interdependence of widely used substances, such as alcohol, tobacco, and marijuana and suggest that policies affecting one substance may have unintended consequences on the others. Understanding these interdependencies is especially relevant in light of the US$ 0.45-per-pack cigarette price increase announced in November 1998 in response to the US$ 206 billion tobacco industry settlement with 46 states. Policy makers must understand whether proposals intended to reduce smoking among youths have unintended consequences among youths and the general population that may cause marijuana use and other substance use to rise.

The goal of this paper is to further explore the interdependencies between tobacco and marijuana using a rich database on drug use, the National Household Survey on Drug Abuse (NHSDA). The NHSDA is a nationally representative survey of the US noninstitutionalized population aged 12 and older. The detailed questions on substance use contained in the NHSDA allow us to better understand how marijuana use responds to changes in policies that affect the price and availability of marijuana and tobacco. We focus our analyses on youths aged 12–20. We chose this age group for several reasons. Recent trends show dramatic changes in marijuana use for youths aged 12–20 but not for adults aged 21–30. Also, many children first experiment with tobacco and marijuana in their teens.

The specific aim of this paper is to estimate the demand for marijuana and tobacco as a function of the policies and prices that affect their use. Often, empirical demand models of marijuana use are not a function of own prices because marijuana prices are not consistently available. The only time series data available for marijuana prices come from the Drug Enforcement Administration. Unfortunately, these data cover a limited number of cities throughout the US, and have very few price observations in each city in any 1 year, with only a few exceptions. In the absence of the price of marijuana, much of the literature (discussed below) includes policies that serve as a proxy for the price of marijuana. We follow this practice. We constructed a measure of the probability of being arrested for marijuana possession by dividing total annual marijuana possession arrests in a state by an estimate of the total number of marijuana users in the state (by age group). We also include state marijuana laws that specify fines and jail sentences for possession, rather than a simple indicator that marijuana use is decriminalized, because there has been nearly no change in marijuana decriminalization during the study period. Finally, we include the price of cigarettes to capture the possibility that tobacco is an economic complement or substitute with marijuana. We estimate comparable models for cigarette demand — both participation and demand conditional on use. Because of our concern that unobservable state-level variables may bias the coefficients on the policies and price variables, all marijuana and tobacco models include state fixed effects.

The results of this paper will help guide the creation of comprehensive policies that curb the use of marijuana in two ways. First, we quantify the effects of policies aimed at curbing the use of marijuana, allowing policy makers to evaluate alternative policy options. For example, we provide estimates of the impact of police efforts to enforce marijuana possession laws and the impact of state-level marijuana possession fines. Second, we present results that clarify the cross-price effects between tobacco demand and marijuana use. With an understanding of this interdependency, policy makers can take into account how policy changes directed at one substance affect the demand for the other.

2. Previous studies

In this section, we discuss previous studies that have examined the interdependence of marijuana and tobacco use. There is an extensive literature on the relationship between cigarette and marijuana use, focusing primarily on tobacco use as a 'gateway' to other drug use, including marijuana. Cigarette smoking is considered a gateway drug by some because youths who start with tobacco and alcohol use are more likely to progress to marijuana and other drug use (Kandel, 1975; Kandel and Faust, 1975; Ellickson et al., 1992; Kandel and Yamaguchi, 1993; Duncan et al., 1998). The seminal work by Denise Kandel in 1975 involved tracking licit and illicit drug use by 7250 high school students in New York over a 2-year period. Kandel divides drug use into six categories — legal drugs, marijuana, pills, LSD, cocaine, and heroin — and finds that drug use tends to be cumulative. In other words, youths in any one category have used all 'lower-ranked' drugs. In subsequent work, Yamaguchi and Kandel (1984) find that current alcohol and cigarette use are both strong predictors of initiation of marijuana use. In more recent work, Duncan et al. (1998) find that cigarettes are a stronger predictor of future marijuana use than alcohol and that higher levels of cigarette use are predictive of greater future marijuana use.

From a physiological perspective, two studies suggest that marijuana may be a substitute for cigarettes among current users of both substances. Simmons and Tashkin (1995) obtained detailed marijuana and cigarette smoking histories from 467 adult regular smokers of marijuana and/or cigarettes and found that those who smoke both cigarettes and marijuana smoke significantly fewer cigarettes per day than those who do not smoke marijuana. However, marijuana use is not significantly different for smokers and nonsmokers of cigarettes. Of those who smoke both, 49% began smoking cigarettes first, while 33% smoked marijuana first. Similarly, a controlled experiment of current marijuana and cigarette users illustrates the interdependence of marijuana and cigarettes among current users. Kelly et al. (1990) find that active marijuana smoking (placebo versus active marijuana cigarettes were used) significantly decreased the number of daily cigarette smoking bouts, increased inter-bout intervals, and decreased inter-puff intervals. Although these studies illustrate the potential correlation between marijuana and tobacco, they are both based on relatively small samples of subjects.

Recent studies by economists have also explored the gateway effect and other relationships among licit and illicit drug use behaviors by examining how changes in prices and policies can affect the use of the targeted substance as well as potentially related substances (DiNardo and Lemieux, 1992; Model, 1993; Thies and Register, 1993; Chaloupka and Laixuthai, 1997; Pacula, 1998a,b; Saffer and Chaloupka, 1998; Chaloupka et al., 1999). However, among these studies, only three have examined the relationship between marijuana and tobacco (Pacula, 1998a,b; Chaloupka et al., 1999). A limitation of all of these studies is that they lack data on the price of marijuana and hence must rely on proxies for the price of marijuana. The only readily available data on the price of marijuana come from Drug Enforcement Administration databases. These databases capture purchases made by undercover federal agents nationwide and police officers in Washington, DC. Because federal interdiction efforts focus primarily on cocaine, heroin, methamphetamines, and other illicit substances other than marijuana, there are relatively few price observations for marijuana in any 1 year in a given city, with the notable exception of Washington, DC. For example, between 1988 and 1997, there were an average of 44 price observations for DC, but only three observations on average in other states. For this reason, this and previous studies do not include the price of marijuana.

In the only published article on the relationship between marijuana and tobacco use, Pacula (1998a) analyzes the 1984 wave of the National Longitudinal Survey of Youth (NLSY) to estimate the joint demand for alcohol and marijuana using a sample of roughly 8000 individuals. Pacula estimates separate demand models for alcohol and marijuana demand and shows that increasing beer taxes decreases the prevalence and consumption of alcohol and decreases the prevalence of marijuana use. She also finds that respondents are more likely to use marijuana in states that have decriminalized marijuana and less likely to use marijuana in states with relatively high cigarette taxes. However, neither of these results is statistically significant.

In a continuation of this research, Pacula (1998b) analyzes two waves of the NLSY and estimates the effect of current and past cigarette prices on current marijuana use. She finds that the two substances are economic complements — higher current and past prices reduce the probability of current marijuana use.

Finally, in a recent working paper by Chaloupka et al. (1999), the authors examine the relationship between marijuana and tobacco use by 8th-, 10th-, and 12th-graders, using cross-sectional data from the 1992–1994 Monitoring the Future Surveys (MTF). They find that higher cigarette prices lead to a decrease in the probability and frequency of marijuana use. Surprisingly, the respective cross-price elasticities are −0.73 and −0.84, for a total elasticity of −1.57. This is larger than the own-price elasticities from their cigarette demand equations, which yielded a participation elasticity of −0.42 and a conditional demand elasticity of −0.71, for a total cigarette price elasticity of −1.13.

As noted, a complication of this literature is that there is no reliable price data for marijuana. Chaloupka et al. (1999) used several variables to proxy for the price of marijuana (e.g. state marijuana law fines and penalties and state decriminalization of marijuana) and found that youths are more likely to use marijuana and to use marijuana more frequently if they live in a state that has decriminalized the possession of small quantities of marijuana. Higher fines for marijuana possession were associated with reduced frequency of marijuana use but did not affect the probability of use. The authors did not include state-level fixed effects because there was not enough variation within states over the relatively short time frame of the data to identify price/policy effects.

In summary, the results of these studies provide some evidence that marijuana and tobacco are economic complements. However, it is difficult to draw strong conclusions from these studies for two reasons. First, all three studies that focus on marijuana and tobacco use rely on relatively few years of time-series cross-sectional data, which limits the variation in cigarette prices. Second, because of the limited number of cross-sections, none of these studies controlled for unobserved, state-specific characteristics. In the absence of state fixed effects, it is unclear whether the estimated effects reflect the preferences of individuals in the state or capture the effect of an exogenous policy change. For example, these studies rely on an indicator variable for states in which marijuana is decriminalized, without controlling for state fixed effects. It is not clear whether this indicator variable captures the effect of decriminalization on use or other state-specific characteristics that may be correlated with use, such as the willingness to accept alternative behaviors or lifestyles. The same may be true for cigarette prices, because they too may capture unobserved characteristics of the state. Failing to control for these unobserved characteristics may bias the estimate of the effects of all state policy and price variables.

The purpose of this paper is to examine the effects of cigarette taxes and nonprice marijuana policies on both marijuana and cigarette use. We are able to control for state effects using 7 years of pooled cross-sections and have a richer database of marijuana policies than have been used before. To proxy for the price of marijuana, we constructed an annual measure of the probability of being arrested for marijuana possession in each state. In addition, for selected years, we collected data on state fines and jail terms for violations of state marijuana laws to proxy for the price of marijuana. By including these state policies and state fixed effects, we are better able to distinguish the effects of state policies and prices from the effects of unobserved state characteristics. Finally, given the changes in cigarette tax policies across states and over time between 1990 and 1996, we are able to identify the effects of cigarette taxes on both the probability and frequency of marijuana use.

3. Data

In this study, we used data from the NHSDA. The NHSDA is a key national indicator of the nation's drug use behavior and problems. The National Institute on Drug Abuse (NIDA) sponsored the NHSDA from 1974 to 1992, and the Substance Abuse and Mental Health Services Administration (SAMHSA) has sponsored the surveys since 1992. The NHSDA is designed to provide data on the extent of drug use and abuse by the noninstitutionalized civilian population aged 12 and older in the US (SAMHSA, 1992, 1994).

We combined data from the 1990–1996 NHSDA to provide estimates of the own- and cross-price and policy effects on the demand for marijuana and tobacco. The NHSDA used identical survey questions in all 7 years for the prevalence of past-month use of marijuana and tobacco and the same five-stage area probability sample design. Sampling weights were computed based on the probability of selection at each stage, and these weights were used in all analyses.

Because of the sensitive nature of the survey topic, self-administered answer sheets were used for marijuana use questions to increase the confidentiality and anonymity of the respondents' answers. This format was designed to minimize underreporting of substance use, which is a potential limitation of self-reported surveys (Hoyt and Chaloupka, 1993). In a 1990 field test of various survey instruments, Turner et al. (1992) found that the self-administered format of the NHSDA decreases the underreporting of substance use compared to an interviewer-administered format. The NHSDA was revised in 1994 so that tobacco use questions were also reported using a self-administered questionnaire, resulting in some noncomparability of pre- and post-1994 estimates. In particular, youth tobacco use increased dramatically as a result of this change. For this reason, we include an additional indicator variable for the 1994–1996 surveys in the youth tobacco demand equations.

Although the NHSDA is the only survey that provides detailed and consistent data on drug use among members of the household population in the coterminous US, the NHSDA has a number of limitations. First, as mentioned above, the data are self-reports of drug use, so their value depends on respondents' truthfulness and memory. Although the self-administered format of the NHSDA decreases the underreporting of substance use in general, any given individual's propensity to report substance use may be affected by the degree of privacy during the interview. To examine the sensitivity of our results to the degree of privacy, we include the interviewer's report of the degree of privacy during the interview as a supplemental covariate in our demand equations. The interviewer indicates the degree of privacy on a scale from 1 (completely private) to 9 (constant presence of another). We included an indicator that represented no significant interruptions (value of 4 or less).

Second, a small proportion (roughly 1%) of the US population is excluded from the surveys. With the exception of 1991, the subpopulations excluded are those residing in non-institutional group quarters (e.g. military installations, college dormitories, group homes), those in institutional group quarters (e.g. prisons, nursing homes, treatment centers), and those with no permanent residence (e.g. the homeless and residents of single room in hotels). In 1991, the target population was extended to all 50 states and included residents of noninstitutional group quarters and civilians living on military bases. If the drug use of excluded groups differs from that of the household population, the NHSDA may provide slightly inaccurate estimates of drug use in the total population. This may be particularly true for the homeless and prison populations.

3.1. Analysis variables

The dependent variables used in our analyses include indicator variables for any past-month use of marijuana, any past-month use of cigarettes, the frequency of marijuana use in the past 30 days (1–30 days) conditional on use, and cigarettes per day conditional on use. The demographic controls used in our analyses are all self-reported measures from the NHSDA public use files. These controls consist of gender, age, number of people living in the household, number of children under age 12 in the household, marital status, race, family income, current school enrolment status, education, and size of the metropolitan statistical area (MSA) of residence. In addition to these self-reported variables, we also included two interviewer-reported variables: urban/rural status of the respondent's current residence and an indicator for the degree of privacy during the interview.

We merged state-level data on prices and policies that affect the use of marijuana and cigarettes with the NHSDA. These data include cigarette excise taxes (state + federal), marijuana possession arrest information for marijuana violations, and state fines and jail terms for marijuana possession law violations. Unlike other studies, we do not include an indicator for states that have decriminalized marijuana possession because there is essentially no variation in this variable within states over time during 1990–1996. In addition, the concept of decriminalization is subsumed in our data on state marijuana possession fines.

Marijuana possession arrest data for youths and adults come from county-level Uniform Crime Reports for 1990–1996. Using these data and marijuana use data from the NHSDA, we created two measures of the probability of being arrested. The first measure is the state marijuana possession arrests for youth divided by the number of current marijuana users aged 12–20 in that state. This measure provides an estimate of the probability of being arrested for youth. The second measure is similar to the first but is calculated for all ages 12 and older, not just youth, by dividing the total marijuana possession arrests for all ages in a state by the number of marijuana users in that state. For both age groups, state-level measures of marijuana use were estimated by taking the weighted prevalence of current marijuana use state-by-state. We then multiplied this prevalence by the population in the respective age group from the NHSDA to estimate the total number of users in a state. The purpose of the first measure is to have a youth-specific measure of the probability of arrest. However, because the denominator, youth state marijuana use, may induce some negative correlation with the dependent variable in the marijuana demand equations, we also use the second measure for all ages to provide a lower and upper bound of the results.

Because the NHSDA was not designed during this time period to support state representative data, some states have small samples (therefore, state-level estimates will not be reported). The limited sample sizes should only increase the standard errors of our estimates and not bias our results of the effect of the probability of arrest on use. [1]

We also have state marijuana law data on fines for various quantities of marijuana possession for 1990–1996 that we collected from state legal code books. The marijuana possession laws specify the minimum and maximum monetary fines and jail terms for varying amounts of marijuana possession. In other words, states usually specify fines and penalties for an ounce of marijuana and then another set of penalties for some larger quantity. To characterize these penalties and document changes in the levels of fines within states over time, it is necessary to obtain the legislative histories of the state laws. This is a research-intensive process. We were able to successfully code all states with the exception of North Carolina, North Dakota, and Tennessee for 1990–1996 and New Hampshire, New York, Rhode Island, and Vermont for 1990–1993. Observations for these states and years were dropped from the analysis.

Characterizing state marijuana laws also poses some challenges not only because the penalties for possession of a given quantity vary from state to state, but also because there is not a standard schedule of quantities that trigger increased sanctions. In essence, the marijuana penalties in each state form a step function where, for example, between any positive quantity and 1 pound, some states may have three distinct penalty levels, while

[1] In previous specifications, we used total state marijuana arrests divided by total arrests as a proxy measure for the probability of being arrested for marijuana possession. The key concept behind this measure is that police have scarce resources that they can choose to devote to drug crimes or other crimes. This is consistent with measures used by Benson and Rasmussen (Benson and Rasmussen, 1991; Benson et al., 1992; Rasmussen and Benson, 1994). Overall, the results presented in this paper are consistent with this alternative measure.

others have only one. To capture these differences, we chose to estimate models with the minimum and maximum penalties for the first and second quantity categories (e.g. typically any positive quantity up to 1 ounce and then 1–4 ounces). In addition to these measures, we include a variable that indicates the height or change in the penalty of this first step and an indicator variable for states with only one level of penalties for all quantities. To reduce the potential for multicollinearity, we chose a final specification that includes the average of the minimum and maximum penalties for each quantity.

