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ABSTRACT

This book describes an analysis of the cost-effectiveness of model school-based drug prevention programs at reducing cocaine consumption. It compares prevention's cost-effectiveness with that of several enforcement programs and with that of treating heavy cocaine users. It also assesses the cost of nationwide implementation of model prevention programs and the implementation's likely effect on the nation's cocaine consumption. There is considerable uncertainty surrounding the magnitude of the effects of school-based drug prevention programs. However, a few things are clear. Nationwide implementation of a model program today is affordable, but it would not dramatically affect the course of drug use and the benefits would take years to accrue. Alternatively, the range of cost-effectiveness estimates derived for this book for prevention is comparable to that previously derived for different enforcement interventions. Furthermore, implementing model prevention programs seems to be justifiable in the sense that the benefits produced would likely outweigh the costs of the resources used. Includes tables and figures depicting results of the analysis. Ten appendixes address the implications and effects of cocaine, marijuana, alcohol, and cigarette use. (Contains 82 references.) (Author/MKA)

An Ounce of Prevention

A Pound of Uncertainty

The Cost-Effectiveness of School-Based Drug Prevention Programs

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DRUG POLICY RESEARCH CENTER

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4

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Cover design by Eileen Delson La Russo

In memory of Peter Rydell,
friend and modeler extraordinaire

This book describes an analysis of the cost-effectiveness of model school-based drug prevention programs at reducing cocaine consumption. We compare prevention's cost-effectiveness with that of several enforcement programs and with that of treating heavy cocaine users. We also assess the cost of nationwide implementation of model prevention programs and the implementation's likely effect on the nation's cocaine consumption.

There is considerable uncertainty surrounding the cost-effectiveness of school-based drug prevention programs—even the model programs to which we restrict the scope of this study. Hence we provide a range of cost-effectiveness estimates. The range is comparable to that previously derived for different enforcement interventions. Nationwide implementation of a model program would be affordable, but it would not dramatically affect the course of drug use, and also the benefits would take years to accrue. Nevertheless, implementing model prevention programs seems to be justifiable in that the benefits produced would likely outweigh the costs of the resources used.

This is the latest in a series of publications by RAND's Drug Policy Research Center that addresses the cost-effectiveness of drug control strategies. The other available volumes are as follows:

- Susan S. Everingham and C. Peter Rydell, *Modeling the Demand for Cocaine*, MR-332-ONDCP/A/DPRC, 1994.

- C. Peter Rydell and Susan S. Everingham, *Controlling Cocaine: Supply Versus Demand Programs*, MR-331-ONDCP/A/DPRC, 1994.

CONTENTS

9

12

13

TABLES

Of the various possible approaches toward alleviating America's problems with illicit drugs, the greatest emphasis—in both political rhetoric and dollars allocated—has long been placed on enforcement against traffickers, dealers, and users. Recently, however, public-opinion polls have shown strong support for prevention—even a preference in some cases.

But how effective is prevention? How much does it cut drug use? How does it compare, dollar for dollar, with other approaches to drug control such as enforcement or treatment? Would it be fiscally feasible to put every American youth through a cutting-edge drug prevention program, and, if so, would it have a modest or substantial effect on the severity of America's drug problems?

We have sought to provide quantitative answers to these questions. The answers all take the form of plausible ranges of values, not just point estimates, because there is considerable uncertainty involved in the data and the calculations—hence, this book's title.

Some of the uncertainty is the natural and largely unavoidable consequence of the complexity of human behavior and social systems. Some arises because most prevention research has focused on only a subset of the factors relevant for estimating prevention's aggregate effect. Indeed, we are not just interested in providing estimates but also in creating a logical structure or framework for understanding how to think about prevention's effectiveness and the associated uncertainties and how research should best proceed to reduce the uncertainty.

17

To begin to answer the general question of how effective prevention is at controlling problems associated with illicit drugs, we start with the following more specific question: Dollar for dollar, by how much *can model* school-based prevention programs reduce the nation's *cocaine* consumption? The italicized words are important, because if we simply ask whether prevention has typically been as cost-effective as other strategies, the answer is even simpler: No. Many programs have been found not to be effective. But we want to give prevention its best shot. Thus, for our analysis, we have chosen two model programs that have been demonstrated to affect student drug use, but which are not yet widely implemented: Project ALERT and Life Skills Training. Both of these are school-based programs that seek to inculcate in adolescents the skills to resist social influences to use drugs. We focus on cocaine use because that is the country's most problematic illicit drug and the one we have previously used in assessing the cost-effectiveness of other drug control interventions.

As suggested above, we find that we cannot answer the cost-effectiveness question as precisely as we would like to. Our estimate of prevention's cost-effectiveness is less certain than our past estimates for enforcement and treatment were. In recognition of the uncertainty, we define the factors contributing to prevention's cost-effectiveness so that they can simply be multiplied together to obtain the final result. Besides our mid-range preferred estimate of each factor, we present a reasonable low and high value. We carry these low and high estimates through the analysis to show how strongly the uncertainty qualifies our conclusion regarding the relative cost-effectiveness of prevention and enforcement.

EFFECT PER PROGRAM PARTICIPANT

Figure S.1 presents the factors in our analysis. We discuss each briefly in turn, from top to bottom.

Proportion of Persons Who Would Have Ever Used Cocaine
X
Lifetime Cocaine Consumption Per User
X
Percentage Reduction in Cocaine Use Due to Prevention Program
X
Discount Factor
X
Social Multiplier
X
Market Multiplier
X
Causation/Correlation Ratio
X
Scale-Up Factor
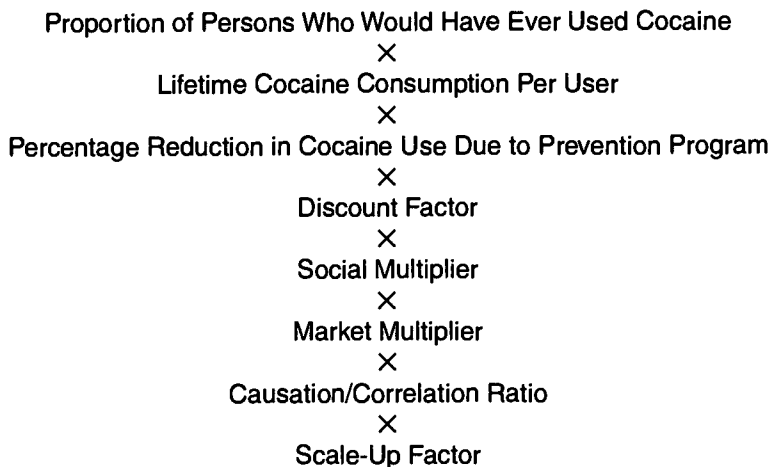
**Figure S.1—Factors Contributing to the Effectiveness of Prevention
at Reducing Cocaine Consumption**

Proportion of Persons Who Would Have Ever Used Cocaine

To know how much prevention will re-
duce consumption, we need to know
how much cocaine the program par-
ticipants would have used without pre-
vention. That depends on the propor-
tion who would ever use cocaine and on
the amount that eventual users would

Low	Middle	High
13%	20%	27%

consume over their lifetime. From the National Household Survey of
Drug Abuse (NHSDA), we know the historically low and high propor-
tions of persons using cocaine. We take these values as our low and
high estimates; for our preferred mid-range estimate, we take the
average of the two.

Lifetime Cocaine Consumption Per User (grams)

This factor is not known and cannot be measured easily, so we developed six alternate ways of estimating it. Each way individually has weaknesses, but collectively they establish a reasonable range.

Low	Middle	High
225	350	475

Percentage Reduction in Cocaine Use Due to Prevention Program

Together the first two factors describe how much cocaine the average adolescent would eventually use in the absence of a prevention program.[1] What effect would model prevention programs have on this? We cannot answer this question directly since data on lifetime cocaine use by program participants are not available.   However, the program evaluations furnish good data

Low	Middle	High
2.9%	7.6%	13.6%

regarding the effect of prevention on marijuana initiation, and we know from NHSDA data that delays in marijuana use are associated with two things: lower likelihood of ever using cocaine and, for those who do use it, lower amount used. Our low estimate of the effect on *marijuana* initiation comes from the 5 percent reduction achieved by Project ALERT, which was implemented only during seventh and eighth grades. The high estimate (17 percent) is the average of ALERT and Life Skills reductions in other indicators of marijuana use. For a mid-range estimate, we split the difference.

But how long can these effects be expected to last—just through adolescence or all life long? We have no information on this because the evaluation data follow subjects only through the end of high

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[1]Because the proportions ever using are less than 50 percent, the *typical* adolescent will use none at all. For accounting purposes, however, it is useful to estimate the average across all individuals.

school. So we assume, in the worst case, that the program merely delays marijuana initiation until after high school. For the best case, we assume that reductions in use are permanent. For a mid-range estimate, we assume that half the reduction is permanent. It is important to realize, however, that both a permanent drop in initiation and "mere" delay are associated with a reduction in lifetime cocaine consumption.

We draw from this association to translate estimates of reduction in marijuana initiation and the permanence of that reduction into estimated changes in cocaine use (see Figure S.2). For program effect, we take the bar in the middle as our preferred estimate and the largest and smallest bars as our high and low.

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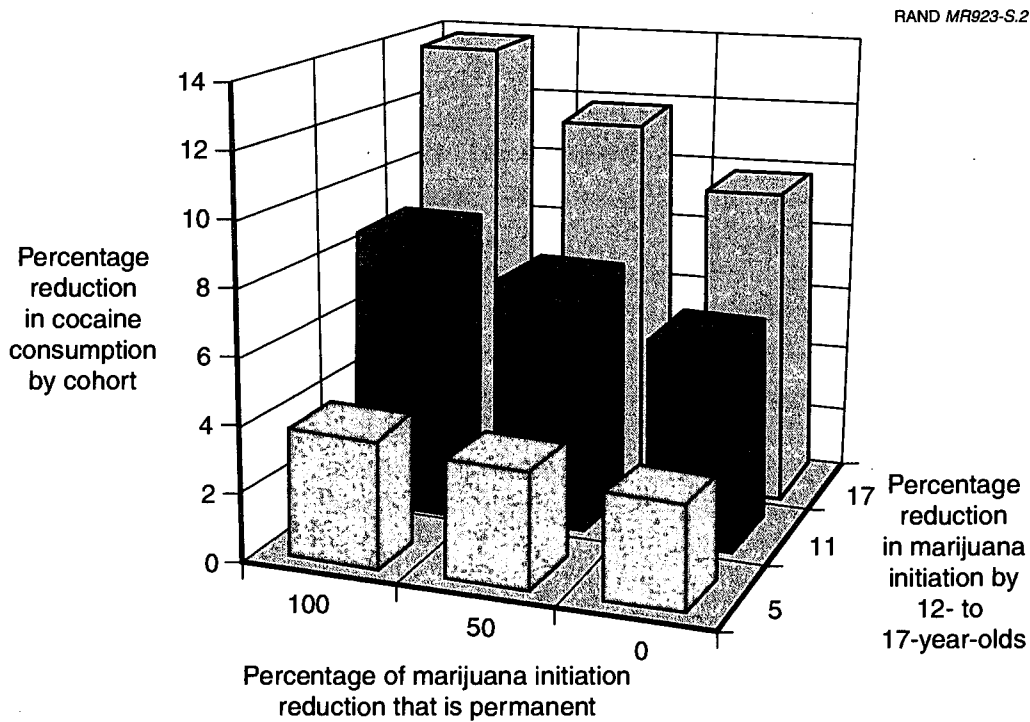
Figure S.2—Reduction in Cohort's Cocaine Consumption for Different
Assumed Values of Marijuana Initiation Reduction and Permanence

Discount Factor

The benefits of prevention accumulate over a number of years. It is a conventional assumption in economic analysis that people would rather have money (or benefits) sooner rather than later. We thus discount future pro-

Low	Middle	High
0.388	0.507	0.692

gram benefits at 4 percent per year (the rate we have used in our previous analyses of enforcement and treatment). We also use lower and higher rates (2 and 6 percent) and in each case convert the annual rates into aggregate discount factors.

Social Multiplier

A prevention program can affect persons *not* participating in it. Project ALERT and Life Skills are both based on the assumption that adolescents use drugs largely because of peer pressures and other social influences. In other

Low	Middle	High
1.0	2.0	2.9

words, if one person initiates use, he or she will influence others to do so. It follows, then, that if that person's use is delayed or prevented by a program, so will the use of the others who would have been influenced. The program effectiveness data cited above reflect these indirect effects on marijuana initiation, but not those pertaining to cocaine use, whose initiation usually occurs after individuals leave secondary school. The social multiplier is the total number of persons not initiating cocaine use per program participant not initiating cocaine use (if it is just the participant, the multiplier is 1). We theorize that social influence is inversely related to the number of heavy users, who offer examples of the ill effects of a drug. From the numbers of light and heavy users over time, we infer a social multiplier between 1.0 and 2.9. We take 2.0 as our "best estimate."

Market Multiplier

Another way a prevention program affects consumption by nonparticipants is through the cocaine market. Reduced consumption by program participants implies a reduction in the demand for cocaine. For any given level of enforce-

Low	Middle	High
1.0	1.3	2.0

ment, decreasing the demand for cocaine increases the amount of enforcement relative to the amount of use. This increase in enforcement pressure will tend to drive up cocaine's price. Even those who did not participate in the prevention program see the higher prices, so they are likely to use less cocaine. We use a model of the cocaine market to arrive at a "best estimate" of the market multiplier. To derive low and high estimates, we use a factor-by-factor analysis of uncertainty similar to that we use for this study as a whole.

Causation/Correlation Ratio

There are also ways in which the program effect given above may be *overestimated.* First, our calculation of reduction in cocaine use is based on a correlation of past cocaine use with past marijuana initiation age. If the preven-

Low	Middle	High
0.5	0.9	1.0

tion program is affecting some underlying propensity to use drugs, our strategy is valid. But if the prevention program only affects marijuana use, not whatever drives cocaine use, future reduction in marijuana initiation may imply nothing about future cocaine use. Evidence is particularly thin regarding this factor. What analysis we *can* do supports the middle estimate, and we believe the low and high estimates reflect a reasonable range. However, this is conjectural; we cannot "prove" that readers who prefer an even lower or higher estimate have beliefs that are inconsistent with data.

Scale-Up Factor

Second, it is not reasonable to assume that model programs implemented in resource-intensive demonstrations under the guidance of their designers will work as well when implemented on a large scale.

Low	Middle	High
0.5	0.6	0.7

There is virtually no basis for estimating the degradation in effectiveness accompanying scale-up. We take a conjecture from a previous study as our middle estimate. The low and high are arbitrarily and symmetrically selected around that middle estimate.