Data on cigarette excise taxes and prices come from the Tax Burden on tobacco, an annual report from the Tobacco Institute that contains state-level information on state and federal excise taxes and average state cigarette retail prices (Tobacco Institute, 1997). Summary statistics (mean and standard deviation) of both NHSDA survey data and state-level policy and price data are listed in Table 1.

Table 1
Descriptive statistics for ages 12–20, NHSDA 1990–1996

	N = 50535	
	Mean	Standard deviation
Age	15.99	2.57
Family size	4.27	1.71
Male	51.1%	50.0%
Divorced	0.4%	6.4%
Married	2.8%	16.5%
White	69.1%	46.2%
Black	14.5%	35.2%
Hispanic	12.1%	32.7%
Other race	4.2%	20.1%
Real family income	$37446.81	$32557.38
Enrolled in school	80.2%	39.9%
High school graduate	75.6%	42.9%
Some college education	9.2%	28.9%
College graduate	0.2%	4.3%
Some graduate school education	0.03%	1.8%
MSA > 1000000	42.2%	49.4%
MSA 250000–1000000	23.6%	42.5%
MSA < 250000	9.8%	29.7%
Rural resident	14.9%	35.6%
Interview with no significant interruptions	76.2%	42.6%
Real tax on cigarettes	$0.575	$0.159
Probability of arrest — youth	2.6%	2.5%
Probability of arrest — all ages	5.2%	4.3%
Average fine — lowest level of quantity possessed	$9186.30	$62604.24
Average fine — next level of quantity possessed	$2764.75	$10388.09
Difference between minimum quantity for first and second possession levels (in grams)	597.2	4347.0
Percent living in states with only one possession level	20.5%	40.4%
Cigarette use in the past month	20.2%	40.2%
Marijuana use in the past month	8.6%	28.0%
Number of cigarettes in past month (smokers)	9.508	9.222
Frequency of past month marijuana use	8.743	9.298

4. Methods

This section describes our study methodology using pooled independent cross-sections of the 1990–1996 NHSDAs. Because we are concerned with the contemporaneous effects of prices and policies on marijuana and tobacco use, we define current users of marijuana and cigarettes as those who have had any use in the past month.

In this paper, we focus on the decision to use marijuana and the frequency of marijuana use in the past month, defined as the number of days of any use. To address the impact of cigarette taxes and policies on marijuana use, we estimate the following demand specification for marijuana:

$$\text{prob}(M > 0) = \Phi(\beta_0 + \beta_1 + \beta_2\text{Year} + \beta_3\text{State} + \beta_4 P_{\text{MM}} + \beta_5 P_{\text{MC}}) \quad (1)$$

where M is current marijuana use (past 30 days), Φ the standard normal cumulative density function, and X a vector of the sociodemographic variables described above. P_{MM} represents the effect of the marijuana 'price' variable where price is measured by the probability of being arrested for marijuana possession, and P_{MC} is the price of cigarettes (using state excise taxes). We expect the sign of β_4 to be negative so that as the probability of getting arrested increases, individuals will be less likely to consume marijuana. If $\beta_5 < 0$, then high cigarette prices lead to a lower likelihood of smoking marijuana. If this is true, then marijuana and cigarettes are economic complements. We also estimate the demand for cigarettes by estimating Eq. (2) below, using similar notation:

$$\text{prob}(C > 0) = \Phi(\delta_0 + \delta_1 X + \delta_2\text{Year} + \delta_3\text{State} + \delta_4 P_{\text{CM}} + \delta_5 P_{\text{CC}}) \quad (2)$$

From Eqs. (1) and (2), we constructed demand elasticities for participation (any use in the past month) that indicate how sensitive demand is to prices and policies.[2] P_{CM} represents the cross effect for the probability of arrest in the cigarette demand equation, and P_{CC} represents the own-price effect for cigarettes. Although it is not a price elasticity, we calculated elasticities for the probability of arrest for marijuana because having a 'unitless' measure facilitates comparisons of the results across regressions. Comparable Eqs. to (1) and (2) for both the frequency of marijuana use and cigarettes smoked per day by current users are estimated using linear regression models.

The prevalence anu intensity of marijuana and cigarette use may vary from state to state because of characteristics about the state not captured by the policy variables above. To account for this variation, we include state indicator variables or fixed effects. Analyses without state fixed effects may improperly attribute the effects of unobserved state characteristics to the policy variables. Therefore, in the absence of state fixed effects, the results inferred from state-level data on prices and policies may reflect a combination of both unobserved state characteristics and the effects of the price and policy variables. The advantage of including state effects in a pooled independent cross-sectional data set is that we con-

[2] We calculate participation elasticities as follows: the marginal effect for the jth variable is calculated as $\beta_j\phi(z)$, where $z = \Phi^{-1}(p)$ and p is the sample mean of the response variable (i.e. indicator variable for smoker), β_j the probit coefficient, Φ the standard normal probability density function, and Φ^{-1} the inverse of the standard normal cumulative density function.

trol for all unobserved state differences, including attitudes, preferences, and other state idiosyncratic characteristics that may affect demand.

Despite the advantages of including state effects, many previous studies have not included them in addition to state-level prices because of concerns over the amount of within-state variation in prices and policies over time. Without sufficient variation in the price/policy variables of interest within a state over time, it is not possible to identify both the state-specific effect and the price/policy variable. In response to this concern, researchers have often omitted state effects in favor of regional effects. While this is a reasonable approach given data limitations, results from these models should be interpreted carefully. When estimating the effects of state policies, regional effects models may simply reflect a correlation between tobacco/marijuana use and prevailing attitudes and behaviors within a state rather than the behavior changes that may result because of these policies. In this paper, we focus on the effect of cigarette excise taxes and marijuana law enforcement — policies that have experienced significant changes in the 1990s (e.g. the probability of arrest has almost tripled from 1.5 to nearly 4.5%). We chose not to examine the demand for alcohol in this paper because state beer taxes remained nearly constant in real terms over the study period. Because beer constitutes the majority of alcohol use, we determined that there was not sufficient variation in beer prices within states over time to identify own- and cross-price effects for alcohol.

To include the effects of national policies (e.g. changes in the national excise taxes on alcohol, increased national efforts to curb the supply of drugs entering the US) and other secular trends in our analyses, we included year indicator variables in the pooled independent cross-sectional data set. Although it is difficult to attribute changes in the national prevalence and intensity of marijuana and tobacco from year to year to specific policy changes, the inclusion of year indicators captures a combination of the effects of national policies and other nationwide secular trends. As noted above, we also include an indicator for the years 1994–1996 in the youth tobacco demand model to reflect the design change that occurred in 1994, causing the prevalence of tobacco use among youths to nearly double (SAMHSA, 1996).

5. Results

5.1. Base models

The results of our two-part models for marijuana and cigarettes are presented in Tables 2 and 3, respectively, with a focus on the key covariates of interest — state cigarette taxes and the probability of arrest for marijuana possession. The full models are presented in the Appendix A. State fixed effects models are presented in the top half of the tables. In both tables, specifications I and II differ only by the way in which we specify the probability of arrest variable. Specification I uses state aggregate measures of youth arrests and youth marijuana demand. Given that the denominator is aggregate youth use, this may bias the results to yield larger (more negative) effects. As a result, we also present specification II, which uses aggregate measures of both arrests and use for all ages. This specification is likely to bias the results toward zero. Hence, together the two specifications give upper and lower boundaries for the actual effect of the probability of arrest. Across both models, the

Table 2
Marijuana demand among 12–20-year-old[a]

	Specification I		Specification II	
	Participation (N = 49239)	Conditional demand (N = 3327)	Participation (N = 49499)	Conditional demand (N = 3327)
State effects				
Real tax on cigarettes	−0.008	−6.613	−0.0001	−0.0641
	(P = 0.635)	(P = 0.006)	(P = 0.38)	(P = 0.008)
	[−0.050]	[−0.436]	[−0.0979]	[−0.441]
Probability of arrest	−1.240	1.474	−0.4475	7.2838
	(P = 0.000)	(P = 0.912)	(P = 0.000)	(P = 0.304)
	[−0.3608]	[0.0044]	[−0.276]	[0.0417]
Pseudo R squared	0.113	0.1164	0.1087	0.1366
Regional effects				
Real tax on cigarettes	0.007	−2.187	−0.0001	−0.0222
	(P = 0.443)	(P = 0.076)	(P = 0.186)	(P = 0.068)
	[−0.0430]	[−0.1443]	[−0.0767]	[−0.153]
Probability of arrest	−0.921	−2.894	−0.3196	−2.5073
	(P = 0.000)	(P = 0.791)	(P = 0.000)	(P = 0.666)
	[−0.2681]	[−0.0086]	[−0.1971]	[−0.0144]
Pseudo R squared	0.1079	0.0989	0.1037	0.1084
No state or regional effects				
Real tax on cigarettes	0.011	−1.750	−0.0001	−0.0181
	(P = 0.104)	(P = 0.077)	(P = 0.129)	(P = 0.064)
	[−0.0741]	[−0.1155]	[−0.0717]	[−0.1248]
Probability of arrest	−0.890	−1.098	−0.3605	−3.8753
	(P = 0.000)	(P = 0.913)	(P = 0.000)	(P = 0.449)
	[−0.2591]	[−0.0032]	[−0.2224]	[−0.0222]
Pseudo R squared	0.1049	0.096	0.1021	0.1035

[a] Own- and cross-price/policy effects, NHSDA 1990–1996 marginal effect (P-value) [elasticity].

state fixed effect results suggest that marijuana and cigarettes are complements (the regional and no fixed effects models are discussed below). The cross-price effect for cigarettes in the marijuana participation and conditional demand models is negative, but statistically significant in only the conditional demand equation. Similarly, the probability of arrest, which can be thought of as a proxy for the price of marijuana in the absence of reliable price data, is negative and statistically significant in both parts of the two-part model for cigarettes for specification I (although small in magnitude, as one would expect).

The estimated cross-price elasticity of demand for cigarettes in specification I (II) of the marijuana equations is −0.05 (−0.10) and −0.44 (−0.44) in the participation and conditional demand models, respectively, assuming that cigarette taxes are fully passed on in the form of higher prices. The own-effect for the probability of arrest is −0.36 in specification I and somewhat smaller in absolute value in specification II, −0.28, as anticipated. In both conditional demand specifications, the probability of arrest is statistically insignificant. Therefore, a 10% increase in the probability that a marijuana user is arrested for possession decreases the probability of use by roughly 3%.

Table 3
Cigarette demand among 12–20-year-old[a]

	Specification I		Specification II	
	Participation ($N = 49253$)	Conditional demand ($N = 7678$)	Participation ($N = 49513$)	Conditional demand ($N = 7648$)
State effects				
Real tax on cigarettes	−0.032	0.832	−0.0005	0.013
	($P = 0.269$)	($P = 0.594$)	($P = 0.116$)	($P = 0.408$)
	[−0.0866]	[0.0519]	[−0.128]	[0.0768]
Probability of arrest	−0.582	−18.971	−0.4032	−1.7228
	($P = 0.000$)	($P = 0.001$)	($P = 0.000$)	($P = 0.622$)
	[−0.0712]	[−0.0530]	[−0.104]	[−0.0095]
Pseudo R squared	0.1389	0.2031	0.1391	0.2106
Regional effects				
Real tax on cigarettes	−0.036	−0.990	−0.0005	−0.0127
	($P = 0.013$)	($P = 0.205$)	($P = 0.001$)	($P = 0.101$)
	[−0.0986]	[−0.0617]	[−0.140]	[−0.0754]
Probability of arrest	−0.552	−19.298	−0.3234	−2.8578
	($P = 0.000$)	($P = 0.000$)	($P = 0.000$)	($P = 0.33$)
	[−0.0675]	[−0.0539]	[−0.0836]	[−0.0158]
Pseudo R squared	0.1361	0.1918	0.1361	0.1953
No state or regional effects				
Real tax on cigarettes	−0.051	−1.014	−0.00071	−0.0132
	($P = 0.000$)	($P = 0.105$)	($P = 0.000$)	($P = 0.035$)
	[−0.1398]	[−0.0632]	[−0.1862]	[−0.0783]
Probability of arrest	−0.532	−24.345	−0.2788	−4.4668
	($P = 0.000$)	($P = 0.000$)	($P = 0.000$)	($P = 0.098$)
	[−0.0650]	[−0.0680]	[−0.0721]	[−0.0248]
Pseudo R squared	0.1346	0.1893	0.1345	0.1906

[a] Own- and cross-price/policy effects, NHSDA 1990–1996 marginal effect (P-value) [elasticity].

With respect to cigarette demand, the own-price elasticities in both parts of the two-part model are imprecisely estimated (Table 3). Estimating cigarette demand for this age group is problematic during the 1990–1996 time period because the method to collect tobacco use among 12–17-year-old changed in 1994 from an interview format to a self-administered questionnaire (as was done for illicit drugs during the entire study period). Although we attempted to capture this effect with an indicator variable for the post-1993 period (and with price times the post-1993 indicator in another specification), we were not able to replicate standard price elasticities for youth. Based on recent estimates for this age group, one would expect total elasticities of demand from −0.4 to −0.6 (Evans and Huang, 1998; Farrelly and Bray, 1998; Tauras and Chaloupka, 1999).

The results from Table 3 yield a participation elasticity for cigarettes for specification I (II) of −0.09 (−0.13) in models with state fixed effects. The corresponding conditional demand models result in positive and statistically insignificant conditional elasticities. However, the probability of arrest in the cigarette demand equations is consistently negative, statistically significant, and small in magnitude across all state fixed effects specifications. This latter result confirms the pattern of complementarity found in Table 2.

Tables 2 and 3 also demonstrate the sensitivity of the estimates across various fixed effects models. Focusing first on the marijuana demand models, the effect of cigarette taxes on participation is statistically insignificant in all models. In specification I, the coefficient on taxes is negative in the state fixed effects models and becomes positive in the models with either regional or no fixed effects. In specification II, the result remains negative across the regional and no fixed effects specifications.

In contrast, the effect of cigarette taxes on conditional marijuana demand remains negative and statistically significant across all specifications, but the elasticity drops considerably as we move from the state to regional and no fixed effects models. This suggests that states with high levels of marijuana use have higher levels of cigarette taxation. The estimated effect for the probability of arrest remains relatively stable across all specifications.

In the cigarette demand models, the coefficient on taxes remains stable across all participation equations, while the standard error decreases from the state to regional to no fixed effects models, becoming statistically significant in the latter two models. However, in the conditional demand models, taxes remain imprecisely estimated in all specifications, with the exception of the no fixed effects model for specification II.

5.2. Gender differences

To further explore the effects of the probability of arrest and cigarette taxes on marijuana demand, we reestimate our models by gender (Table 4) using the probability of arrest defined for youth (specification I). These models reinforce the notion that marijuana and

Table 4
Demand for cigarettes and marijuana by gender[a]

	Marijuana		Cigarettes	
	Participation	Frequency	Participation	Frequency
Male				
Real tax on cigarettes	−0.040	−13.470	−0.070	2.441
	(P = 0.119)	(P = 0.000)	(P = 0.092)	(P = 0.283)
	[−0.223]	[−0.788]	[−0.185]	[0.144]
Probability of arrest	−1.367	7.799	−0.838	−14.314
	(P = 0.000)	(P = 0.681)	(P = 0.000)	(P = 0.107)
	[−0.337]	[0.020]	[−0.099]	[−0.038]
Number of observations	24319	1879	24326	4013
Adjusted R squared	0.1202	0.1048	0.1513	0.2245
Female				
Real tax on cigarettes	0.024	0.061	0.017	−1.095
	(P = 0.228)	(P = 0.984)	(P = 0.66)	(P = 0.605)
	[0.191]	[0.050]	[0.049]	[−0.073]
Probability of arrest	−1.076	−3.074	−0.381	−20.850
	(P = 0.000)	(P = 0.867)	(P = 0.005)	(P = 0.003)
	[−0.386]	[−0.011]	[−0.049]	[−0.062]
Number of observations	24900	1448	24920	3635
Adjusted R squared	0.1129	0.1349	0.139	0.1908

[a] Own- and cross-price policy effects, NHSDA 1990–1996 marginal effect (P-value) [elasticity].

cigarettes are economic complements among males. For males, the effects of cigarette taxes on marijuana demand increase considerably relative to the results for the overall sample, while the effects for the probability of arrest remain stable. These results also show that the only result that remains robust for females is the deterrent effect of the probability of arrest on marijuana participation.

Turning to cigarette demand, the tax coefficient remains imprecisely estimated in all models with the exception of the participation model for males, where taxes are statistically significant at the 10% level and yield a reasonable price elasticity of roughly −0.2.