Overall Effectiveness (grams per participant)

Multiplying all the preceding factors together gives an overall effectiveness estimate. Our best estimate is that a model school-based prevention program will reduce future cocaine consumption by a net present value of about 3.8 grams per participating adolescent.

Low	Middle	High
1.01	3.77	8.82

To get the low and high estimates for overall effectiveness, we assumed for each factor that all values between the low and high factor estimates were equally likely and multiplied together values randomly drawn from the ranges given.[2] We did this thousands of times. The low estimate is the value lower than all but 5 percent of the results and the high is the value higher than all but 5 percent.

Combining all the factors allows us also to say something about the avenues of prevention's effectiveness. Only about a quarter of the reduction in cocaine consumption is in the form of reduced initiation by program participants—a typical objective of prevention. About 10 percent is in the form of reduced consumption by partici-

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[2]These random draws were made for all factors except the discount factor, which was held at 0.507, the value corresponding to a discount rate of 4 percent. This distinction was made because variation in discount rates reflects variation in decisionmakers' relative valuation of present and future events, not uncertainty about cocaine consumption or program performance.

pants who eventually initiate anyway. The rest, almost two-thirds, is from reduced consumption by nonparticipants—mostly through direct social influences, some through indirect market influences.

COST-EFFECTIVENESS

Cost-effectiveness is estimated by dividing the effectiveness estimates by the cost of the prevention program.

Program Cost (dollars per participant)

Program cost is straightforwardly calculated once it is defined. It is in defining program cost that people are likely to disagree. Everyone would agree that program cost should include at least the cost of the program materials

Low	Middle	High
$1.94	$67.12	$146.50

and of the time required to train the teacher. That minimal definition of cost results in our low estimate.

We believe, however, that the cost of the program should also include the lost learning opportunity involved in having students take the drug use prevention program instead of some academic subject. That opportunity cost is difficult to evaluate. However, states and school districts have apparently thought the value of academic learning was, on average, high enough to justify allocating funds to teacher salaries and to school buildings. For a middle estimate, we thus add to the low estimate the cost of the teacher's time in offering the program. To get a high estimate, we add to the middle estimate a per-student, per-class-session share of a typical school facilities budget. We prefer the high estimate for the reasons just given and because it is consistent with the approach taken in our analyses of enforcement and treatment (where we counted facility and other opportunity costs).

Cost-Effectiveness (kilograms of cocaine consumption averted per million dollars spent)

Dividing the effectiveness numbers by the cost numbers gives cost-effectiveness. We show here the estimates derived from our preferred high-cost estimate only. These numbers are of limited interest in themselves; they need to be compared with cost-effectiveness estimates for other cocaine control strategies (all of which were based on full accounting of costs). This comparison is done in Figure S.3 for the following programs:

Low	Middle	High
7 kg./$M	26 kg./$M	60 kg./$M

- Coca leaf eradication and seizures of coca base, cocaine paste, and cocaine in the source countries.

- Extended sentences for typical cocaine dealers.

- Interdicting cocaine en route from source countries.

- Expanding the mix of enforcement strategies used against typical dealers before the advent of mandatory sentencing.

- Current mandatory sentences of dealers prosecuted at the federal level.

- Expanding the mix of enforcement strategies used against dealers prosecuted at the federal level before the advent of mandatory sentencing.

- Treating heavy users with a program effective enough to keep the great majority off cocaine during treatment and cause a small minority to remain off heavy use after leaving treatment.

Our best estimate of prevention's cost-effectiveness suggests that model school-based prevention programs can reduce cocaine consumption as much per dollar spent as some enforcement programs do. The uncertainty is such, however, that low and high estimates would imply that prevention is not as cost-effective as *any* of the enforcement approaches shown or is more cost-effective than almost *all* of them. However, even if we give prevention the benefit of the

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| Cost-effectiveness at reducing cocaine consumption (kilograms averted per million program dollars spent) |
| Model prevention (low estimate) |
| Source country control |
| Longer sentences, typical dealers |
| Interdiction |
| Model prevention (middle estimate) |
| Domestic enforcement, typical dealers |
| Longer sentences, federal defendants |
| Model prevention (high estimate) |
| Federal enforcement |
| Treatment |

Figure S.3—Comparison of Prevention With Other Cocaine Control Programs

doubt and take the high estimate, it is not as cost-effective as treating heavy users—at this point in the cocaine epidemic (we will return to this temporal qualification below).

Narrowing the reasonable range of cost-effectiveness values for prevention would require more information about the various factors in the analysis. It is interesting in this regard that most of the research funded to date on prevention has been devoted to measuring one factor—program effectiveness narrowly considered. This factor accounts for only a small portion of the uncertainty.

AGGREGATE EFFECTS AND OTHER BENEFITS

Cost-effectiveness estimates are important but not the only informa-
tion required for allocating resources or formulating policy. For such
purposes, the total or aggregate effects of a program must also be
understood. What would be the costs and consequences of nation-
wide implementation of cutting-edge school-based drug prevention
programs?

It is easy to demonstrate that a full-scale national prevention pro-
gram is affordable. Using our preferred (high) cost estimate, putting
all adolescents through a program like the models we discuss here
would cost about $500 million to $600 million a year. That is about
1.5 percent of current national drug control spending.

We can also demonstrate that implementing such a program today
would not come close to eliminating cocaine use. We would expect
reductions of between 2 percent and 11 percent relative to a hypo-
thetical, no-prevention baseline.

A corollary of this is that prevention cannot substitute for enforce-
ment in a legalization regime. It has been argued that if the money
saved by not having to enforce drug prohibition were used to fund
drug prevention, the latter could offset any increase in use resulting
from relaxation of controls. However, this hope cannot be justified
even by our most optimistic estimates of prevention's effectiveness.

Furthermore, it would be years before the benefits of prevention
manifest in indicators of national use. With our middle estimate of
effectiveness, it would take a nationwide model prevention program
10 years to reduce the number of past-year cocaine users by 2.5 per-
cent relative to a scenario in which model programs were not im-
plemented. It would take 20 years to reduce the number of users by 5
percent and 40 years to achieve a 7.5 percent reduction.

Greater effects and cost-effectiveness might be possible in the early,
explosive stages of epidemic growth when preventing one person
from initiating could prevent a cascade of subsequent initiations.
Unfortunately, there is a considerable delay between implementa-
tion of a prevention program and when it begins to affect initiation
into and use of cocaine. But drug epidemics are difficult to predict
and, indeed, are not even always recognized as problematic until af-

ter the peak years of initiation have passed. It would thus be difficult for a reactive prevention strategy to affect those early growth stages of an epidemic. An alternative would be to run prevention continuously as insurance against future drug epidemics.

The limited effects of a nationwide program notwithstanding, even today the magnitude of the benefits prevention would bring through reduced cocaine use appears to justify the costs of prevention. We estimate that $2.40 in social costs would be averted for every dollar of resources used by a model prevention program. Again, however, this estimate is subject to substantial uncertainty: The actual cost averted per dollar could be as low as 60 cents or as high as $5.60.

Although we developed our methodology to estimate effects on cocaine use, it can also be used to obtain rougher estimates of effects on marijuana, cigarettes, and alcohol use. The estimated reductions are more modest: 0.6–3.4 percent for marijuana use, 0.1–1.2 percent for cigarette use, and 0.1–0.6 percent for drunkenness and heavy alcohol use. The less "deviant" the substance, the smaller the projected effect on use in percentage terms. However, cigarettes and heavy alcohol use are associated with such great problems that these reductions could generate considerable benefits. Our point estimates are that a dollar spent on prevention might avert $0.75 and $0.80 in social cost associated with cigarettes and alcohol, respectively, although again there is a wide range of uncertainty surrounding those point estimates. There may be still other benefits of prevention. Strengthening the resistance skills and perceived self-efficacy of adolescents may dissuade them from associating with gangs, getting pregnant, dropping out of school, and other behaviors potentially injurious to their health or economic prospects. But we do not estimate the magnitude of any of these benefits.

CONCLUSION

There is considerable uncertainty surrounding the magnitude of the effects of school-based drug prevention programs. However, a few things are clear. Nationwide implementation of a model program today is affordable, but it would not dramatically affect the course of drug use and the benefits would take years to accrue. On the other hand, the range of cost-effectiveness estimates we derive here for prevention is comparable to that previously derived for different en-

forcement interventions. Furthermore, implementing model pre-
vention programs seems to be justifiable in the sense that the bene-
fits produced would likely outweigh the costs of the resources used.

ACKNOWLEDGMENTS

INTRODUCTION

Drug problems are widely acknowledged to be serious, particularly among adolescents. This is even recognized by teenagers themselves. When a CBS News/*New York Times* poll asked 13- to 17-year-olds what they thought was the biggest problem facing people their age, 39 percent said drugs (Goodstein, 1998).

Various programs are being pursued in an effort to ameliorate America's drug problem. These programs range from interventions in source countries to border control, enforcement within U.S. borders, treatment of users, and efforts intended to prevent use. They currently cost about $40 billion a year across all levels of government in the United States. This is a lot of money, but drug consumption remains at levels many find unacceptable. To achieve better results, either the allocation of funds across the different drug control strategies needs to be reexamined or even more money is needed in total.

When public officials decide how to allocate funds across drug control strategies, they must take many things into account. Among these are

- moral values and preferences (does a user deserve punishment? is it better to reform a user or prevent a teenager from becoming one?)

- institutional and political considerations (how easy is it to get from the current array of organizations and assets bearing on drug control to some supposedly "better" array?)

32

- cost-effectiveness (what allocation of funds will achieve the objective at the lowest cost?).

AN ANALYSIS OF COSTS AND EFFECTIVENESS

Of the various factors that must be taken into account in deciding how to allocate public funds, we restrict ourselves in this book to issues of cost and effectiveness. It is important to keep in mind that these are not the only criteria on which judgments are to be made among alternative strategies, but they are important criteria: First, because public officials have a responsibility not to waste taxpayers' money. Second, people may differ irreconcilably over moral issues, and agencies may fight to protect their budgets and "turf," but cost and effectiveness are measurable and at least have the potential for engendering consensus.

This is not to say that cost-effectiveness analysis is entirely isolated from subjective considerations. "Effectiveness" implies making judgments about an objective—to answer the question, "effective at what?" Objectives vary with morals and interests. To a politician interested in getting elected, a "drug-free America" may have a nice ring, no matter how unrealistic it may be in practical terms. To a suburban parent, the most important objective may be ensuring that kids never start using drugs. To an inner-city resident, it may be stopping the flow of cocaine into the neighborhood. To someone who believes that a better-educated citizenry or a more competitive workforce or a lower national debt should be the top priority, all drug control objectives may be, at most, a means to an end.

In this book, we take reduction in U.S. drug consumption, and particularly cocaine consumption, as the objective of the nation's drug control efforts. We focus on cocaine because it is the most problematic illicit drug in the United States today.[1] We focus on the quantity consumed because it can be argued that it is the best single measure of the magnitude of the nation's drug problem (Rydell,

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[1] Most drug use prevention programs are delivered to school-aged children, and they follow those children at most through high school. Thus, the only drug-use outcomes measured are for alcohol, tobacco, and marijuana, since most people do not start using the so-called "hard" drugs until they are somewhat older. We infer reduction in cocaine consumption from measures of marijuana consumption by adolescents.

Caulkins, and Everingham, 1996).[2] We also address more briefly the overall social value of cocaine control—a value that must be established before drug control objectives can be compared analytically with other public policy goals.

Once an objective is chosen, a way must be found to express the achievements of diverse drug control programs in terms of that objective. Preventing a youth from initiating cocaine use, treating a heavy user, arresting a drug dealer—these must all be expressed in terms of drug consumption averted. Fortunately, we have been able to make considerable progress in comparing the cost-effectiveness of treatment programs and of enforcement programs of various kinds (Rydell and Everingham, 1994; Everingham and Rydell, 1994; Caulkins et al., 1997). That leaves prevention—the topic of this book.

Prevention is a timely issue, as there are signs that, after many years of policy emphasis on enforcement, prevention may be about to have its day. A Gallup poll conducted several years ago found that more people (93 percent of respondents) favored antidrug education in public schools as a way of dealing with the problem of drugs than any other strategy (Gallup, 1995, cited in Maguire and Pastore, 1996, p. 168).[3] When asked to choose the most effective among education, interdiction, conviction and punishment, and treatment, 40 percent chose the first of these. That was twice the percentage in a *Washington Post* survey conducted in 1990, when education came in third of the four (cited in Blendon and Young, 1998). Within the past year,

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[2]Reducing the amount consumed differs, of course, from reducing the number of users, and choosing the latter instead of the former could result in different policy choices. We focus on consumption because users vary greatly in the amount of cocaine they consume. In addition to the greater harm that heavy users bring to themselves and those close to them, they each account for more demand in the marketplace than light users do, more revenue to cocaine dealers, and potentially, more cocaine-market-related crime. Yet an analysis based on the number of users counts heavy and light users the same. An analysis based on consumption effectively assigns to each user a weight proportional to that user's consumption.

[3]This finding resulted from a question asking respondents to take their impressions of cost and effectiveness into account. Among strategies "strongly favored," prevention came in second of 16 alternatives, behind more severe criminal penalties (cited in Blendon and Young, 1998). When people were asked in what areas the country should increase its efforts or where money should be spent to fight the war on drugs, education and prevention were rated ahead of punishment and conviction and ahead of treatment, but behind interdiction (Gallup, 1995, cited in Maguire and Pastore, 1996, pp. 168–169).

Barry McCaffrey, Director of the Office of National Drug Control Policy, has declared that "Our Number 1 priority is prevention," and the federal government has begun spending $195 million per year on a new drug prevention media campaign; the latter was launched by a joint announcement from President Clinton and then–House Speaker Newt Gingrich.[4]

It is not, however, by accident or by extraordinary foresight into the evolution of the policy debate that we have left prevention to last. Because of conceptual difficulties and data shortages, prevention is the most difficult of the major strategies to analyze. Our prevention estimates are thus the most uncertain. To some extent this uncertainty is a consequence of the fact that most prevention programs are administered to preteens, while cocaine use does not normally start until the late teens and early twenties. Since it is not possible to observe directly all program costs and benefits, assessing prevention's cost-effectiveness necessarily involves the use of mathematical models. There are uncertainties at each step of the bridge the modeling builds between observed outcome measures and the bottom-line estimate of cost-effectiveness. However, as Bankes (1992) and Hodges (1991) point out, one does not necessarily need complete information on a system to model it usefully. Rather a model may illuminate system behavior and assist policy choices, with only partial knowledge.