5.3. Marijuana possession laws

When deciding whether or not and how frequently to use marijuana, youths may also consider the penalties for marijuana possession in addition to the probability of arrest. As noted above, state marijuana laws generally take the form of a step function that is at the discretion of state legislators. To quantify the effect of these penalties on use, we reestimated our marijuana demand models including the average penalties (average of the minimum and maximum fines) for the first two steps in the penalty functions of states.[3]

Each step is characterized by the quantity interval (generally starting with any positive quantity up to 1 ounce and then from 1 to 4 ounces) and the penalty for possession of these amounts. Because a few states have one set of penalties for all possession violations, we also include an indicator variable for these states. Finally, because the height of this first step — or the difference in the penalty between the first and second quantity interval — varies from state to state, we also include the amount in grams required to trigger the second level of penalties. In theory, this variable is important because it captures the effect of more or less stringent penalty scales. One would expect, ceteris paribus, requiring greater quantities of marijuana to trigger a higher level of fine might encourage more and/or more frequent use.

Table 5 shows that higher penalties for possession are correlated with a lower probability of use in the past 30 days. Both the average fine for the lowest quantity level and next highest quantity level are negative and statistically significant. However, higher fines do not appear to affect the frequency of marijuana use among young adults. The amount required to trigger a higher penalty level (the difference between the first and second amounts) had no effect on the probability of use.

Therefore, both a higher probability of arrest and higher fines decrease the probability of use, but not the frequency of use. Consistent with the results for the probability of arrest, higher penalties discourage the probability of marijuana use in the past 30 days but do not discourage the frequency of use. In addition, the effect of higher cigarette taxes on frequency of marijuana use remains robust in this alternative specification.

We also estimated the effects of these penalty variables in cigarette demand and, once again, confirmed a pattern of complementarity. However, none of the effects were statistically significant (data not shown).

[3] We also estimated models including both the minimum and maximum fines, and although the results were qualitatively similar, the individual minimum and maximum variables were not precisely estimated, possibly due to a high degree of correlation among the penalties for the first two quantity categories. As a result, we chose to present only those models with average fines.

Table 5
Effects of marijuana possession fines on marijuana use, NHSDA 1990–1996

	Participation ($N = 44659$)	Frequency ($N = 3013$)
Minimum and maximum fines		
Real cigarette tax	0.0155	−9.2915
	($P = 0.365$)	($P = 0.000$)
Average fine — lowest amount	−0.00327	0.25
	($P = 0.062$)	($P = 0.298$)
Average fine — next highest amount	−6.07E−05	−0.00429
	($P = 0.042$)	($P = 0.286$)
Quantity difference between first and second amounts	0.000448	0.597
	($P = 0.902$)	($P = 0.239$)
Indicator for one penalty level	0.0081	−1.2559
	($P = 0.541$)	($P = 0.544$)
Pseudo R squared	0.1036	0.1362

6. Conclusion

Two clear policy implications emerge from the various models that we present in this paper. First, higher cigarette taxes decrease the intensity of marijuana use and may have a modest effect on the probability of use, especially among males. Overall, the total marijuana demand cross-price elasticity for cigarettes indicates that a 10% increase in cigarette prices would lead to a 5.4% decrease in total marijuana use (with a 95% confidence interval of 0–11%). We also found that these cross-price effects were driven by the males in the sample. For males, 10% increase in cigarette prices would lead to a 10% decrease in total marijuana demand (95% confidence interval of 3–19%). Therefore, although some have suggested that increases in cigarette prices may lead to an increase in marijuana use, the evidence presented in this paper suggests that these fears may be unfounded. All of the evidence in this paper supports that there is a complementary relationship between marijuana and cigarettes and that policies that are aimed at reducing cigarette use are likely to also reduce marijuana use. Second, both higher fines for marijuana possession and increased probability of arrest decrease the probability that a young adult will use marijuana, but these policies have little effect on the frequency of use.

Finally, in policy analyses of this sort, it is critical to recognize the importance of differences in the social and political environments across states and that these differences may be correlated with both public policies and aggregate behavior (e.g. cigarette excise taxes are extremely low, while smoking rates are high). As a result, we note the sensitivity of our results to whether or not state-specific indicator variables are included in the specifications to control for these cross-sectional differences. We conclude that specifications with these state-specific indicator variables yield the most reliable estimates. This approach ensures that the estimated policy effects are driven by correlations between changes in policies and behavior over time within states, rather than spurious cross-sectional correlations.

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Appendix A

The full models are presented in Table 6

Table 6
Basic participation model with own- and cross-price/policy effects for youths[a]

	Cigarettes (N = 49253)	Marijuana (N = 49239)
Real tax on cigarettes	−0.032	−0.008
	(P = 0.269)	(P = 0.635)
Probability of arrest-youth	−0.582	−1.240
	(P = 0.000)	(P = 0.000)
Age	0.171	0.120
	(P = 0.000)	(P = 0.000)
Age squared	−0.004	−0.003
	(P = 0.000)	(P = 0.000)
Family size	−0.011	−0.006
	(P = 0.000)	(P = 0.000)
Divorced	0.088	0.044
	(P = 0.001)	(P = 0.003)
Married	−0.026	−0.037
	(P = 0.004)	(P = 0.000)
Male	0.013	0.023
	(P = 0.000)	(P = 0.000)
African American	−0.124	−0.012
	(P = 0.000)	(P = 0.000)
Hispanic	−0.064	−0.017
	(P = 0.000)	(P = 0.000)
Other race	−0.056	−0.034
	(P = 0.000)	(P = 0.000)
Real family income	0.000	0.000
	(P = 0.000)	(P = 0.000)
Student	−0.113	−0.021
	(P = 0.000)	(P = 0.000)
High school dropout	0.080	0.024
	(P = 0.000)	(P = 0.000)
Some college	−0.045	0.002
	(P = 0.000)	(P = 0.561)
College graduate	−0.036	−0.026
	(P = 0.29)	(P = 0.184)
Some graduate/professional school	−0.120	
	(P = 0.064)	

Table 6 (*Continued*)

	Cigarettes (N = 49253)	Marijuana (N = 49239)
MSA < 250000	0.011	−0.008
	(P = 0.176)	(P = 0.072)
MSA 250000–1000000	0.018	0.005
	(P = 0.012)	(P = 0.245)
MSA > 1000000	0.007	0.006
	(P = 0.316)	(P = 0.126)
Rural resident	−0.023	−0.024
	(P = 0.001)	(P = 0.000)
Interview with no significant interruptions	0.010	0.007
	(P = 0.019)	(P = 0.004)
Pseudo R squared	0.1389	0.113

[a] Also included but not shown: state and year effects, and a dummy variable for year >1994

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Table 15. Percentage of Alternative High School Students Who Used Selected Drugs by Sex, Race/Ethnicity, and Grade, 1998

Drug use behavior	Sex		Race/Ethnicity			Grade Level				All Groups
	Male	Female	White, non-Hispanic	Black, non-Hispanic	Hispanic	9th	10th	11th	12th	
Lifetime marijuana	88.0	82.1	89.4	77.7	84.0	81.0	85.3	86.0	86.8	85.4
Current marijuana[1]	58.2	46.7	56.7	47.2	50.6	51.2	52.9	55.7	51.2	53.0
Lifetime cocaine use[2]	38.6	33.0	43.8	5.7	46.4	32.7	36.4	37.8	36.5	36.1
Current cocaine use[1]	17.1	13.1	17.7	3.6	19.4	14.8	16.6	15.9	14.1	15.3
Lifetime crack or freebase use	23.5	19.4	26.2	3.5	26.8	20.9	22.9	24.2	18.9	21.6
Lifetime use of illegal steroids	9.8	7.4	10.5	6.6	6.9	12.0	9.6	6.9	7.6	8.7
Lifetime injected drug use	6.8	4.4	7.0	4.1	4.5	7.6	5.6	5.4	4.9	5.7
Episodic heavy drinking[3]	55.4	42.9	58.7	28.4	52.4	43.8	48.1	51.5	51.7	49.8
Current cigarette[1]	67.7	59.8	78.6	43.3	53.0	64.5	64.3	64.8	62.2	64.1

— Data not available.

[1]Used one or more times during the past 30 days.

[2]Ever tried any form of cocaine, including powder, crack, or freebase.

[3]Drank five or more drinks of alcohol on at least one occasion on one or more days during the past 30 days.

Source: "Youth Risk Behavior Surveillance—National Alternative High School Youth Risk Behavior Survey, United States, 1998," *Morbidity and Mortality Weekly Report* ...nters for Disease Control and Prevention, Public Health Service, Department of Health and Human Serv...es.

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Table C.5 Average Age at First Marijuana Use Among Persons Reporting First Use of Marijuana at Age 25 or Younger in 1995 to 1997, by State: 1999

State	Average Age	95% C.I.	State	Average Age	95% C.I.
National	16.2	(16.1 -16.4)	Missouri	16.2	(15.3 -17.0)
Alabama	16.6	(15.6 -17.6)	Montana	15.1	(14.5 -15.7)
Alaska	16.0	(14.8 -17.2)	Nebraska	16.1	(15.0 -17.1)
Arizona	15.3	(14.4 -16.2)	Nevada	15.1	(14.3 -15.8)
Arkansas	15.9	(15.4 -16.4)	New Hampshire	16.5	(14.7 -18.2)
California	16.1	(15.8 -16.4)	New Jersey	16.9	(16.0 -17.7)
Colorado	15.9	(15.2 -16.6)	New Mexico	15.9	(15.1 -16.7)
Connecticut	16.8	(15.1 -18.6)	New York	16.7	(16.1 -17.3)
Delaware	16.2	(15.2 -17.1)	North Carolina	16.1	(15.6 -16.7)
District of Columbia	16.4	(15.0 -17.8)	North Dakota	16.9	(16.0 -17.7)
Florida	16.1	(15.6 -16.6)	Ohio	16.6	(16.2 -17.0)
Georgia	15.8	(15.1 -16.6)	Oklahoma	15.9	(14.9 -16.8)
Hawaii	16.5	(14.7 -18.4)	Oregon	16.0	(14.8 -17.2)
Idaho	16.7	(15.8 -17.6)	Pennsylvania	16.6	(16.2 -16.9)
Illinois	15.7	(15.4 -16.0)	Rhode Island	16.3	(15.1 -17.5)
Indiana	16.3	(15.7 -17.0)	South Carolina	16.0	(15.1 -16.8)
Iowa	16.9	(16.2 -17.7)	South Dakota	16.8	(15.5 -18.1)
Kansas	15.7	(15.0 -16.5)	Tennessee	16.9	(15.3 -18.4)
Kentucky	16.5	(15.8 -17.2)	Texas	16.1	(15.8 -16.5)
Louisiana	16.0	(15.2 -16.9)	Utah	15.6	(15.0 -16.2)
Maine	17.1	(15.5 -18.7)	Vermont	16.7	(15.1 -18.2)
Maryland	16.0	(14.9 -17.1)	Virginia	16.5	(15.4 -17.6)
Massachusetts	16.2	(15.2 -17.2)	Washington	15.8	(15.3 -16.3)
Michigan	16.6	(16.0 -17.2)	West Virginia	16.3	(15.5 -17.1)
Minnesota	15.6	(15.0 -16.2)	Wisconsin	16.6	(15.4 -17.7)
Mississippi	16.8	(16.1 -17.5)	Wyoming	15.9	(15.2 -16.6)

*Low precision; no estimate reported.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 CAI.

Mental Disorders of Eskimos Seen at a Community Mental Health Center in Western Alaska

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The charts of 343 Eskimos seen at a community mental health center in northwestern Alaska were reviewed, and data on demographic characteristics, *DSM-III-R* diagnoses, and history of suicide attempts were collected. Substance use disorders were the most common group of mental disorders. Substance use patterns differed substantially according to age and gender. Both children and adults had high rates of attempted suicide (66 percent and 67 percent). Rates of bipolar disorder and eating disorders were substantially lower than those seen in mental health clinics serving the general U.S. population. (*Psychiatric Services* 49:1485–1487, 1998).

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Rapid sociocultural changes in Native American communities are reflected in the nature and extent of mental health problems in these populations (1). Several community surveys have reported high rates of psychiatric disorders. Using struc-

Table 1

Psychiatric diagnoses of adult Eskimo patients evaluated at a community mental health center in Nome, Alaska[1]

Diagnosis	All patients (N=255) N	All patients %	Men (N=115) N	Men %	Women (N=140) N	Women %
Substance abuse or dependence[2]	189	74.1	98	85.2	91	65.0
Alcohol	129	50.6	57	49.6	72	51.4
Cannabis	33	12.9	20	17.4	13	9.3
Polysubstance[3]	26	10.2	20	17.4	6	4.3
Inhalant	1	.4	1	.9	0	–
Mood disorders[4]	117	45.9	35	30.4	82	58.6
Major depression, unipolar[5]	95	37.3	27	23.5	68	48.6
Dysthymia	15	5.9	6	5.2	9	6.4
Bipolar disorder	7	2.8	2	1.8	5	3.6
Psychotic disorders[6]	47	18.4	28	24.4	19	13.6
Schizophrenia[7]	29	11.4	21	18.3	8	5.7
Schizoaffective disorder	13	5.1	5	4.4	8	5.7
Other psychotic disorders	5	2.0	2	1.7	3	2.1
Personality disorders	32	12.6	13	11.3	19	13.6
Anxiety disorders	31	12.2	9	7.8	22	15.7
Panic disorder	16	6.3	4	3.5	12	8.6
Posttraumatic stress disorder	14	5.5	4	3.5	10	7.1
Generalized anxiety disorder	1	.4	1	.9	0	–
Adjustment disorder	25	9.8	12	10.4	13	9.3
Organic disorder	15	5.9	9	7.8	6	4.3
Dementia or delirium	11	4.3	6	5.2	5	3.6
Mental retardation	4	1.6	3	2.6	1	.7
Eating disorders	4	1.6	0	–	4	2.9
Other diagnoses[8]	5	2.0	1	.9	4	2.9

[1] All statistical test reports are for comparisons between men and women.
[2] χ^2=12.9, df=1, p<.001
[3] χ^2=10.5, df=1, p<.005
[4] χ^2=19.1, df=1, p<.001
[5] χ^2=16, df=1, p<.001
[6] χ^2=4.18, df=1, p<.05
[7] χ^2=8.6, df=1, p<.005
[8] Includes somatoform disorders, pedophilia, malingering, and multiple personality disorder

tured diagnostic interviews. Kinzie and associates (2) found that 69 percent of the adult population in an American Indian village had a definite or probable mental disorder, compared with 32 percent in the Epidemiologic Catchment Area study of the general U.S. population (3). Rates of addictive diseases and depression are especially high (2,4,5). For example, lifetime prevalence rates of alcohol dependence or abuse within selected American Indian communities range from 27 to 51 percent (2,6).

Most epidemiological surveys in Native American populations have used problem checklists or questionnaires to measure psychopathology (4,5). Some have used structured diagnostic interviews to classify American Indian subjects using a modern diagnostic system (2,6), but sample sizes have been small and such studies have not been performed in Eskimo populations.

During his clinical work at the regional community mental health center in Nome, Alaska, one of the authors (RJG) noted a low rate of bipolar disorder (manic-depressive illness) and eating disorders among Eskimo patients attending the clinic. This observation, along with the lack of studies measuring rates among Eskimos of psychopathology according to our current diagnostic classification system, prompted the study described in this paper. We report the rates of psychiatric disorders among Eskimos attending a community mental health center in Western Alaska and offer possible explanations for these findings.

Methods

The Eskimos in the Bering Straits region of Alaska are members of four distinct ethnic groups: Inuit, Inupiat, Yupik, and Siberian Yupik. Although the people retain much of their traditional values and life style, almost all are literate in English.

Any person in the region is eligible to receive services at the community mental health center (CMHC) in Nome. Many patients are referred by their local medical provider.

The study population includes all Eskimo patients evaluated by the staff psychiatrist (RG) at the CMHC between October 1990 and April 1993. Charts were reviewed retrospectively, and data on demographic characteristics, *DSM-III-R* diagnoses, and history of suicide attempts were collected. Between-group differences were analyzed using two-tailed chi square tests with Yates correction.

Results

Over the two years of the study, 343 Eskimo patients– 88 children and adolescents between the ages of six and 17 years and 255 adults age 18 and older– were evaluated. Among the children and adolescents there was a slight preponderance of females (N=49). The mean age of the children and adolescents was 14±2.3 years. The mean age of the adult patients was 37±15 years.

Most youths had problems with substance use disorders (N=49), but females most frequently used alcohol, and males surprisingly were more likely to have problems with inhalants. Eating disorders were infrequent (N=3).

Table 1 outlines the distribution of diagnoses among the adult patients. Substance use disorders were prevalent, especially among men. Whereas inhalant use was much less frequent among adults compared with children, alcohol and cannabis use were much more frequent.

Most surprising was the low frequency of bipolar disorder (manic-depressive illness) and eating disorders. One would expect that approximately equal numbers of patients would have bipolar disorder as would have schizophrenia in a mental health clinic (7), but less than 3 percent of these Eskimo patients received a diagnosis of bipolar disorder.

Sixty-six percent of the children and 67 percent of the adults reported a previous suicide attempt. An interesting finding was that the two age groups differed in the gender distribution of the suicide attempters (χ^2= 11.6, df=1, p<.001), with females reporting higher rates among youths and adult men and women reporting approximately equal rates. This difference suggests that suicide may have different etiologies in different age groups.

Discussion and conclusions

Perhaps our most striking finding is the high prevalence of substance use disorders in both children and adults attending the mental health clinic. The substances of choice appeared to be alcohol, cannabis, and inhalants, and preference varied by age and by gender. Although striking, the finding was not unexpected, as high rates of substance use have been noted in surveys fairly consistently across Native American ethnic groups (8). Reasons for these consistent findings are unknown, but may include biological, psychological, and sociocultural factors (2,4).

Of concern was a 67 percent rate of reported suicide attempts. This rate compares with rates of 4 to 25 percent reported in other mental health clinics (7). Suicide rates vary widely among Native American tribes and subgroups (8). Acculturation and high rates of alcoholism have been the most common explanations given for Native American suicide (9), but a more recent study in this Eskimo population suggested that early parental loss and limited grieving mechanisms are important factors (10).

This study supported our prediction of low rates of eating disorders and bipolar disorder. In the general U.S. population, an outpatient mental health clinic might expect that approximately 8 percent of its adult patients would have bipolar disorder (7). In contrast, we found a rate of 3 percent in our clinic. To our knowledge, this is the first time that a low rate of bipolar disorder has been reported in a Native American population.

A low rate of eating disorders is easily explained by culturally based differences in attitudes towards body weight and appearance. It is more difficult to explain the low rate of bipolar disorder. One possibility is sampling bias– that is, the expected number of persons with bipolar disorder lived in the community, but they did not come to our clinic. We believe this explanation is unlikely given the disabling nature of the illness and the lack of alternative mental health resources in the region.

Another possible explanation is that our subgroup, or Native Americans as a whole, have low genetic loading for bipolar disorder or a different phenotypic expression of the illness. For example, high rates of cannabis use may have led to a psychotic diathesis of a bipolar predisposition, thereby causing a predominantly psychotic manifestation of bipolar illness. In support of this explanation, we found that cannabis use was highly correlated with assignment of a psychotic disorder (χ^2=23.6, df=1, p<.001). In addition, our clinical observation was that many of the patients that were diagnosed with schizophrenia appeared to respond to mood stabilizers.

The limitations of the study include its use of retrospective analysis and assignment of diagnoses based on the clinical evaluations of a single psychiatrist. This study highlights the need for similar studies in other Native American clinic populations, as well as systematic community surveys employing structured diagnostic interviews and a modern diagnostic classification system. ♦

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Marijuana impairs growth in mid-gestation fetuses

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Abstract

Marijuana (*Cannabis sativa*) is the most commonly used illicit drug by pregnant women, but information is limited about the effects of prenatal cannabis exposure on fetal development. The present study evaluated the influence of early maternal marijuana use on fetal growth. Women electing voluntary saline-induced abortions were recruited at a mid-gestational stage of pregnancy (weeks 17–22), and detailed drug use and medical histories were obtained. Toxicological assays (maternal urine and fetal meconium) were used in conjunction with the maternal report to assign groups. Subjects with documented cocaine and opiate use were excluded. Main developmental outcome variables were fetal weight, foot length, body length, and head circumference; ponderal index was also examined. Analyses were adjusted for maternal alcohol and cigarette use. Marijuana (n=44)- and nonmarijuana (n=95)-exposed fetuses had similar rates of growth with increased age. However, there was a 0.08-cm (95% CI −0.15 to −0.01) and 14.53-g (95% CI −28.21 to 0.86) significant reduction of foot length and body weight, respectively, for marijuana-exposed fetuses. Moreover, fetal foot length development was negatively correlated with the amount and frequency of marijuana use reported by the mothers. These findings provide evidence of a negative impact of prenatal marijuana exposure on the mid-gestational fetal growth even when adjusting for maternal use of other substances well known to impair fetal development.

Keywords: Fetal growth; Cannabis; Alcohol; Intrauterine growth; Nicotine

1. Introduction

Cannabis is the most commonly used illegal drug among young women of childbearing age in most western societies. For example, 13–23% of females aged 16–24 report marijuana use in the UK [4]. Approximately 4% of women in the USA report using illicit drugs (i.e., marijuana, cocaine, heroin, hallucinogens, inhalants, nonmedical psychotherapeutic) during pregnancy, with marijuana being the most commonly (75%) used drug among pregnant women [43]. Significant advances have been made in recent years as to the biology of the cannabis system, including identification of the cannabis receptors, their distribution in the brain and periphery, pharmacology, and signaling pathways [28,40]. Despite the growing interest in the biological actions of cannabis, there are still only few studies regarding the consequences of cannabis exposure, especially in relation to the development of the human fetus. Such information is essential when considering the current debates as to the potential legalization of marijuana in many western societies and the establishment of national policies related to marijuana's effects on health.

Prenatal effects of maternal drug use on fetal development are primarily assessed by measures of fetal growth.

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There is evidence in the literature that marijuana-exposed infants at birth have reduced weight [15,21,25,50] and/or head circumference [15,19], as well as decreased gestation length [6,20,21,25]. These findings are not, however, unequivocal since some studies have failed to document significant effects of in utero cannabis exposure on fetal development [17,39] or gestation length [32,48]. There has also been a report of increased birthweight in association with prenatal marijuana use, primarily associated with marijuana use during the third trimester [8]. Multiple reasons could account for the discrepant findings, such as the stringency of the control group, the accuracy of drug use, genetic and social differences of the populations investigated, and the developmental time period when growth parameters are evaluated. Another important consideration for the conflicting results in the literature may relate to confounding effects of maternal use of other drugs, such as cigarette and alcohol. Animal studies in which experimental conditions can be well controlled have documented reduced weight in association with prenatal cannabis exposure [1,3], thus substantiating the findings in humans of a negative impact of maternal marijuana use on fetal growth. The potential long-term prenatal effects of marijuana exposure have also been examined in longitudinal studies. Such investigations have documented specific abnormalities in cognitive (e.g., verbal skills, memory) and behavioral (impulsivity, inattention, social disturbances) functioning in offspring of women who used marijuana during pregnancy [14,18,22,41].

Despite increasing evidence of developmental impairment, as well as behavioral disturbances with in utero marijuana exposure, there is still a virtual lack of information as to the influence of early prenatal marijuana exposure on human fetal growth. Only few studies to date have investigated the prenatal effects of marijuana, and those have mainly evaluated newborns, which make it difficult to dissociate the influence of early versus late in utero drug exposure. Since women are more likely to discontinue their drug use during the early stages of pregnancy, evaluation of an early to mid-developmental time period is of particular importance. To assess developmental events prior to the third trimester, the current project examined standard measures of development in aborted fetuses. The study focused on the early to mid-gestation (weeks 17–22) developmental period since it is the latest stage during which normal specimens can be obtained from voluntary abortions. Thus, in comparison to most investigations that have examined the cumulative impact of cannabis exposure throughout the entire prenatal developmental period, the present study sought to identify the influence of early to mid-gestational marijuana exposure on fetal growth. We hypothesized that indices of fetal growth, particularly body weight, would be reduced in association with maternal marijuana use during early to mid-gestation pregnancy.

2. Methods

2.1. Sample collection

Pregnant women who had elected to carry out a voluntary saline-induced abortion at Kings County Hospital, Brooklyn, NY, between January 2000 and December 2002 were the focus group of the project. The study was approved by the Institutional Review Boards of Kings County Hospital Center and SUNY Downstate. Women were recruited for the study if they were estimated (based on ultrasound and/or maternal physical exam) to be at a mid-gestational stage (weeks 17–22) of pregnancy. The women provided written consent to participate in the study that entailed completion of a verbal questionnaire (requiring about 45 min to obtain demographic and drug use information) and urine analysis. Participants could withdraw at any time from the study. The questionnaires, maternal urines, and fetal samples were given coded identifiers, and no patient identifiers, such as the person's name, remained on any of the data sheets or drug screening results. Fetuses were included in the study if the postmortem interval did not exceed 24 h. The fetus was examined by the pathologist and/or study coordinator for routine physical measures, such as gender, birth weight (g), body length (supine length on a horizontal, flat surface; cm), foot length (cm), and occipital–frontal head circumference (cm). Meconium was also collected for subsequent toxicological analysis. An estimate of symmetrical fetal development was determined by ponderal index ([weight (g)/length $(cm)^3 \times 100$]).

2.2. Maternal demographic, drug use, and medical history assessment

Mothers were interviewed by a study coordinator using a standardized interview format established by Drs. Nancy Day and Gale Richardson (Department of Psychiatry and Epidemiology, University of Pittsburg, PA) [41]. The interview included information pertaining to maternal demographic status (e.g., race, age, educational level), maternal substance use (illicit and prescription drugs), and medical history. The substance use questions were structured to assess drug use patterns prior to pregnancy and during pregnancy. In addition to the amount and pattern of marijuana use, the interview included questions related to the use of cigarettes, beer, wine, liquor, cocaine, crack, amphetamines, heroin and other opiates, hallucinogens, and inhalants. The questions included information regarding the number of joints smoked per day (blunts were calculated to be equaled to three joints), number of cigarettes smoked per day, and number of standard drinks consumed per day. Standardization of the number of drinks for each type of alcohol (i.e., beer 12 oz, wine 5 oz, and liquor 1.5 oz) and calculation of the total amount of drug usage for each substance during prepregnancy (the year prior to pregnancy) and the pregnancy were calculated according to established published protocols (see below).

2.3. Toxicological assessment

Maternal urine screening for illicit drug use was performed by EMIT® (Dade-Behring, Irvine, CA) with a cut-off of 50, 300, and 300 ng/ml for cannabinoid, cocaine, and opiate, respectively. Meconium toxicology was carried out at the Karolinska hospital to screen (immunoassay) for cannabis, cocaine, opiates, and amphetamine using CEDIA urine screening reagents (Microgenics, CA, USA; Hitachi 917; Roche Diagnostics, IN, USA). Immunoassay response greater than the limit of detection (approximate limits: cannabis 10 ng/g, opiate 75 ng/g, cocaine 15 ng/g, amphetamines 50 ng/g, benzodiazepines 40 ng/g, phencyclidine 1 ng/g) was taken as a positive indication, and a second analysis was carried out using gas chromatography–mass spectroscopy (SIM-GC-MS).

Subjects were included in the cannabis-exposed group based on positive toxicology (urine and/or meconium) and/or positive maternal self-report. The remaining subjects were assigned to the non-cannabis-exposed group.

2.4. Data analysis

The presence and extent of marijuana use was assessed using two approaches: a dichotomized variable, positive maternal report and/or positive toxicology, and as continuous variables, number of joints normally used and frequency (times/week) of maternal use. Maternal marijuana use was expressed as average daily joint (ADJ) to dissociate the contribution of the pattern of marijuana exposure on fetal development; only subjects who provided detailed drug use history were included in such analyses. Weekly drug use was converted to daily use based on the yearly pattern of use; for example, seven joints per week was converted as 7 joints/week × 4 weeks/month)/(31 days/month) = 0.89 joint/day. Marijuana users were divided into four groups: no use, light users (0>ADJ<0.4), moderate users (0.4≥ADJ<0.89), and heavy users (ADJ≥0.89) according to the characterization by Goldschmidt et al. and Richardson et al. [22,41]. An ADJ of 0.4 is approximately three joints/week. The influence of other drugs was also assessed as to maternal use (yes/no) and the amount of use. For alcohol use, 1 drink = 12 oz. can beer, 4 oz. glass wine, 1.5 oz. shot of liquor, and average daily volume (ADV, drink/day) of 1 drink/day = 0.5 oz. absolute alcohol [41].

Four fetal outcome measures (body weight, body length, foot length, and head circumference) were assessed. Outliers, as assessed by residual plots, Cooks Distance Influence, and normality plots, were excluded from analysis to minimize the influence of a few cases on the results. An examination of the normal residual distribution was carried out for each dependent variable; no transformation of the data was carried out since the residuals were normally distributed. Univariate nonparametric analyses were carried out to obtain an overall estimate of the general characteristics of the groups. Univariate analyses do not consider the influence of other variables, thus, such analyses were not used when assessing specific significant effects of prenatal drug exposure. General linear models were conducted with confounding variables (e.g., gestation age, fetal gender, maternal age, maternal education, and married status) for the initial model testing. Cannabis exposure was always included in the model since this was the primary question of interest. Backward selection regression was carried out sequentially deleting the variables that had the weakest effect on the equation. Except for maternal alcohol and cigarette use, only variables with a $p<0.10$ correlation with the dependent variable were included in the final statistical analysis. Linear modeling was also performed to assess the amount and frequency of marijuana use on the developmental outcome variables. Data related to women who reported no marijuana use but whose toxicology revealed positive cannabis exposure were excluded from all analyses that evaluated the pattern and frequency of drug use. Statistical analyses were carried out using JMP (5.1) software package (SAS Institute, Cary, NC, USA). Statistical significance was set as $p<0.05$ and trends considered for $p<0.10$.

3. Results

3.1. Maternal characteristics of the marijuana-exposed and nonexposed groups

There was an approximately 30% refusal rate for participation in the study by pregnant women who fulfilled the project criteria. A total of 139 subjects gave informed consent, completed the questionnaire, and were included in the study (demographics in Table 1). In this cohort, 31.7% had evidence, from either toxicology and/or maternal report, of marijuana use during pregnancy. Of the women in the cannabis-use group who admitted marijuana intake, 81.8% reported using marijuana at a rate of 1.39±0.4 ADJ before pregnancy, a pattern that decreased to 0.85±0.2 during pregnancy. There were no significant demographic differences between the groups in regard to race (approximately 80% Black), marital status (predominantly unmarried), or maternal age (Table 1). To minimize the potential bias of fetal growth deficits associated with young mothers, fetuses from 15-year-old and younger (four cases) subjects were excluded from analyses regarding drug effects on fetal growth. The noncannabis group was slightly more educated (11.9 vs. 11.3 years; $p=0.0264$). The groups also differed in regard to the use of cigarettes and alcohol. Women in the cannabis-use group had an approximately fourfold higher rate of cigarette smoking and twofold higher amount of alcohol intake during

Table 1
Maternal characteristics of marijuana users (documented marijuana use before and/or during pregnancy) and nonusers as determined by positive toxicology and/or positive maternal self-report

Variables	Nonusers	Users	p-value
N	95	44	
Maternal age (years)	23.4±0.7	22.4±0.6	0.8380
Maternal education (years)	11.9±0.2	11.3±0.2	0.0192
Race/ethnicity			0.0753
White	5	0	
Black	80 (84.2%)	35 (79.5%)	
Hispanic	9	9	
Married status	7 (7.4%)	4 (9.1%)	0.4295
Cigarette use before pregnancy	28 (29%)	31 (70.5%)	
Average amount/day	2.1±0.4	5.9±0.9	<0.0001
Cigarette use during pregnancy	17 (18%)	17 (39%)	
Average amount/day	1.2±0.3	4.9±0.[illegible]	<0.0001
Nonsmoker	83	1[illegible]	
*≤5 cigarettes/day	11	1	
5 to 10 cigarettes/day	5	10	
≥10 cigarettes/day	2	6	
Alcohol use before pregnancy	28 (29%)	27 (70%)	
Average daily volume (drink/day)	0.30±0.1	0.45±0.1	0.0005
Alcohol use during pregnancy	22 (23%)	24 (54%)	
Average daily volume (drink/day)	0.22±0.09	0.42±0.1	<0.0001
None	73	20	
Light	13	11	
Moderate	4	8	
Heavy	5	5	
Use of other illicit drugs* during pregnancy	6	0	

The values represent mean±SE; percentage of some variables in parentheses.
p-Values for nominal and ordinal variables are based on Chi-squared tests, and continuous variables are based on nonparametric univariate analyses. ADV—average drinks/day; light—ADV ≤0.4 drink/day; moderate—0.4>ADV<1 drink/day; heavy—ADV>1 drink/day. *Cocaine and opiate.

pregnancy (Table 1). Both groups reduced their cigarette use during pregnancy with a greater decrease reported by the noncannabis group (44% vs. 17%). The use of alcohol decreased for 27% during pregnancy in the noncannabis group but only 5% in the cannabis users. Overall, few women reported moderate-to-heavy cigarette smoking (5.8% of the cannabis group) or heavy alcohol use (7.2% of the cannabis group) as characterized as >10 cigarettes/day and ≥1 drink/day, respectively. Many women (45.3% in the control group; 50% in the cannabis group) reported morning sickness or heartburn during the first trimester of pregnancy that purportedly affected their eating pattern and lead to weight loss. There was no statistical difference (p=0.550) between the cannabis and control groups in relation to the number of subjects reporting weight loss during pregnancy as a consequence of morning sickness or heartburn.