Some readers will undoubtedly find disconcerting the uncertainty surrounding our estimates. They will say that, considering the importance and immediacy of the problem of drug consumption by youth, research presuming to contribute to its solution ought to come up with "an answer." We believe, however, that while our results are imprecise, they support some important, qualitative conclusions about the comparability of prevention and enforcement in terms of cost-effectiveness and about the affordability and impact of a national prevention program. We also present a simple, mathematical framework for guiding analysis of prevention's effectiveness. This framework clarifies the sources of uncertainty, can help guide

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[4]McCaffrey's statement was reported by Reuters on March 19, 1998, following his meeting with Mexican drug officials. For the Clinton-Gingrich announcement, see Bennet (1998). For more on the media campaign, see ONDCP (1998).

future research, and facilitates reestimating effectiveness as better information becomes available.

Of course, where estimates may take on any value across a substantial range, readers may be understandably suspicious that the analysts have chosen values to suit their prejudices. To avoid that, we have taken pains to ensure that our conceptual framework is clear and rigorously lays out what factors influence prevention's cost-effectiveness. Readers may then substitute different values for those factors than the ones we choose, if they so desire. We also take care throughout the book to show a reasonable range of values that might be substituted, along with the results of estimates that differ from our own.

Since cost-effectiveness is what can be accomplished per dollar (in other words, effectiveness divided by cost), we take the obvious approach of presenting the effectiveness analysis first (Chapter Two) and then factoring in cost (Chapter Three). In Chapter Four, we consider some additional benefits of prevention, particularly its effectiveness at reducing the consumption of drugs other than cocaine. We strive in these chapters to give a complete description and intuitive explanation of our analysis without bogging the reader down in details; those details are contained in the various appendixes.

BEYOND COST-EFFECTIVENESS

As suggested above, deciding whether to expand drug use prevention involves taking more factors into account than whether prevention yields greater results from the next million dollars spent than would other drug control alternatives. While some important factors can directly be addressed by an analysis of costs and effectiveness, others cannot.

For example, how does drug prevention stack up against other programs unrelated to drug use? After all, when new money becomes available—whether through unexpected surpluses or through a lower level of need for certain programs—there are many claimants for the new funds. Why should the money be spent on drug prevention—or enforcement or treatment—instead of on primary-grade class-size reduction, imprisonment of violent offenders, or subsidization of AIDS treatment? Comparing such different programs re-

quires comparing their outputs—reduced drug consumption, a better educated citizenry, reduced crime, a higher quality of life for persons with AIDS. One approach to comparing disparate outputs is to attempt some sort of dollar valuation of all of them. We do not attempt to do that here, but we take a step in that direction by converting the cocaine, alcohol, and smoking reduction benefits of drug prevention into dollar terms (see Chapter Four). At a minimum, that allows us to see whether a dollar's worth of benefits are realized from a dollar invested in drug use prevention.

Even if we restrict ourselves to reducing drug consumption as an objective, there is more to be drawn from considerations of cost and effectiveness than just cost-effectiveness. In particular, what is the total potential benefit of drug use prevention? If prevention were to be fully funded, what could be accomplished? We discuss such questions in Chapter Five, along with the implications of our analysis for future drug epidemics and for the debate over drug legalization.

AN ISSUE NOT YET EXAMINED

There have been hundreds of evaluations of drug use prevention programs, but they do not answer the questions addressed here. Most have sought (often unsuccessfully) to demonstrate whether a specific program results in a statistically significant reduction in drug use or has some other desirable outcome. Many try to show how to improve prevention programs, e.g., by finding merit in using peers to help run the program (Tobler, 1986; Ellickson and Bell, 1990a).

Such information is clearly valuable, but for a number of reasons, this literature does not help us determine how effective or cost-effective prevention is in general (Gorman, 1995). Many prevention evaluations do not report magnitudes of effects. Few if any try to make the connection between measured outcome variables and lifetime use. Few conduct long-term follow-up evaluations, and none of which we are aware has yet rigorously measured outcomes beyond the end of high school. Very few consider costs (Lipsey, 1997). Only a handful discuss cost-effectiveness (e.g., Connell, Turner, and Mason, 1985; Kim et al., 1995), and none provides a quantitative estimate of lifetime use averted per program dollar spent.

There have been meta-analyses—studies that have sought to pool and draw conclusions about prevention in general from published evaluations. For example, Tobler (1986, 1997) and Tobler and Stratton (1997) have done meta-analyses concluding, among other things, that interactive programs are superior and that effect sizes for smaller programs are larger than for bigger programs. However, meta-analysis has limitations for informing the sort of policy question we address here (Matt, 1997; Hedges, 1997). Another effort to take an overview was the National Structured Evaluation of Alcohol and Other Drug Abuse Prevention (CSAP, 1995). This study was the "structured evaluation of different approaches . . . to reduce alcohol and drug abuse" (P.L. 100-690) that was mandated by the Anti-Drug Abuse Act Amendments of 1988. It was intended to respond to policymakers' questions concerning the state of knowledge about prevention, but it did not address cost-effectiveness.

The net result of these past efforts is that many scholars seem favorably disposed toward prevention generally. But skeptics are beginning to raise concerns about some of the evaluations that have been done and whether indeed prevention is cost-effective (or effective, for that matter) (Gorman, 1995; Moskowitz, 1993; Lynskey, 1998). Hence, focusing on the global question of how cost-effective prevention is in general addresses an important topic not yet answered in research conducted to date. This and other ways this study differs from previous evaluations are summarized in Table 1.1.

OUR FOCUS

Drug prevention is appealing because it has the potential to avert a problem before it starts. This appeal has led to the development of numerous approaches to preventing drug use. Indeed, as Moore (1990) points out, even law enforcement can be thought of as prevention because it helps reduce the availability and increase the price of drugs; it also reinforces social norms against drug use. But even if we restrict the scope to programs that seek to persuade potential new users to stay away from drugs, there is still a very wide range of prevention programs.

Mass media campaigns, such as those by the Partnership for a Drug-Free America, are among the most prominent. Unfortunately, it has

Table 1.1

Differences Between This Study and Previous Ones

Characteristic	This Study	Typical Study
Drugs in focus	Mainly cocaine	Many substances
Consumption measure	Lifetime quantity used	Whether drug is tried during teen years
Effect characteristic of interest	Magnitude	Statistical significance
Economic information offered	Program cost, relative to effect size	Little or none
Population of interest	Program participants and nonparticipants	Program participants only
Programs compared	Prevention with other program types	One prevention program with another

been very difficult to rigorously assess their effects for a variety of methodological reasons, and we found insufficient evidence in the literature upon which we could build a cost-effectiveness analysis.

Community-based programs have been a popular intervention in the 1990s. Although Caulkins et al. (1994) find that such programs have promise, they have also been difficult to analyze rigorously.

Most methodologically strong, published evaluations address school-based programs for 9- to 15-year-olds (CSAP, 1995; White and Pitts, 1998). Multiple evaluations based on sound experimental or quasi-experimental design have found solid evidence of effects on risk factors and attitudes and on behaviors that are correlated with drug use (although some evaluations have not). Furthermore, reviews of past studies (for example, Falco, 1992), and reports on specific interventions (Ellickson and Bell, 1990a and 1990b; Ellickson, Bell, and Harrison, 1993; Botvin et al., 1990a; Botvin et al., 1990b; Botvin et al., 1995) show unambiguously that some school-based prevention programs can decrease drug use itself. However, the emphasis in prevention research has so far (appropriately) been on establishing statistically significant effects. Our goal is to translate, extend, and interpret those results in terms of the magnitude of effects on lifetime quantities of drugs consumed; to estimate the value of the resources the programs utilize; and from those two

factors, to compute a cost-effectiveness ratio and the costs and benefits of national implementation.