3.2. Toxicological evidence

Toxicological evidence was not found to match the maternal self-report in all cases. Of the cannabis-exposed group, eight (18.2%) had a positive toxicology although the mothers failed to report marijuana use during pregnancy (Table 2). In addition, 10 subjects (23% of the group) had negative toxicological evidence for cannabis exposure, but the mothers reported marijuana use during pregnancy (predominantly light; 0.37±0.15 ADJ). Women who were identified as heavy users were more likely to have toxicological evidence of recent marijuana use (50%) as compared to women who were identified as light users (16.7%). Women who reported heavy marijuana use also smoked approximately twice as many cigarettes both before and during pregnancy than women reporting light marijuana use. Heavy marijuana users reported having approximately double alcohol consumption before pregnancy but reported similar alcohol intake during pregnancy as the light marijuana users. None of the women in the marijuana-use cohort had toxicological or self-report evidence of either cocaine or opiate use. There were six cocaine/opiate cases (two subjects using both drugs) users in the noncannabis group. Due to the sample size of the cocaine and heroin subjects, the influence of these drugs could not be evaluated in the statistical analyses, and these subjects were excluded from all analyses regarding marijuana effects on fetal growth.

3.3. Fetal characteristics

The overall characteristics of the fetuses in the cannabis-exposed and nonexposed groups are presented in Table 3. The average gestational age at the time of the voluntary abortion was similar between the nonexposed (19.8±0.2 weeks) and cannabis-exposed (20.0±0.3 weeks) groups. There was also no significant difference in the gender of the fetuses, 53.6% and 59.1% males in the nonexposed and cannabis-exposed groups, respectively. There was a very strong correlation (Spearman ρ=0.898 to 0.958) between the primary growth outcome measurements. Head circumference was the least correlated with the other developmental

Table 2
Prevalence of cannabis use reported for the "cannabis users" group

Cannabis users	Before pregnancy	During pregnancy
Frequency of cannabis use (time/day)	0.43±0.1	0.35±0.1
No. reporting nonuse	8	8*
No. reporting light use	17	13
No. reporting moderate use	4	7
No. reporting heavy use	15	10
ADJ	1.39±0.4	0.85±0.2

* Negative maternal report but positive cannabis toxicology. ADJ—average number of joints/day: light use—0>ADJ<0.4; moderate use—0.4≥ADJ<0.89; heavy use—ADJ≥0.89.

Table 3
General overall comparison of the fetuses in the cannabis and noncannabis groups

Variables	Noncannabis	Cannabis
Gestation age (weeks)	19.8±0.2	20.0±0.3
Fetal gender (male/female)	51/44	26/18
Fetal weight (grams)	274.5±11.7	272.3±18.7
Fetal foot length (cm)	3.07±0.1	3.0±0.1
Fetal body length (cm)	23.1±0.4	23.03±0.5
Fetal head circumference (cm)	16.14±0.2	15.78±0.5
Ponderal index	2.14±0.04	2.09±0.05

The values are expressed as mea[illegible]

measures, but the correlation was nevertheless strong (Spearman ρ approximately 0.90). There was, however, a very weak correlation between the fetal growth measures and the secondary outcome variable, ponderal index. The correlations with ponderal index ranged from 0.0382 (non-significant) for body length to 0.2990 (p=0.008) for head circumference. Statistical analyses revealed that gestation age had the greatest impact on all outcome measures of fetal growth (approximately 59–73% prediction); thus, this variable was always included in the general linear models. All developmental measures increased with increasing gestational age though lower growth was evident in the cannabis-exposed group (Table 4); body weight: cannabis exposed, R^2=0.756; nonexposed, R^2=0.747; body length: cannabis exposed, R^2=0.693; nonexposed, R^2=0.729; foot length: cannabis exposed, R^2=0.694; nonexposed, R^2=0.720; head circumference: cannabis exposed, R^2=0.610; nonexposed, R^2=0.722. The estimated growth pattern over the developmental period examined is consistent with published [2,11,47] fetal growth rates of approximately 64 g, 0.32 cm, 1.79 cm, 1.31 cm increase of fetal weight, foot length, body length, and head circumference, respectively, for the 17–22 weeks age range. There was only a weak association between ponderal index and gestational age: cannabis exposed, R^2=0.233; nonexposed, R^2=0.0259.

Other than gestation age and drug exposure (see below), no other variable obtained from the maternal report, such as initial weight loss as a consequence of morning sickness or heartburn, illness, injury, or radiation (X-ray) during pregnancy or family history of medical problems (e.g., mental retardation or birth defects), was associated with the measurements of fetal growth.

3.3.1. Cannabis exposure on fetal growth

Cigarette smoking and alcohol intake were always included as confounding variables in the final statistical models due to the greater abundance of these substances in women in the cannabis-use group and the known influence of these substances on fetal development [9,26].

Cannabis-exposed fetuses were lighter in weight with an estimated 14.53 g difference as compared to the non-cannabis fetuses (p=0.037; Table 4). Of the other substances, only maternal alcohol use contributed significantly to fetal weight growth (estimated 13 g reduction; Table 4). Maternal marijuana use before pregnancy was also examined in an attempt to ascertain the potential cumulative effects of very early drug exposure on fetal development. Only a trend for reduced fetal weight was observed when the cumulative pre- and pregnancy marijuana history was evaluated for cannabis (p=0.093) and alcohol (p=0.056) exposure (Table 5). Examination of the pattern of marijuana intake during pregnancy revealed a trend (p=0.085) for increased amounts of maternal marijuana use during pregnancy to be associated with reduced fetal body weight. No significance was observed in regard to the frequency of marijuana use (Table 6).

Foot length growth was also significantly influenced (0.08 cm reduction) by maternal marijuana use, as well as by alcohol and cigarette exposure during pregnancy (Table 4). The cumulative pre- and pregnancy exposure period was similarly associated with a significant negative impact of

Table 4
Estimates and 95% confidence intervals (CIs) of the effect of maternal cannabis use (positive toxicology and/or maternal report) during pregnancy on parameters of fetal development adjusting for maternal cigarette and alcohol use (yes/no)

	R^2	Estimate	CI lower	CI upper	p-value
Weight					
	0.764				
Gestation age		64.33	57.53	71.13	<0.0001
M. cannabis use		−14.53	−28.21	0.86	0.0374
M. cigarette use		−7.94	−21.34	5.45	0.2425
M. alcohol use		−13.33	−24.97	−1.69	0.0252
Foot length					
	0.761				
Gestation age		0.32	0.29	0.36	<0.0001
M. cannabis use		−0.08	−0.15	−0.01	0.0227
M. cigarette use		−0.09	0.02	0.16	0.0168
M. alcohol use		−0.07	−0.13	0.01	0.0214
Body length					
	0.671				
Gestation age		1.79	1.56	2.02	<0.0001
M. cannabis use		−0.05	−0.41	0.52	0.8245
M. cigarette use		−0.08	−0.53	0.37	0.7266
M. alcohol use		−0.44	−0.84	0.04	0.0299
Head circumference					
	0.722				
Gestation age		1.31	1.15	1.47	<0.0001
M. cannabis use		−0.07	−0.42	0.28	0.6927
M. cigarette use		−0.03	−0.36	0.30	0.8664
M. alcohol use		−0.22	−0.49	0.05	0.1072
Ponderal index					
	0.079				
Gestation age		0.04	0.01	0.06	<0.0001
M. cannabis use		−0.03	−0.09	0.02	0.2501
M. cigarette use		−0.03	−0.03	0.08	0.3114
M. alcohol use		−0.00	−0.05	0.05	0.9539

Reference group=nonusers.
N=85 for the noncannabis group; N=38 for the cannabis group.
M.=Maternal.

Table 5

Estimates and 95% confidence intervals (CIs) of the effect of combined maternal marijuana use (yes/no) reported before and during pregnancy on parameters of fetal development

	R^2	Estimate	CI lower	CI upper	p-value
Weight					
	0.757				
Gestation age		64.74	57.83	71.64	<0.0001
M. cannabis use		−10.41	−22.59	1.77	0.0931
M. cigarette use		−1.54	−13.46	10.38	0.7982
M. alcohol use		−10.65	−21.58	0.29	0.0562
Foot length					
	0.736				
Gestation age		0.33	0.29	0.36	<0.0001
M. cannabis use		−0.08	−0.14	−0.01	0.0181
M. cigarette use		−0.02	−0.04	0.08	0.5061
M. alcohol use		−0.07	−0.13	0.01	0.0204
Body length					
	0.696				
Gestation age		1.82	1.59	2.04	<0.0001
M. cannabis use		−0.22	−0.61	0.18	0.2773
M. cigarette use		−0.11	−0.49	0.28	0.5868
M. alcohol use		−0.29	−0.65	0.06	0.1053
Head circumference					
	0.682				
Gestation age		1.20	1.04	1.35	<0.0001
M. cannabis use		−0.16	−0.45	0.14	0.2973
M. cigarette use		−0.00	−0.29	0.30	0.9661
M. alcohol use		−0.31	−0.58	0.04	0.0251
Ponderal index					
	0.076				
Gestation age		0.04	0.014	0.07	<0.0032
M. cannabis use		−0.03	−0.07	0.02	0.3092
M. cigarette use		−0.00	−0.05	0.05	0.9025
M. alcohol use		−0.01	−0.05	0.04	0.8107

Reference group=nonusers.

N=84 for the noncannabis group; N=44 for the cannabis group. M.=Maternal.

maternal marijuana use on foot length (0.08 cm; p=0.018; Table 5) and alcohol use (0.07 cm; p=0.020). There was a significant negative association with the amount and frequency of maternal marijuana use during pregnancy and the fetal foot length measures (Table 6). Fetal subjects exposed to moderate maternal marijuana use/day (0.40≥ADJ<0.89) during pregnancy had significantly (p=0.036) reduced foot growth with a trend (p=0.082) observed for subjects with heavy marijuana exposure (ADJ>0.0819; Table 6).

Maternal cannabis use during pregnancy or during the cumulative pre- and during pregnancy period failed to show a significant association with body length development (Tables 4 and 5). Consistently, there was no significant contribution of the pattern of marijuana use reported during pregnancy on body length (Table 6). However, maternal alcohol use during pregnancy was associated with a significant reduction of fetal body length (0.44 cm; p=0.029; Table 4).

Head circumference was not significantly associated with maternal marijuana, cigarette, or alcohol use during pregnancy (Table 4). However, the cumulative maternal alcohol use reported before and during pregnancy revealed a significant effect of alcohol exposure on head circumference measures in this sample (p=0.025; Table 5). There were no significant effects evident for the amount or frequency of marijuana use on head circumference (Table 6).

Ponderal index was not significantly associated with maternal use of marijuana, cigarette, or alcohol either during pregnancy or when considering the cumulative prepregnancy period (Tables 4–6).

Table 6

Fetal development in regard to the amount (no. of joints normally used) and frequency (times/week) of maternal marijuana use reported during pregnancy after adjusting for gestation age, as well as the reported amount of maternal alcohol and cigarette use[a]

	Estimate	CI lower	CI upper	p-value
Weight				
Amount	−9.34	−21.05	1.38	0.0850
Frequency	−34.98	−80.32	10.36	0.1290
0>ADJ<0.40	−15.09	−53.71	23.52	0.4399
0.4≥ADJ<0.89	−54.19	−103.59	−4.78	0.0319
ADJ≥0.89	−22.17	−65.90	21.54	0.3167
Foot length				
Amount	−0.08	−0.14	−0.02	0.0071
Frequency	−0.27	−0.50	−0.03	0.0257
0>ADJ<0.40	−0.10	−0.30	−0.10	0.3270
0.4≥ADJ<0.89	−0.28	−0.54	−0.02	0.0362
ADJ≥0.89	−0.20	−0.43	0.03	0.0819
Body length				
Amount	−0.18	−0.55	0.19	0.3465
Frequency	−0.47	−1.92	0.99	0.5273
0>ADJ<0.40	−0.09	−1.29	−1.29	0.8766
0.4≥ADJ<0.89	−1.13	−2.73	−0.46	0.1618
ADJ≥0.89	−0.15	−1.56	1.27	0.8386
Head circum.				
Amount	−0.11	−0.42	0.21	0.5146
Frequency	−0.14	−1.34	1.06	0.8137
0>ADJ<0.40	−0.14	−1.03	0.75	0.7574
0.4≥ADJ<0.89	−0.33	−1.58	0.90	0.5904
ADJ≥0.89	−0.02	−1.19	1.16	0.9741
Ponderal index				
Amount	0.00	−0.05	0.05	0.9713
Frequency	−0.12	−0.30	0.06	0.1911
0>ADJ<0.40	−0.05	−0.11	0.20	0.5450
0.4≥ADJ<0.89	−0.10	−0.32	0.11	0.3357
ADJ≥0.89	−0.06	−0.25	0.12	0.5052

The pattern of marijuana use is also presented in regard to the categorized groups for the average number of joints/day (ADJ) as compared to the reference nonuser group (ADJ=0).

N=85 for the nonuser group; N=30 for the group reporting marijuana use during pregnancy (N=13, 7, and 10 for light, moderate, and heavy use, respectively).

[a] Subjects reporting nonuse but had positive cannabis toxicology were excluded.

4. Discussion

Consistent with previous evaluations of the trend of drug use in the innercity community currently investigated [35], pregnant women in this population had a high rate of marijuana use (31.6%) with a lower incidence of cocaine (4%) and opiate (2.2%) intake. The high use of marijuana in many communities [4,43] is disturbing in light of the current findings revealing an adverse influence of early prenatal cannabis exposure on various indices of fetal growth. Despite the strong correlations apparent between the four primary developmental measures, body weight and foot length were the variables that that were significantly associated with maternal marijuana use in the mid-gestation fetuses.

The present data extend previous observations of a harmful influence of maternal marijuana use on fetal growth evaluated in newborns by documenting that the negative impact of marijuana exposure is already apparent by mid-gestational fetal development. A number of investigations have provided evidence that the pattern of marijuana exposure is an important variable in regard to fetal growth. Newborn infants with frequent and regular marijuana exposure throughout pregnancy have significant reductions in birth weight as compared to those with infrequent exposure to the drug [15]. Fried et al. [20] also reported reductions of fetal growth in subjects with >5 joints/week. In addition, increasing frequency of marijuana use was found to be directly associated with increasing decrements in birth weights in one cohort of women; the findings were not, however, replicated in a second cohort in that investigation [30]. The current study provides evidence which substantiates that the pattern, particularly the amount, of maternal marijuana use is linked with a greater reduction of fetal growth, primarily for foot length in this population. Although one would predict that heavy maternal marijuana use would be most associated with the greatest impairment of fetal growth, significance was only primarily evident for those with moderate, regular marijuana exposure of approximately three to six joints/week. It is impossible, however, to exclude the impact of heavy marijuana exposure given the small sample size in our population.

As compared to body weight and foot length, there was no significant effect of maternal marijuana use on fetal body length and head circumference in the present study. Positive relationships between utero marijuana exposure and head circumference [17,39] or body length [50] have been documented in newborns. Thus, these growth parameters may be more sensitive to marijuana exposure during later stages of pregnancy. The greater sensitivity currently observed for foot growth with early prenatal marijuana (and cigarette) exposure could be related to the special feature of fetal blood flow which makes the lower limbs particularly sensitive to hypoxia [31]. It is well established that intrauterine hypoxia is induced by cigarette and marijuana exposure which could therefore account for the greater impact of these substances on fetal foot length growth. Although ponderal index is widely used as an indicator of fetal growth, this variable was only weakly correlated to the other fetal growth measures and to gestational age in the early developmental period studied. The lack of significant effect observed for the ponderal index is not unexpected since variables of direct growth measures, such as body weight, have been shown to be a better predictor of in utero growth retardation than ponderal index [23], and ponderal index, which reflects symmetrical growth, is more sensitive to disturbances in late pregnancy [42]. Moreover, no alterations in ponderal index have previously been observed in association with prenatal marijuana exposure [16,19].

Consistent with findings from other investigations, women in the current marijuana group were more likely to smoke cigarettes and drink alcohol during pregnancy [10,20,48]. Of these substances, alcohol use had the most significant adverse impact on all primary growth measures perhaps due to fewer women reducing their alcohol intake as compared to cigarette smoking during pregnancy. Although both nonusers and marijuana users tended to decrease their cigarette smoking approximately twofold during pregnancy, the same was not apparent for alcohol use. Alcohol intake during pregnancy was reported in 54% and 23% of the marijuana and nonuser groups, respectively. Moreover, the number of subjects with moderate and heavy cigarette use was quite low which also could account for the weak contribution observed for maternal cigarette smoking on fetal development in this population. The lack of significant effect of cigarette on fetal growth parameters, except for foot length, is most likely due to the findings that cigarette smoking during early pregnancy has a weaker impact on fetal growth as compared to cigarette smoking in the later stages of pregnancy [33,38,45]. Nevertheless, the fact that significant marijuana-related effects were revealed when adjusted for both maternal cigarette and alcohol use emphasize an important contribution of marijuana exposure during the first half of pregnancy on fetal growth.

Consideration of the cumulative prepregnancy history of marijuana use revealed similar but weaker influence on all growth measures compared to drug exposure limited to pregnancy. It would thus appear that drug exposure during the very early stages of pregnancy, where maternal drug use patterns frequently mimic the prepregnancy period, had minimal contribution to the developmental effects currently observed. A limitation of any study of this kind is the reliance on the self-report of drug use. It is generally acknowledged that self-report of illicit drug use is more biased than the self-report of legal substances, such as alcohol and cigarettes. Underreporting of drug use is particularly evident during pregnancy [13,29]. The fact that women in the present study were already in the process of a voluntary abortion at the time of recruitment for the study might lend itself to more accurate drug reporting. In line with this speculation, 23% of women admitted marijuana use during pregnancy although the drug toxicology was negative. However, we also determined that approximately 18% of the women in this study denied marijuana use during pregnancy but had a positive toxicology. It is quite likely that the number of subjects with early light marijuana use could be misjudged if there is a long time span between the mother's last marijuana use and the time of testing. Such limitations make it likely that there is an underestimation of the influence of marijuana in the present study since there may be marijuana users in the nonuser group. Moreover, light and chronic marijuana users would be expected to metabolize cannabinoids at different rates [34] which could also confound the interpretation of the urine analyses.

Other considerations should be noted about the current study. It included only women who were in the process of a voluntary saline-induced abortion, were within the mid-gestation stage of pregnancy, provided a detailed report of their drug use and medical history, and had a fetal expulsion time ≤24 h (required for subsequent neurochemical/molecular analyses being carried out on the fetal brain specimens). As such, the sample size in this study was small and limited the statistical power. Another limitation of the present investigation is the lack of direct information regarding maternal nutrition [36,49] and body weight measurement [12] which can impact fetal growth. Furthermore, fetal growth is also influenced by maternal weight gain during pregnancy [37,44], but this could not be assessed in the current study because maternal contact only occurred at a single time period. Nevertheless, information obtained from maternal interviews revealed that a similar percentage (between 45% and 50%) of women in the marijuana user and nonuser groups had morning sickness and/or heartburn that negatively affected their eating habit and weight gain during the first trimester of pregnancy. Moreover, there was no significant correlation between the reported early maternal weight loss and the fetal growth measures. The fact that the statistical analyses covaried for maternal nicotine and alcohol use, which are often associated with poor nutrition [7,24], would also appear to discount general deficits in maternal nutrition as a major factor for the fetal growth impairments documented in the marijuana-exposed group.

The consequences of the long-term impact of prenatal marijuana effects on fetal development on health and behavior are still being evaluated. Follow-up studies of children exposed to marijuana during pregnancy have found inconsistent long-lasting impairments in growth parameters beyond infancy [5,19]. However, longitudinal studies have generally documented significant associations of in utero marijuana exposure with impairments of cognitive development and behavioral disturbances, such as inattention, hyperactivity, and impulsivity [18,22,41]. Thus, early exposure to marijuana that appears to affect fetal growth could potentially have an impact on brain development. Several studies have documented an association between small birth weight and impaired neurobehavioral function (e.g., see Ref. [27]). Our recent studies have revealed that mid-gestation fetuses exposed to marijuana have discrete molecular alterations in brain regions that are important for emotional function and behavior [46].

Overall, the current investigation provides data suggestive of detrimental effects of early maternal marijuana intake on the mid-gestation fetus. The results also emphasize that the pattern of maternal marijuana use is of importance to fetal foot length and body weight growth. Thus, in addition to alcohol and cigarettes, information should be given to women about the potentially harmful effects on fetal development of smoking even marijuana during early pregnancy.

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Effects of prenatal cigarette and marijuana exposure on drug use among offspring

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Abstract

The present study investigated whether maternal cigarette smoking and marijuana use during pregnancy were associated with an increased risk of initiation and daily/regular use of such substances among one hundred fifty-two 16- to 21-year-old adolescent offspring. The participants were from a low risk, predominately middle-class sample participating in an ongoing, longitudinal study. Findings indicated that offspring whose mothers reported smoking cigarettes during their pregnancy were more than twice as likely to have initiated cigarette smoking during adolescence than offspring of mothers who reported no smoking while pregnant. Offspring of mothers who reported using marijuana during pregnancy were at increased risk for both subsequent initiation of cigarette smoking (OR=2.58) and marijuana use (OR=2.76), as well as daily cigarette smoking (OR=2.36), as compared to offspring of whose mothers did not report using marijuana while pregnant. There was also evidence indicating that dose–response relationships existed between prenatal exposure to marijuana and offspring's use of cigarettes and marijuana. These associations were found to be more pronounced for males than females, and remained after consideration of potential confounds. Such results suggest that maternal cigarette smoking and marijuana use during pregnancy are risk factors for later smoking and marijuana use among adolescent offspring, and add to the weight of evidence that can be used in support of programs aimed at drug use prevention and cessation among women during pregnancy.

Keywords: Cigarettes; Marijuana; Prenatal; Adolescent; Initiation and regular use of drugs; Gender differences

1. Introduction

Cigarettes and marijuana are two of the most commonly used non-medicinal drugs that are taken during pregnancy [13,34]. Results from several epidemiological studies indicate that maternal cigarette smoking during pregnancy might induce a predisposition in offspring to initiate cigarette smoking later in life [9,25,30]. In a retrospective study of two separate cohorts, the authors reported approximately a 4-fold increased risk of tobacco use and persistence in tobacco use among female (but not male) offspring who were exposed to tobacco prenatally; this association was found to be independent of mothers' postnatal smoking and the child's age [30]. In another report, it was found that maternal cigarette smoking during pregnancy was significantly associated with higher levels of child behaviour problems and that these behaviour problems increased the likelihood of smoking among daughters between the ages of 9 and 17 [25]. Results from a prospective study of lower class 10-year-old children also suggest that maternal cigarette smoking during pregnancy heightens the risk for tobacco experimentation [9]. Children exposed to tobacco at the level of at least one-half pack of cigarettes per day during gestation had a 5.5-fold increased risk for early tobacco experimentation, after controlling for prenatal exposure to other substances and their mothers' current smoking habits. The only factor that produced a greater risk of early experimentation was exposure to smoking within the child's peer group [9]. These above studies suggest a positive association between maternal cigarette smoking during pregnancy and risk of subsequent smoking initiation among offspring that is independent of the mother's postnatal smoking.

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Recent results from a 30-year prospective study indicate that maternal cigarette smoking during pregnancy is also a risk factor for subsequent nicotine dependence among offspring [4]. In this report, offspring whose mothers reported smoking a pack or more of cigarettes per day while pregnant were significantly more likely to meet the *Diagnostic and Statistical Manual of Mental Disorders—Third Edition* [3] criteria for lifetime tobacco dependence than offspring of mothers reporting no maternal smoking during pregnancy. This association remained significant after adjustments for offspring's gender and age, and maternal socio-economic status (SES) and age at pregnancy. In contrast to the finding pertaining to lifetime tobacco dependence, this prospective study reported similar odds of ever smoking and becoming a regular smoker (defined as smoking daily for 30 days or more at some point during one's lifetime) for offspring of maternal smokers and non-smokers [4]. This latter observation is inconsistent with previous reports from other investigators (e.g., Refs. [9,25,30]). It is of interest to note that data on offspring's postnatal exposure to cigarette smoke was not collected or considered in this study [4].

While the mechanisms underlying the association between maternal cigarette smoking during pregnancy and subsequent smoking among offspring remain to be determined, a physiological explanation is plausible. Nicotine and other substances in cigarette smoke cross the placental barrier. The nicotine that passes from the mother to the fetus stimulates nicotinic receptors, which are reported to be present in the fetal brain as early as the first trimester [26]. Since nicotine stimulates the actions of cholinergic neurons and enhances activity in dopaminergic systems involved in addictive behaviour [11], it has been suggested that nicotine and other substances released by maternal smoking may affect the fetus, perhaps through the nicotine input into the mesolimbic dopaminergic reward system, so as to predispose the brain in a critical period of its development to the subsequent addictive influence of nicotine consumed later in life [7,10,30, 31,33,36]. These effects, which may occur even at low nicotine doses and in the absence of notable fetal abnormalities [35], may result in a greater liability to nicotine dependence than in those offspring who have not been prenatally exposed to cigarette smoke [4].

Although several studies have examined the effects of prenatal marijuana exposure on birth outcomes [39], growth measures [8,20], and cognitive and attentional deficits [15,18,19,24], none have assessed the effects of such exposure on subsequent marijuana or cigarette use in offspring. Since nicotine and marijuana both activate the mesolimbic dopaminergic pathway [11,22,41], maternal marijuana use during pregnancy may heighten the risk for the initiation and daily use of cigarettes among adolescent offspring. In a similar vein, an association between prenatal exposure to cigarette smoke and subsequent marijuana use among offspring is also plausible. One study [4] has investigated this hypothesis, but found no evidence of such an association. Given the slight overrepresentation of lower income levels in this study's cohort, however, it is unclear whether this result should be interpreted as possibly representative of only other lower-class samples or if it can be generalized to those that are middle-class.

The objectives of the current study were to determine whether maternal cigarette smoking and marijuana use during pregnancy increased adolescent offspring's risk for: (a) initiating cigarette smoking and marijuana use; and (b) developing patterns of daily cigarette smoking and regular marijuana use. The possibility that dose–response relationships exist between prenatal exposure to cigarettes and marijuana and offspring's use of these substances was also assessed. This article reports findings from the Ottawa Prenatal Prospective Study (OPPS), an ongoing longitudinal investigation that was initiated in 1978, which has examined the neurobehavioural and developmental effects of prenatal exposure to cigarettes and marijuana in a low-risk cohort assessed from birth through adolescence.

2. Methods

2.1. Ottawa prenatal prospective study cohort

The OPPS is designed to explore the effects on offspring of maternal soft drug use during pregnancy in a predominately low-risk, White, middle-class sample. Participants in the OPPS have previously been the focus of a number of inquiries, with cigarettes and marijuana being the primary drugs of interest in recent reports. A total of 698 pregnant women initially volunteered to participate in the OPPS after learning of the study through their doctors, or through notices posted in the waiting rooms of the prenatal clinics in four of the largest hospitals in the Ottawa region. The study was described as an investigation of the influence of prenatal lifestyle habits on the developing fetus [17]. In order to obtain the necessary information, interviews were conducted, usually in the mother-to-be's home, during each trimester remaining in the pregnancy at the time of entrance into the study. The same interviewer was used throughout the entire pregnancy and this, combined with the repetition of interviews, appeared to enhance the rapport between the participant and the interviewer, while also providing an opportunity to assess the consistency of the self-report data [17]. Data were collected on a number of variables including the amounts and patterns of maternal drug use, maternal age, parents' level of education, and the family's SES. For cigarette use, a nicotine score was derived by multiplying the daily average of the number of cigarettes smoked by the nicotine content of the brand specified. Marijuana use was recorded in terms of the number of joints smoked per week. Measurement of alcohol consumption included beer, wine, and liquor use: both the quantity and the pattern of consumption were recorded and converted to ounces of absolute alcohol (AA) per day.

Of the 698 pregnant women who were interviewed, a cohort of 190 children was selected for follow-up studies beyond birth. Included in this cohort were children of women who reported any use of marijuana during pregnancy, children of women who drank alcohol beyond a daily average of 0.85 ounces AA, and children whose mothers smoked an average of at least 16 mg of nicotine (equivalent to one package of cigarettes of average strength) per day during pregnancy. In addition to these 140 youth, children of 50 women who were non-users of marijuana, who abstained or drank little alcohol, and who were non-smokers were randomly selected to also be included in follow-up studies for comparison purposes.

Of these 190 participants who were initially selected for follow-up studies, 152 adolescents ranging in age from 16 to 21 years inclusive were available to participate in the present study. Of the remaining 38 not tested, most involved families had moved out of the Ottawa area. In addition, two families withdrew from the study and a few adolescents were unavailable for testing. No differential loss of participants with respect to drug variables occurred. The final sample of 152 adolescents consisted of 85 males and 67 females, with five 16-year olds, seventy-seven 17-year olds, forty-two 18-year olds, nineteen 19-year olds, seven 20-year olds, and two 21-year olds.

2.2. Instruments and procedure

As part of an overall neuropsychological assessment, an extensive battery of tests was administered to the participants by a trained female assessor who was blind with respect to maternal drug history. Informed consent was obtained from all of the participants, and all testing was conducted in controlled conditions in the laboratory at Carleton University, Ottawa, Canada. Included in the battery was a *Drug History Questionnaire* (DHQ). This questionnaire was developed for use in this research and was adapted from that used in the Monitoring the Future Study (MTF), a longitudinal survey focusing on the lifestyles, attitudes, and preferences of American youth [28]. The DHQ was intended to serve as a measure of participants' history of use, as well as their current use of a number of both licit and illicit substances including cigarettes and marijuana. Variables of interest in the current study from the DHQ include adolescents' initiation and daily (in the case of cigarettes)/regular (in the case of marijuana) use of cigarettes and marijuana, the average number of cigarettes smoked per day, and the mean number of joints smoked per week.

Cigarette smoking initiation referred to adolescents' ever use of cigarettes (beyond just trying out one or two) and it was dichotomously coded (i.e., never smoked cigarettes vs. have smoked cigarettes). Daily cigarette smoking was defined as smoking cigarettes at least once per day and was similarly coded as a binary variable (i.e., not smoking cigarettes at least once per day vs. smoking cigarettes at least once per day). The initiation of marijuana use was defined as the ever use of this substance and was coded as a dichotomy (i.e., never tried marijuana vs. have tried marijuana). Regular marijuana use, however, was defined as using this substance at least once per week, and was similarly coded as a binary variable (i.e., not using marijuana at least once per week vs. using marijuana at least once per week).

A urine sample was obtained from the participants during the neuropsychological assessment, and was immediately frozen until later analyzed by immunoassay for the presence of cotinine (a major degradation product of nicotine metabolism), cannabinoids, cocaine, opiates, and amphetamines. This allowed for a direct, objective validation of the self-report measures of recent drug use. Each urine specimen was analyzed under blind conditions according to the manufacturer's instructions using sensitivity measures that distinguished concentrations from zero with at least 95% confidence. For all assays, the dilution of urine was corrected for by creatinine adjustment.

2.3. Statistical procedures

2.3.1. Group categorization

For analytic purposes, maternal cigarette smoking (average use across pregnancy) was categorized into two groups: non-smoking and smoking (>0 cigarettes/day). Similarly, maternal average marijuana use across pregnancy was also categorized into two groups: no use and use (>0 joints/week). In the case of cigarettes, the control group was comprised of non-smokers. For marijuana, the control group included mothers reporting no use of marijuana during their pregnancies. Prenatal drug exposure was dichotomized in order to increase the sample size of individual cells, which allowed for a satisfactory subject to variable ratio and a reasonable amount of statistical power for the logistic regression analyses [40]. Using two categories for the two prenatal drug exposure variables, a sample of 152 allowed for the detection of a medium effect size (f=.025) with approximately 87% power [6].