We do not, however, consider all school-based programs, or the average school-based program. Instead we focus on the performance of *best-practice* school-based prevention programs for two reasons. First, the results of some evaluations suggest that average school-based programs have little effect or none at all; where there is no effect, cost-effectiveness analysis is of no utility. The most widely used prevention program is DARE, Drug Abuse Resistance Education, which has received higher ratings than many other programs on the basis of the content of the program (Drug Strategies, 1996). However, it seems to have only modest effects on most of what it seeks to influence, and it has essentially no effect on marijuana use at follow-up (Tobler, 1997), which is a key indicator in our analysis of cocaine consumption. Second, prevention programs are diverse (CSAP, 1995), and there is no central source of information about the various programs, so it is not possible even to describe what average-practice prevention programs are let alone obtain data on their average performance.

It will be important to keep in mind that this focus on best-practice programs qualifies the degree to which the results of this analysis can be compared with those of our previous studies. In our assessments of the cost-effectiveness of enforcement and treatment programs, we used data for typical programs or data aggregated across all programs. Thus, when we say that prevention is more or less cost-effective at reducing cocaine consumption than these previously evaluated approaches, we are talking about only best-practice school-based prevention, examples of which we will describe in Chapter Two. Our conclusions are not necessarily applicable to other school-based programs or to non–school-based prevention strategies. Also, the comparison is with *typical* enforcement and treatment programs. We have not attempted to identify innovative enforcement or treatment approaches that might prove more cost-effective than typical programs.

EFFECTIVENESS AT REDUCING COCAINE CONSUMPTION

To estimate the effectiveness of school-based prevention programs at reducing cocaine consumption, we multiply together a set of eight factors. In the next chapter, we will divide the product by cost to get cost-effectiveness. The eight factors are as shown in Figure 2.1.

RAND MR923-2.1

Proportion of Persons Who Would Have Ever Used Cocaine
X
Lifetime Cocaine Consumption Per User
X
Percentage Reduction in Cocaine Use Due to Prevention Program
X
Discount Factor
X
Social Multiplier
X
Market Multiplier
X
Causation/Correlation Ratio
X
Scale-Up Factor

Figure 2.1—Factors Contributing to the Effectiveness of
Prevention at Reducing Cocaine Consumption

41

The first four of these factors determine the effect on cocaine consumption by members of a cohort receiving the prevention classes under "favorable conditions." Factors five and six are multipliers that recognize the ripple effect that running prevention programs with some individuals can have on other individuals' behavior. Factors seven and eight are qualifiers that adjust for ways that the first four factors might overstate prevention's effectiveness. At this point, the reader should not worry if the meaning of some of the factors is unclear; they are explained fully below. We list the factors here as a visual outline for this chapter.

In addition to providing a bottom-line numerical "answer," we hope to contribute to the understanding of prevention's cost-effectiveness in two ways: first, by constructing the analysis so that the factors that contribute to the answer are clear, and second, by explicitly giving low, "best," and high estimates for each factor at each step of the analysis. The "best" estimate is the one we prefer, and the low and high estimates bracket a range across which reasonable persons might disagree. That is, while we believe the scientific evidence most strongly supports the "best" estimate, some evidence supports all numbers in the range. In deference to that, we typically refer to our "best" estimates as "baseline" or "base" or "middle" estimates. We carry all estimates through the entire analysis. We hope that in structuring the analysis the way we have, the sources of uncertainty are obvious and their effect on the bottom line transparent.

EFFECT ON COHORT MEMBERS PARTICIPATING IN PROGRAM

We are interested in estimating the quantity or weight of cocaine consumption averted per million program dollars spent. From evaluations in the literature, data from surveys, and models of drug use, we can infer the degree to which prevention programs reduce cocaine use by participants—i.e., we can calculate a percentage reduction. These sources do not give us the effectiveness value we want: They do not tell us how much consumption is reduced overall. We need to figure that out by applying the inferred percentage decrease in cocaine consumption to the total usage. We discuss these factors in reverse order—total usage (number of users times amount per user), then percentage decrease in usage. Finally, we discuss the dis-

count rate, which ensures that future benefits and costs are valued appropriately, relative to current ones.

Proportion of Cohort That Would Otherwise Use Cocaine

To estimate total usage, we need to know how many people use cocaine and how much the typical user consumes. We tackle the first of these here, in terms of the fraction of people in a cohort (all the people born in the same year) who use cocaine at some point in their lives. We cannot, of course, determine that fraction with certainty for current and future cohorts. The best that we can do is look at the proportions for past cohorts and make reasonable extrapolations.

Figure 2.2 shows the proportion of people who reported in the 1991, 1992, or 1993 National Household Survey of Drug Abuse (NHSDA)[1] that they had ever used cocaine. The proportion ever using,[2] also known as "lifetime prevalence," varies by age. The proportion is low for people who are young in 1992 simply because the median age of cocaine initiation is a little over 20, so a survey of teens will not capture many eventual users. We would expect rates for those cohorts to rise in time as they age. The proportion is low for people who were over 45 in 1992 because they passed through the ages of peak initiation during a time period when cocaine was neither plentiful nor popular. Neither of those extremes provides the best basis for predicting proportions of future cohorts that will ever use cocaine.

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[1]For a description of the NHSDA, see SAMHSA (1995). Briefly, it is a large (now about 30,000 respondents), anonymous survey of drug use, drug problems, and related issues. The self-report and household orientation generate obvious strengths—e.g., its national sample and rich set of questions. The survey also has limitations: It excludes the homeless, whose use rates are probably higher than those of the housed population, and, although the survey is adjusted for nonresponse, it may nonetheless underestimate use by household members, who may underreport their consumption (see, e.g., GAO, 1993; Mieczkowski, 1996). We use the NHSDA in ways that take advantage of its strengths, but turn to other sources when its limitations would be problematic— e.g., for estimates of lifetime consumption (see Table 2.1).

[2]The data shown in the figure have been smoothed in two ways. First, we averaged three years of data for each age shown. Thus, for age 45, we averaged the percentage of 45-year-olds reporting they had ever used cocaine in the 1991, 1992, and 1993 surveys. Second, these average numbers were then averaged with those of the two immediately adjacent cohorts (i.e., a three-year moving average was calculated). Thus, to derive the percentage in the figure shown for 45-year-olds, the three-survey averages for 44-, 45-, and 46-year-olds were averaged.