2.3.2. Confounding variables

Because this study is a quasi-experimental design, random assignment to groups was not possible. As a result, there was a risk that observed differences would represent only the pre-existing differences in history and background. A number of variables, therefore, were examined as potential confounds prior to conducting any statistical analyses, as has been done in previous publications involving the OPPS (e.g., Refs. [16,21]). Variables that were examined included family income during pregnancy, average level of parental education, postnatal passive cigarette and marijuana smoke exposure, parity, gender of the offspring, maternal use of alcohol and caffeine during pregnancy, and the offspring's age at time of testing. Mother's age at the time of the child's birth was also considered since women who smoke cigarettes during pregnancy tend to be younger than non-smokers [12].

Using one-way analyses of variance, each of the potentially confounding variables were tested for an association with prenatal cigarette smoke and marijuana exposure, respectively, using an alpha level of .10. Variables that were found to significantly relate to either drug at an alpha level of .10 or less were then further examined for an association with each of the outcome variables. Those variables that were found to be significantly related to the outcome variable of interest at an alpha level of .05 or less were subsequently included as covariates in the analysis [27]. Hence, the covariates differed from one analysis to another depending on which potential confounds met the aforementioned criteria. Maternal alcohol use during pregnancy and the maternal use of the drug that was not considered to be of primary interest (i.e., in the case of cigarettes, use of marijuana) were statistically controlled in all analyses.

2.3.3. Analysis

A series of logistic regression analyses for analysis of covariance [38] were performed in order to assess the contribution of prenatal exposure to cigarettes and marijuana on adolescents' initiation and daily/regular use of these substances, while controlling for any potential confounding variables. Separate logistic regression models were conducted for the initiation of cigarette smoking and marijuana use, respectively, as well as for the daily/regular use of these two substances. Each of these models was then recalculated separately for male and female offspring in order to examine possible gender differences. All logistic regression models included two binary predictor variables: one representing maternal cigarette smoking during pregnancy and the other representing maternal marijuana use during pregnancy. An alpha level of .05 was used to test the significance of the omnibus tests for each of the logistic regression models. The significance of each predictor variable was then tested using the Wald test [40] with an alpha level of .05. For the gender-specific analyses, these procedures were followed separately among samples of male and female offspring.

Multiple regression was also utilized to examine the predictive relation of the amount of prenatal exposure to cigarettes (mg nicotine/day) and marijuana (joints/week) to offspring's average use of cigarettes (per day) and marijuana (joints/week). Separate regression models were conducted for offspring's use of cigarettes and marijuana, and each of these models were then recalculated by gender in order to test for possible sex differences. In all of these analyses, maternal cigarette smoking and marijuana use during pregnancy, as well as offspring's use of these substances, were treated as continuous variables.

Assumptions regarding independence of errors, absence of multicollinearity and outliers, normality, linearity, homogeneity of regression slopes (and regression planes and hyperplanes in the case of two or three covariates, respectively), adequacy of expected frequencies, and linearity in the logit were satisfied for all statistical analyses. Maternal cigarette smoking and marijuana use, as well as subjects' use of cigarettes (in terms of amount of nicotine consumed per day), marijuana, and alcohol were all log transformed prior to statistical analyses to reduce positive skewness. Maternal alcohol use was normalized using an inverse transformation, while passive exposure to nicotine at assessment was normalized using a square root transformation [40].

3. Results

The Pearson correlation between the number of cigarettes reported being smoked on average per day and the amount of metabolite detected in the urine was found to be .73 ($p<0.0001$). A Pearson correlation of .71 ($p<0.0001$) was obtained for the concordance between the number of joints reported being smoked on average per week and the amount of metabolite detected in the urine. These high concordance rates validated using self-reported drug use in the statistical analysis as well as lending credence to the self-report information provided on the DHQ that could not be tested by the pharmacological analysis of the urine.

Drug usage and demographic characteristics of the sample across levels of maternal cigarette smoking and marijuana use during pregnancy are presented in Table 1. Annual family income, parity, and mother's age at time of birth are similar to those of women who gave birth in the Ottawa region at the time that participants in the OPPS sample were recruited for the study [14]. More than half (52.6%; $n=80$) of the adolescents in the OPPS reported having initiated cigarette smoking at some point in their lifetime, with the mean age of initiation being 13.99 years ($SD=1.75$). No significant gender differences were found with respect to the initiation of cigarette smoking, $\chi^2(1, N=152)=.32$, $p=0.57$. Of those 80 youth who reported initiating cigarette smoking, 87.5% ($n=70$) reported daily smoking at some point in their lifetime, with such use first occurring at the mean age of 14.72 years ($SD=1.61$). There was also no significant gender difference found with respect to daily cigarette smoking among the adolescents in the OPPS, $\chi^2(1, N=152)=.08$, $p=0.78$.

In terms of marijuana use, the majority (73.7%; $n=112$) of youth reported having initiated the use of this substance at some point in their lifetime, with the average age of initiation being 14.64 years ($SD=1.57$). No significant gender difference was found with respect to the initiation of marijuana use, $\chi^2(1, N=152)=.02$, $p=0.89$. Of those 112 youth in the OPPS who reported initiating the use of marijuana, only 27.6% ($n=31$) reported having used

Table 1
Drug usage and demographic characteristics of prenatal drug exposure groups

	Cigarettes (mg nicotine/day)		Marijuana (joints/week)	
	Not exposed (0)	Exposed (>0)	Not exposed (0)	Exposed (>0)
Sample size	65	87	103	49
Family characteristics				
Annual income (×CAN$1000)[a]	35.93	28.50**	36.33	21.90****
Mother's age[a]	29.66	26.85****	29.54	24.92****
Average parent education[a,b]	3.02	2.29****	2.82	2.15****
Parity	2.02	1.78	2.03	1.57**
Prenatal substance exposure				
Nicotine (mg/day)	.00	1.01****	.55	.64
Marijuana (joints/week)	.21	.30	.00	.80****
Alcohol (oz AA/day)	.86	.87	.87	.86
Postnatal substance exposure				
Passive exposure to nicotine until age 15 (no. of years exposed as a proportion of age)[c]	.25	.66****	.42	.58
Passive exposure to nicotine at assessment (no. of hours exposed both in and out of home)	2.36	3.64***	2.74	3.83**
Passive exposure to marijuana at assessment[d]	1.23	1.21	1.05	1.57**
Offspring characteristics				
Sex, % female	42.00	46.00	44.00	45.00
Age at assessment (years)	18.01	18.07	18.04	18.06
Age of cigarette smoking initiation (years)	14.19	13.89	14.02	13.94
Cigarettes smoked/day	2.39	3.92	2.50	4.89**
Nicotine (mg/day)[e]	.31	.48	.33	.57*
Age of marijuana use initiation (years)	14.68	14.61	14.77	14.42
Joints smoked/week	.23	.24	.14	.42****

Note. Statistical tests were one-way between-subjects analyses of variance.
[a] Values are based on figures obtained at birth of offspring.
[b] Education coded as: 1=did not finish high school; 2=graduated high school; 3=graduated college or university; 4=post-graduate degree.
[c] Data for only 80 subjects on this variable.
[d] Coding for passive exposure to marijuana: 1=more than one month ago; 2=between 1 week and 4 weeks ago; 3=between 2 and 6 days ago; 4=yesterday; 5=today.
[e] Calculated by multiplying the number of cigarettes smoked per day by the nicotine content of the brand specified.
* p=0.05.
** p=0.01.
*** p=0.001.
**** p=0.0001.

marijuana on a regular basis at some point, with such use first occurring at the mean age of 15.30 years (SD=1.67). No significant gender difference was found with respect to using marijuana on a regular basis, $\chi^2(1, N=152)=1.17$, p=0.28.

3.1. Correlates of cigarette smoking behaviour

Offspring of mothers who reported smoking cigarettes while pregnant were more than twice as likely to have initiated smoking later in adolescence, compared to offspring of non-smokers (referent group) after adjusting for maternal marijuana and alcohol use during pregnancy, maternal age at time of pregnancy, and family income (Table 2). Results also revealed that offspring who were prenatally exposed to marijuana had almost three times the odds of initiating cigarette smoking during adolescence compared to offspring whose mothers reported no use of marijuana while pregnant. In terms of gender differences, male offspring of mothers who reported smoking cigarettes while pregnant were more than three times as likely to have initiated smoking during adolescence compared to offspring of non-smokers (Table 3). The association between maternal marijuana use during pregnancy and male offspring's initiation of cigarette smoking was found to be even more pronounced, with offspring exposed in utero to marijuana being almost five times as likely to have initiated cigarette smoking. The associations between

Table 2
Associations between prenatal exposure to cigarettes and marijuana and offspring's cigarette smoking initiation and daily cigarette smoking

Offspring Cigarette Smoking Behaviour (N=152)	N	B	SE	OR (95% CI)[a]
Cigarette smoking initiation[b]				
Prenatal cigarette exposure				
Not exposed	65			1.00
Exposed	87	.77*	.36	2.16 (1.06, 4.39)
Prenatal marijuana exposure				
Not exposed	103			1.00
Exposed	49	.95*	.43	2.58 (1.11, 6.00)
Daily cigarette smoking[c]				
Prenatal cigarette exposure				
Not exposed	65			1.00
Exposed	87	.34	.40	1.41 (.65, 3.06)
Prenatal marijuana exposure				
Not exposed	103			1.00
Exposed	49	.86*	.44	2.36 (1.00, 5.57)

Note. B=unstandardized regression coefficient; SE=standard error; OR=odds ratio; CI=confidence interval.

[a] The reference category for all odds ratios is offspring who were not prenatally exposed to the drug of interest.

[b] Based on a logistic regression model including maternal alcohol use during pregnancy, maternal age at time of pregnancy, and family income as covariates.

[c] Based on a logistic regression model including maternal alcohol use during pregnancy, maternal age at time of pregnancy, family income, and average parental education as covariates.

* $p \leq 0.05$.

prenatal exposure to cigarettes and marijuana and smoking initiation were found to be generally less pronounced for female offspring (Table 3).

With respect to the correlates of daily cigarette smoking, the results showed that offspring of mothers who reported using marijuana during pregnancy were more than twice as likely to smoke cigarettes at least once per day during adolescence compared to offspring whose mothers did not report using marijuana while pregnant, after controlling for maternal marijuana and alcohol use during pregnancy, maternal age at time of pregnancy, family income, and average parental education (Table 2). No significant association was observed between prenatal exposure to cigarettes and later daily cigarette smoking (Table 2), and similar results were yielded from the sex-specific analyses (Table 3). However, the results did show that male offspring of mothers who reported using marijuana while pregnant had more than three times the odds of smoking cigarettes at least once per day as an adolescent compared to offspring of non-users (Table 3). No significant association was observed between in utero exposure to marijuana and daily cigarette smoking among female offspring.

When prenatal exposure to cigarettes (mg nicotine/day) and marijuana (joints/week), and offspring's use of cigarettes (per day) were treated as continuous variables, regression analyses revealed a significant positive association between mother's use of marijuana during pregnancy and the average number of cigarettes smoked per day by offspring (r=.23, p<0.01), controlling for maternal alcohol and cigarette use during pregnancy, family income, and mother's age at time of pregnancy (Table 4). Although prenatal exposure to cigarettes was also positively related to offspring's average number of cigarettes smoked per day, this result only approached statistical significance (r=.15,

Table 3
Gender-specific associations between prenatal exposure to cigarettes and marijuana and offspring's initiation and daily cigarette smoking

Offspring Cigarette Smoking Behaviour (N=152)	Males				Females			
	N	B	SE	OR (95% CI)[a]	N	B	SE	OR (95% CI)[a]
Cigarette smoking initiation[b]								
Prenatal cigarette exposure								
Not exposed	38			1.00	27		.59	1.00
Exposed	47	1.19*	.51	3.28 (1.20, 8.95)	40	.33		1.39 (.44, 4.44)
Prenatal marijuana exposure								
Not exposed	58			1.00	45			1.00
Exposed	27	1.48*	.62	4.37 (1.31, 14.64)	22	.41	.65	1.51 (.43, 5.38)
Daily cigarette smoking[c]								
Prenatal cigarette exposure								
Not exposed	38			1.00	27			1.00
Exposed	47	.77	.55	2.17 (.75, 6.31)	40	−.10	.67	.90 (.24, 3.37)
Prenatal marijuana exposure								
Not exposed	58			1.00	45			1.00
Exposed	27	1.27*	.62	3.56 (1.06, 11.97)	22	.44	.69	1.55 (.40, 5.95)

Note. B=unstandardized regression coefficient; SE=standard error; OR=odds ratio; CI=confidence interval.

[a] The reference category for all odds ratios is offspring who were not prenatally exposed to the drug of interest.

[b] Based on a logistic regression model including maternal alcohol use during pregnancy, maternal age at time of pregnancy, and family income as covariates.

[c] Based on a logistic regression model including maternal alcohol use during pregnancy, maternal age at time of pregnancy, family income, and average parental education as covariates.

* $p \leq 0.05$.

Table 4
Multiple regression analyses predicting offspring's average use of cigarettes and marijuana joints from prenatal exposure to cigarettes and marijuana

Offspring Substance Use (N=152)	B	SE	β	(95% CI)
Cigarettes/day[a]				
Prenatal cigarette exposure[b]	1.39	.73	.16	(.06, 2.83)
Prenatal marijuana exposure[c]	2.85*	1.00	.25	(.86, 4.83)
Joints/week[d]				
Prenatal cigarette exposure[b]	−.08	.06	−.12	(−.20, .03)
Prenatal marijuana exposure[c]	.30**	.07	.34	(.15, .44)

Note. B=unstandardized regression coefficient; SE=standard error; β=standardized regression coefficient; CI=confidence interval.

[a] Based on a regression model including maternal alcohol use during pregnancy, family income, and mother's age at time of pregnancy as covariates.

[b] Expressed in terms of mg nicotine/day.

[c] Expressed in terms of joints/week.

[d] Based on a regression model including maternal alcohol use during pregnancy, family income, mother's age at time of pregnancy, and average parental education as covariates.

* $p \leq .01$.

** $p \leq 0.0001$.

p=0.06; Table 4). When gender differences were examined, the results indicated that prenatal exposure to both cigarettes (r=.25, $p<0.05$) and marijuana (r=.24, $p<0.05$) were significantly associated with increases in the number of cigarettes smoked per day by male offspring, after controlling for potentially confounding variables (Table 5). No significant associations were observed, however, between prenatal drug exposure and the number of cigarettes smoked per day by female offspring (Table 5).

No significant interactive effects of prenatal cigarette and marijuana exposure were noted in any of the above analyses.

3.2. Correlates of marijuana use behaviour

In terms of offspring's marijuana use, findings indicated that offspring of mothers who reported using marijuana while pregnant were almost three times as likely to have initiated the use of marijuana during adolescence compared to offspring of non-users, and this was found to be independent of maternal cigarette and alcohol use during pregnancy (Table 6). However, no significant association was observed between prenatal exposure to cigarettes and offspring's initiation of marijuana use. When gender differences were examined, the results indicated a significant relationship between maternal use of marijuana during pregnancy and the subsequent initiation of marijuana use during adolescence among males. Specifically, male offspring of mothers who reported using marijuana while pregnant had nearly four times the odds of initiating marijuana use compared to offspring whose mothers did not report using marijuana during pregnancy (Table 7). In contrast, no significant associations were observed between prenatal drug exposure and marijuana use initiation among female offspring. Moreover, there was no evidence of a significant association between offspring's regular use of marijuana during adolescence and prenatal exposure to either cigarettes or marijuana for either gender (Table 7).

When prenatal exposure to cigarettes and marijuana and offspring's use of marijuana were treated as continuous variables, results from a multiple regression analysis indicated a significant positive association between maternal use of marijuana during pregnancy and offspring's average number of joints smoked per week (r=.29, $p<0.0001$), independent of mother's use of cigarettes and alcohol during pregnancy, family income, mother's age at time of pregnancy, and average parental education (Table 4). No significant association was noted between prenatal exposure to cigarettes and the average number of joints smoked per week by offspring (Table 4). Results from the

Table 5
Gender-specific multiple regression analyses predicting offspring's average use of cigarettes and marijuana joints from prenatal exposure to cigarettes and marijuana

Offspring substance use (N=152)	Males (n=85)				Females (n=67)			
	B	SE	β	(95% CI)	B	SE	β	(95% CI)
Cigarettes/day[a]								
Prenatal cigarette exposure[b]	2.30*	.93	.26	(.44, 4.15)	1.05	1.27	.12	(.27, 2.63)
Prenatal marijuana exposure[c]	2.82*	1.22	.26	(.40, 5.24)	2.66	1.76	.21	(.59, 7.56)
Joints/week[d]								
Prenatal cigarette exposure[b]	−.01	.09	−.01	(−.18, .16)	−.16	.08	−.31	(−.32, −.01)
Prenatal marijuana exposure[c]	.38**	.10	.42	(.18, .58)	.13	.11	.18	(−.08, .35)

Note. B=unstandardized regression coefficient; SE=standard error; β=standardized regression coefficient; CI=confidence interval.