RAND *MR923-2.2*
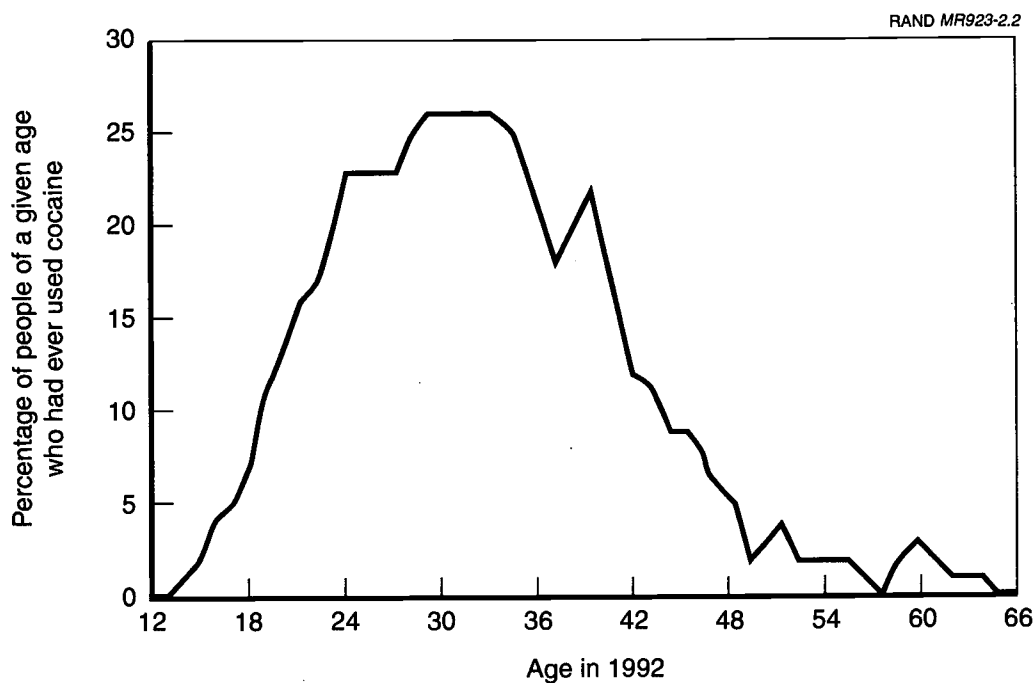
NOTE: Calculated from NHSDA data.

**Figure 2.2—Proportion of People in Cohorts Who Reported Having Ever
Used Cocaine (three-year moving average)**

It could be argued that we have already seen the worst of cocaine ini-
tiation, at least for a while, because the dangers of cocaine are now
widely recognized. The peak lifetime prevalence in Figure 2.2 is 26.4
percent (for those who were in their early thirties in 1992), so we take
our upper-bound estimate to be 27 percent.

For the lower-bound estimate we work from the fact that, in steady
state, if the number of people initiating cocaine use in a given year is
divided by the cohort size, the result is the proportion ever using co-
caine. Rates of drug use among youth and among the general popu-
lation declined steadily from 1979 to a "trough" in 1991 or 1992
(depending on which measure is used) and have risen since. One
scenario for a low estimate would envision that the low rates of
initiation in 1991 and 1992 are sustained indefinitely. Johnson et al.
(1996) report that there were 485,000 and 547,000 cocaine initiations
in 1991 and 1992, respectively. Dividing the average of these two

figures by an average birth cohort size of 3.75 million individuals yields 13.8 percent, so we take 13 percent as our lower estimate of baseline lifetime prevalence of cocaine use. Obviously this is not literally a bound. There is no theoretical reason why future baseline rates of cocaine initiation could not be zero—in which case, prevention would obviously have zero cost-effectiveness in terms of reducing cocaine use. But we believe the 13 percent figure is reasonably conservative. We take as our middle estimate 20 percent, the average of our low and high estimates.

Lifetime Consumption

Now, we want to be able to multiply the percentage of people ever using cocaine by the amount they use in a lifetime, on average. We found no estimate of that parameter in the literature. Nor is there any single best way to estimate its value. So we developed six alternative approaches, as summarized in Table 2.1. Details are contained in Appendix A.[3]

The simplest estimate comes from dividing total consumption over a period by the number of people who initiated cocaine use during that period. For the period 1962–1991 (years for which data are available from Rydell and Everingham, 1994), the result is 169 grams.

That estimate is almost certainly too low. Some people who initiated during that period have used and will go on to use more cocaine after 1991. Since the denominator is initiates between 1962 and 1991, a good case can be made for adding that post-1991 use. However, a smaller number of people using during that period initiated before 1962, and that use might be subtracted. Everingham and Rydell (1994) provide estimates of a number of relevant variables: the numbers of light and heavy cocaine users at the beginning and end of the period, the total cocaine use and number of initiates over the

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[3]All of the following estimates assume no effect to date of model school-based prevention programs. That is, theoretically they should be adjusted upwards to arrive at a baseline free of the effect of programs already in place. No such adjustment is necessary, however, because model programs are currently operating at only a tiny fraction of all schools, and other programs have been shown to be ineffective.

Table 2.1

Alternative Estimates of Average Lifetime Cocaine Consumption

Method	Estimate of Average Lifetime Consumption
Consumption over historical period, based on grams, unadjusted	169 grams
Consumption over historical period, based on grams, adjusted for use outside period	433 grams
Consumption over historical period, based on years of use, adjusted for use outside period	457 grams
Infinite-horizon Everingham & Rydell model[a]	405 grams
Finite-horizon Everingham & Rydell model[a]	292 grams
Steady-state assumption	532 grams

[a]Everingham and Rydell, 1994.

period, and the ratio of heavy to light use. From these, it is possible to calculate algebraically the adjustments for post-1991 use and use by pre-1962 initiates (see Appendix A). The adjustments result in the addition of 264 grams to the unadjusted 169, to yield a total lifetime consumption of 433 grams.

A different approach to estimating lifetime consumption is to multiply the amount a light cocaine user consumes per year of use (from Everingham and Rydell, 1994) by the number of years a typical cocaine consumer spends as a light user. The same is then done for heavy use (taking into account the probability that a typical user will turn to heavy use), and the two amounts are added together. Appendix A gives the methods for calculating years of light and heavy use and for making adjustments analogous to those made above for estimates based on grams of use. This approach yields a lifetime consumption estimate of 457 grams.

A fourth estimate (405 grams) can be derived from the Everingham and Rydell (1994) model of cocaine use. It probably overstates lifetime consumption because its Markovian structure ignores aging, so it projects probabilities of use into middle and old age that are too high. Truncating that model's projection of lifetime use at a time horizon consistent with that used in past studies gives the fifth estimate (292 grams).

The final estimate is methodologically the weakest because it is only valid if cocaine use is in steady state, which it is not. If use were in steady state, then lifetime consumption could be found by dividing national consumption in one year (291 metric tons in 1992 according to Rydell and Everingham, 1994) by initiation in that year (547,000 according to Johnson et al., 1996), yielding the final estimate of 532 grams.[4]

Considering the strengths and limitations of these approaches, we set our low, mid-range, and high estimates as follows. Given that 169 grams is almost certainly too low but that we have a 292-gram estimate that we think is unbiased, we set a low plausible estimate at 225 grams. We have three estimates falling into a fairly narrow band from 405 to 457 grams, so those must carry some weight in deciding on a mid-range "best" estimate. Thus, we set the mid-range estimate at 350 grams, roughly halfway between the no-known-bias 292-gram figure and the cluster of three estimates over 400 grams. Because the highest figure of 532 grams is methodologically the weakest, we set the high estimate just above the cluster of three at 475 grams.

Program Effect

The product of the first two factors shows how much cocaine the average young person would consume in a lifetime if he or she did not participate in a prevention program.[5] We now turn to the question of how much a prevention program can reduce this consumption. This is obviously a key factor in our analysis, so we devote more space to describing the analysis of this factor. We break the discussion into subsections addressing our use of risk factors in predicting reductions in cocaine use, the nature of the two programs upon which we base the "design" of our model program's effectiveness, and the numerical results.