[a] Based on a regression model including maternal alcohol use during pregnancy, family income, and mother's age at time of pregnancy as covariates.

[b] Expressed in terms of mg nicotine/day.

[c] Expressed in terms of joints/week.

[d] Based on a regression model including maternal alcohol use during pregnancy, family income, mother's age at time of pregnancy, and average parental education as covariates.

* $p \leq 0.05$.

** $p \leq 0.0001$.

Table 6
Associations between prenatal exposure to cigarettes and marijuana and offspring's initiation and regular use of marijuana

Offspring Marijuana Use Behaviour (N=152)	N	B	SE	OR (95% CI)[a]
Marijuana use initiation[b]				
Prenatal cigarette exposure				
Not exposed	65			1.00
Exposed	87	.13	.38	1.14 (.53, 2.41)
Prenatal marijuana exposure				
Not exposed	103			1.00
Exposed	49	1.02*	.46	2.76 (1.11, 6.86)
Regular marijuana use[b]				
Prenatal cigarette exposure				
Not exposed	65			1.00
Exposed	87	.25	.42	1.29 (.57, 2.93)
Prenatal marijuana exposure				
Not exposed	103			1.00
Exposed	49	−.23	.45	.79 (.33, 1.90)

Note. B=unstandardized regression coefficient; SE=standard error; OR=odds ratio; CI=confidence interval.

[a] The reference category for all odds ratios is offspring who were not prenatally exposed to the drug of interest.

[b] Based on a logistic regression model including maternal alcohol use during pregnancy as a covariate.

* $p \leq 0.05$.

gender-specific analyses demonstrated that prenatal exposure to marijuana was significantly associated with increases in the number of joints smoked per week by male offspring (r=.36, p<0.0001), after controlling for potentially confounding variables (Table 5). There was no significant evidence, however, indicating that a similar relationship existed among female offspring (Table 5). Findings also failed to suggest that maternal use of cigarettes during pregnancy was significantly related with offspring's average number of joints smoked per week for either males or females (Table 5).

No significant interactive effects of prenatal cigarette and marijuana exposure were noted in any of the above analyses.

4. Discussion

The results in this report continue the presentation of findings arising from the OPPS—a long-term prospective study of the effects of in utero exposure to cigarettes and marijuana. The current investigation was undertaken to examine the effects of maternal cigarette smoking and marijuana use during pregnancy on subsequent initiation and daily/regular use of these two substances among 16- to 21-year-old offspring. Findings indicated that approximately 53% of offspring reported initiating cigarette smoking, which is consistent with results from various prevalence studies in Canada and the United States [2,5,28]. In contrast, about 46% of the OPPS sample reported daily cigarette smoking, which is considerably higher than rates reported by the MTF Study [28] (i.e., 24%) and the Youth Risk Behavior Surveillance (YRBS) Study [5] (i.e., 16%). The 74% rate of marijuana use initiation in the present work was also substantially higher than corresponding rates reported by other investigators (i.e., 45% in the MTF Study [28] and 40% in the YRBS [5]). Finally, about 20% of offspring in the present study reported regular use of marijuana. Although this rate appears to be similar to that reported by the MTF Study [28] and the YRBS [5], it should be

Table 7
Gender-specific associations between prenatal exposure to cigarettes and marijuana and offspring's initiation and regular use of marijuana

Offspring Marijuana Use Behaviour (N=152)	Males				Females			
	N	B	SE	OR (95% CI)[a]	N	B	SE	OR (95% CI)[a]
Marijuana use initiation[b]								
Prenatal cigarette exposure								
Not exposed	38			1.00	27			1.00
Exposed	47	.40	.51	1.49 (.54, 4.06)	40	−.18	.59	.84 (.27, 2.63)
Prenatal marijuana exposure								
Not exposed	58			1.00	45			1.00
Exposed	27	1.30*	.68	3.67 (.97, 13.88)	22	.75	.65	2.11 (.59, 7.56)
Regular marijuana use[b]								
Prenatal cigarette exposure								
Not exposed	38			1.00	27			1.00
Exposed	47	.27	.53	1.31 (.47, 3.69)	40	.36	.72	1.43 (.35, 5.81)
Prenatal marijuana exposure								
Not exposed	58			1.00	45			1.00
Exposed	27	−.14	.56	.87 (.29, 2.61)	22	−.36	.75	.70 (.16, 3.01)

Note. B=unstandardized regression coefficient; SE=standard error; OR=odds ratio; CI=confidence interval.

[a] The reference category for all odds ratios is offspring who were not prenatally exposed to the drug of interest.

[b] Based on a logistic regression model including maternal alcohol use during pregnancy as a covariate.

* $p \leq 0.05$.

noted that the definition of regular marijuana use in the current investigation (i.e., at least once per week) is more conservative than that of the MTF Study (i.e., use in the past 30 days) and the YRBS Study (i.e., use at least once during the past 30 days). Thus, the rate of regular marijuana use, like daily cigarette smoking, is actually higher in the current work compared to both of these national prevalence studies [5,28].

A number of unique findings were observed in the current study. Most notably was the evidence of a positive association between maternal marijuana use during pregnancy and risk of subsequent marijuana use initiation among adolescent offspring. Although no significant association was observed in relation to offspring's regular use of marijuana, this finding should be interpreted with some caution as this observation was based on only 31 offspring reporting regular use of marijuana. When the drug use variables were treated as continuous variables, there appeared to be a dose–response relationship between the number of joints smoked per week by the mother while pregnant, and those later consumed by her offspring. Interestingly, the impact of prenatal marijuana exposure on offspring's marijuana use was found to be stronger for male than female offspring. The present work is also the first to suggest that the impact of maternal marijuana use during pregnancy is not specific to subsequent marijuana use. That is, the results revealed a positive association between prenatal exposure to marijuana and offspring's risk of both cigarette smoking initiation and daily smoking during adolescence, with the impact of such exposure also being more pronounced among male offspring. A dose–response relationship was also noted between mother's use of marijuana during pregnancy and the number of cigarettes smoked per day by offspring.

The present findings are also consistent with earlier investigations (e.g., Refs. [9,25,30]) reporting a positive association between maternal cigarette use during pregnancy and risk of subsequent cigarette smoking initiation among adolescent offspring. However, there was no significant evidence in the current work suggesting that prenatal exposure to cigarette smoke increases offspring's risk of daily smoking during adolescence, and this result is congruent with findings pertaining to regular cigarette smoking from other researchers [4]. Although Kandel and colleagues [30] reported that maternal cigarette smoking during pregnancy increased female offspring's odds of persisting in cigarette smoking, their definition of persistent smoking (i.e., smoking in last three months) differed from that used for daily smoking in the present study (i.e., smoking at least once per day), which was adapted from the MTF Study [28]. Results from the present investigation also indicate that the impact of maternal cigarette smoking during pregnancy on offspring's smoking initiation is stronger for males than females. While this result is in accordance with the findings concerning the impact of prenatal marijuana exposure, it is inconsistent with the results of other investigators [30] who have reported a greater maternal effect of cigarette smoking for females. It is unclear why this discrepancy has occurred, but it may possibly be the result of differences between the OPPS sample and the cohorts of youth studied by Kandel et al. [30]. While the OPPS sample is predominately White and middle-class, Kandel et al. studied a cohort of youth that is representative of adolescents in New York State public high schools during 1971–72, and another cohort that is representative of youth born from 1957 through 1963 in the coterminous United States.

Consistent with the results of other investigators [4], the current work did not find evidence supporting an association between maternal cigarette smoking during pregnancy and risk of later marijuana use among offspring. Such findings suggest that the impact of prenatal cigarette smoke exposure is specific to subsequent cigarette smoking and that any fetal perturbances associated with maternal smoking during pregnancy do not affect adolescent marijuana use. This observed specificity is also in accordance with other studies suggesting a pathophysiological pathway between prenatal exposure to cigarettes and smoking initiation (e.g., Refs. [29,30]), and suggests that the pathophysiological pathways for marijuana use are different from those for cigarette smoking [4]. Interestingly, these findings regarding the specificity of cigarettes are the opposite of what was observed with respect to marijuana, with the current study indicating that maternal marijuana use during pregnancy is associated with subsequent marijuana and cigarette use among adolescent offspring.

The present findings indicate that fetal exposure to cigarettes and marijuana may have a significant impact on the initiation of cigarette smoking and marijuana use in adolescent offspring, and a number of mechanisms may contribute to this relationship. In utero cigarette exposure may cause physiological changes resulting in increases in nicotine receptors that may increase susceptibility to later cigarette use [7,10,31,33,36]. Such exposure to cigarettes and marijuana may also predispose the brain via the mesolimbic dopaminergic reward system to the subsequent addictive influence of nicotine consumed later in life [30]. Interestingly, results from a recent animal study [1] indicate that maternal cigarette smoking during pregnancy alters offspring's subsequent response to nicotine in adolescence, suggesting that biological mechanisms underlie the association between prenatal cigarette smoke exposure and adolescent smoking in offspring. As data indicates that the cannabinoid receptors are present in the placenta [37] and the fetal and neonatal brain [23], it is possible that prenatal exposure to marijuana also sensitizes the brain to the subsequent influence of marijuana consumed later in life. The transmission of a genetic predisposition for drug use, or other conditions such as depression or anxiety that are associated with smoking and marijuana use [9,29], is yet another potential explanation for the observed association between maternal cigarette and marijuana use during pregnancy and offspring's subsequent use of these two substances.

In interpreting and generalizing the present findings, several caveats must be considered. The relatively small sample size, particularly for the gender-specific analyses, may lead to instability in the coefficients. The current work was also unable to statistically control for the effect of postnatal tobacco exposure, as there was an excess of missing data on this variable. In addition, this study did not collect data on parent's current use of cigarettes or other substances. Finally, since the OPPS is comprised of a predominately White, middle-class, low-risk sample, the findings from the current work may not be generalizable to other ethnic groups or other samples of youth that are not similarly low-risk.

In summary, the present findings indicate differential effects of maternal cigarette smoking and marijuana use during pregnancy on risk of subsequent substance use among adolescent offspring. While prenatal exposure to cigarettes appears to be linked with offspring's cigarette smoking initiation, the data suggest that in utero exposure to marijuana is associated with cigarette smoking and marijuana use initiation, as well as daily cigarette smoking in offspring. There also appears to be dose–response relationships between prenatal exposure to marijuana and offspring's use of cigarettes and marijuana. In contrast to reports from national prevalence studies [5,28], the current investigation yielded substantially higher rates of daily cigarette smoking and the initiation and regular use of marijuana among adolescent offspring of maternal drug users. Therefore, from a public health perspective, a reduction in rates of cigarette smoking and marijuana use may not only yield direct health benefits for the substance users themselves, but it may also have unanticipated benefits for their offspring including reduced risk of subsequent cigarette smoking and marijuana use later in life which may in effect influence the use of other illicit substances [32].

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PRICE AND ENFORCEMENT EFFECTS ON COCAINE AND MARIJUANA DEMAND

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This article estimates equations for past year cocaine and marijuana use among adult and juvenile respondents of the 1990–97 National Household Surveys on Drug Abuse. Unlike most previous studies, we control for the monetary price of marijuana, probabilities of arrest for marijuana and cocaine possession, and state fixed effects. Results indicate that cocaine prices are inversely related to adult cocaine and marijuana demand but are unrelated to juvenile drug demand, marijuana price effects are always statistically insignificant, estimated price effects are inflated when state effects are omitted, and increases in each arrest probability diminish both types of drug use. (*JEL* K42, I18, D12)

I. INTRODUCTION

The responsiveness of cocaine and marijuana demand to changes in their prices is a key determinant of the effectiveness of illegal drug enforcement policy. By harassing sellers and seizing drugs, enforcement attempts to reduce the consumption of illegal drugs by restricting their supply and thereby raising their prices. Even if enforcement is able to increase drug prices, its success in reducing illegal drug use depends on the elasticity of drug demand with respect to drug prices. Reciprocally, unless this elasticity is close to zero, legalization of cocaine and marijuana would likely increase their consumption substantially by drastically reducing their prices.

A complementary goal of enforcing cocaine and marijuana possession violations is to reduce their demand at prevailing prices. This occurs through both incarceration of drug users who will no longer be able to purchase drugs and deterrence of drug consumption by potential users. Price and enforcement effects may be dissimilar if consumers respond differently to changes in their budget constraints than to changes in expected punishment. In particular, the relative magnitudes of the responses iu drug demand to changes in possession arrest probabilities and prices is an important determinant of how enforcement resources can most efficiently be allocated between buyers and sellers. But in spite of this policy relevance, there is little direct evidence on the relationship between

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ABBREVIATIONS

DEA: Drug Enforcement Administration
FBI: Federal Bureau of Investigation
MSA: Metropolitan Statistical Area
MTF: Monitoring the Future
NHSDA: National Household Surveys on Drug Abuse
STRIDE: System to Retrieve Information from Drug Evidence

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arrest probabilities for cocaine and marijuana possession and demand for these drugs.

Meanwhile, the relationship between the consumption of cocaine and marijuana is both theoretically and empirically uncertain. In theory, cocaine and marijuana act as substitutes in the production of intoxication but also can provide complementary intoxicating effects. Empirically, this relationship determines whether policies designed to reduce demand for one drug have effects on the other that reinforce or counteract the impacts of policies designed specifically for that other drug. For instance, marijuana possession arrests more than doubled nationally between 1990 and 1997, both in number and as a fraction of total arrests. This might have reinforced any effect of cocaine possession enforcement on cocaine use if the two drugs are complements but had an unintended counteractive effect if they are substitutes.

This study provides evidence on the impacts of cocaine and marijuana prices and possession violation enforcement on the demand for these drugs. We analyze data on past year cocaine and marijuana use among 12- to 39-year-old respondents to the annual 1990–97 National Household Surveys on Drug Abuse (NHSDA). Along with various individual characteristics, the set of explanatory variables includes regional prices of cocaine and marijuana, state-level measures of the probability of arrest for cocaine and marijuana possession, and fixed effects for states and years. Our goals are to estimate the size of the response in the demand for cocaine and marijuana to changes in their prices, to do the same with respect to changes in possession violation enforcement intensity, and to examine whether unmeasured state characteristics can potentially bias estimated price and enforcement effects.

The analysis is novel in several ways. Most important, it is the first study of cocaine demand to control for state fixed effects in nationally representative data. Previous studies impute cocaine prices at the state level and use both cross-state and temporal variation in prices to identify price effects. But it is possible that a substantial component of cross-state price variation is explained by unobservable, time-invariant state-level factors that also explain cross-state variation in illegal drug use. For instance, states with more permissive attitudes regarding drug use are likely to have both lower cocaine prices and higher drug prevalence rates than antidrug states. Cocaine price elasticities estimated from analyses that ignore these fixed state effects might overstate the impact of an exogenous price change on the change in drug use within an average state, which is the true elasticity of interest.

The inclusion of state fixed effects necessitates constructing our cocaine prices differently than previous studies. Motivated by evidence from Rhodes et al. (1994) and Caulkins (1995) that region and population size are the crucial geographic determinants of cocaine prices, we calculate cocaine prices that vary by census division and metropolitan area size. This allows for the temporal price variation necessary to simultaneously identify cocaine price effects and fixed state effects. In addition, our analysis is the first to examine the effect of marijuana prices on cocaine demand and to explicitly estimate the effect of cocaine possession arrest rates on cocaine and marijuana demand and of marijuana possession arrest rates on cocaine demand.

The article proceeds as follows. The next section reviews the relevant literature. Section III discusses the NHSDA data as well as separate data on cocaine and marijuana prices and possession arrests with which we merge the NHSDA data. Section IV presents our empirical methodology. Section V reports and examines the estimation results, and section VI summarizes our conclusions.

II. PREVIOUS STUDIES

Several recent studies estimate elasticities of cocaine and marijuana demand with respect to the price of cocaine. Typically the dependent variables in these studies are binary indicators of whether or not the respective drug is used, so that estimated price elasticities are with respect to participation in drug use rather than actual drug consumption.[1]

1. Some of the reviewed studies also estimate equations for the number of times cocaine or marijuana is used by users and then calculate a price elasticity of consumption frequency by adding the elasticities of participation and frequency conditional on participation. Although it is possible that prices and arrest rates have differential effects on these two decisions, we confine