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[4]Besides being based on a method that presumes steady state, this estimate is also suspect because consumption in 1992 was relatively high while initiation was relatively low, thus yielding an unrepresentatively high estimate.

[5]This is, of course, a statistical measure useful for calculating program effectiveness. Few people consume this average amount; most will never use cocaine.

Use of Risk Factors. We are interested in projecting prevention programs' effects on lifetime cocaine use. However, the literature describes benefits of school-based prevention programs in terms of changes in marijuana, cigarette, and alcohol use. Program evaluations typically do not follow subjects long enough to pick up effects on cocaine use, so the latter must be inferred from changes in the effects measured. In particular, we use marijuana initiation as a risk factor for eventual cocaine consumption; we rely on good evidence suggesting that marijuana-using adolescents are at higher risk of eventual cocaine use than non-marijuana-using adolescents. Data from prevention program experiments enable us to estimate how the program affects the age at which marijuana use starts. Data from the National Household Survey of Drug Abuse enable us to estimate how great a reduction in cocaine consumption is associated with a delay in, or complete prevention of, marijuana initiation. Aspects of this approach need to be explained both in general (the use of risk factors) and specifically (why focus on marijuana initiation and early use).

First, it is important to state clearly and unequivocally that this analytical strategy in no way presumes a causal relationship between the risk factor (marijuana use) and cocaine use. To argue that drug abuse prevention programs cut cocaine consumption, we must look upon marijuana, early cigarette, and early alcohol use as a signal of cocaine use, but we do not assume a causal "gateway" relationship exists.[6] What we do assume is that both early marijuana use and later cocaine use arise from the same propensities to use drugs and that prevention program effects are the result of changes in these propensities. Thus, we can take reduction in marijuana use as a measure of the change in drug use propensity. Therefore, marijuana use reduction can be employed to estimate reduction in cocaine use. No harm occurs to the analysis if the association of cocaine and marijuana use depends on propensity to use drugs, provided that the prevention programs affect that propensity as well as the tendency to use marijuana alone.

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[6]The gateway linkage is difficult to pin down (Yamaguchi and Kandel, 1984; DuPont, 1989; Ellickson, Hays, and Bell, 1992).

This proviso is important. If prevention programs merely treat a "symptom" (age of marijuana initiation), not the underlying "disease" (propensities that drive lifetime cocaine consumption), our approach would tend to overestimate prevention's cost-effectiveness. (Conversely, if prevention programs do a better job of treating the general drug disease than the specific marijuana symptoms, then our approach would underestimate prevention's effectiveness.)[7] We compensate for this possible overestimate through the causation-to-correlation ratio (see Figure 2.1)—the ratio of the true effect to the apparent effect suggested by the association in the NHSDA data. We will have more to say about that ratio below.

But why focus on marijuana use, instead of some other characteristic putting adolescents at higher risk of cocaine use? Given the evidence that some prevention programs can affect attitudes toward and knowledge about drugs without affecting drug-use behaviors (Botvin 1990), we choose to focus on a risk factor that reflects actual (or at least self-reported) behavior. Behavior that pertains directly to substance use has obvious merit, given that we are principally concerned with cocaine use. That narrows the field of risk factors to early use of alcohol, cigarettes, or marijuana. We choose marijuana for several reasons:

- Marijuana is an illegal drug. Although alcohol and tobacco are illegal for juveniles, we believe that willingness to use a drug whose recreational use is illegal for everyone has more bearing on lifetime use of illicit drugs than does precocious use of conventionally used substances.

- The correlations between cocaine use and adolescent marijuana use are stronger than the correlations between cocaine use and adolescent use of either alcohol or cigarettes (CASA, 1994, p. iii).

- There is some evidence, albeit thin, that the correlations between early alcohol and cigarette use and subsequent cocaine use are mediated through early marijuana use (Becker, 1997).

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[7]We do not mean to suggest by our characterizing marijuana use as a "symptom" that marijuana use is not itself worthy of prevention.

Figures 2.3 and 2.4 show the nature of the correlation between age of marijuana initiation and subsequent cocaine use. Figure 2.3 displays the extent to which people who initiate marijuana use at a later age have a smaller chance of ever using cocaine. Figure 2.4 shows that those who do initiate cocaine use tend to use less over their lifetime the later they started using marijuana. Again, total usage is probability of use (prevalence) multiplied by lifetime consumption per user, so the association of age of first marijuana use with cocaine consumption is the "product" of the effects shown in the two figures.

Some readers may be concerned about our use of the National Household Survey on Drug Abuse. The NHSDA misses or under-samples heavy drug users, and although respondents are surprisingly forthcoming about their status as drug users (e.g., whether they have used in the past twelve months), they substantially understate the quantities (weight) they consume. Because of these limitations, we

RAND MR923-2.3
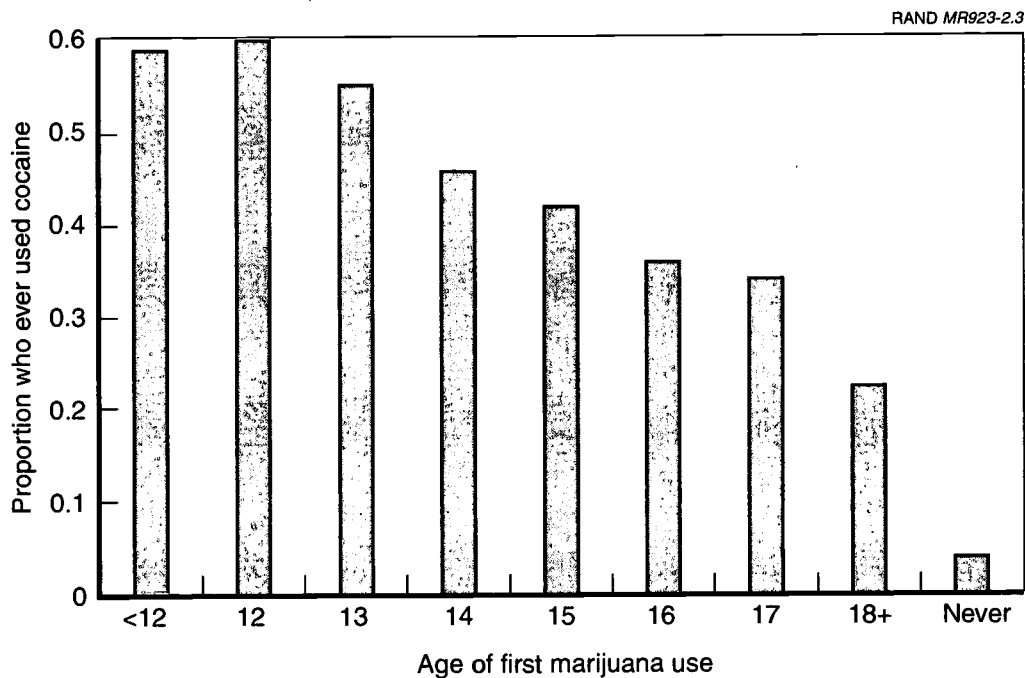
NOTE: Calculated from 1991–1993 NHSDA data.

Figure 2.3—Later Marijuana Initiation Is Associated With Reduced Risk of Ever Using Cocaine

RAND *MR923-2.4*

Relative frequency of reported lifetime instances of cocaine use by those ever using it

Age of first marijuana use
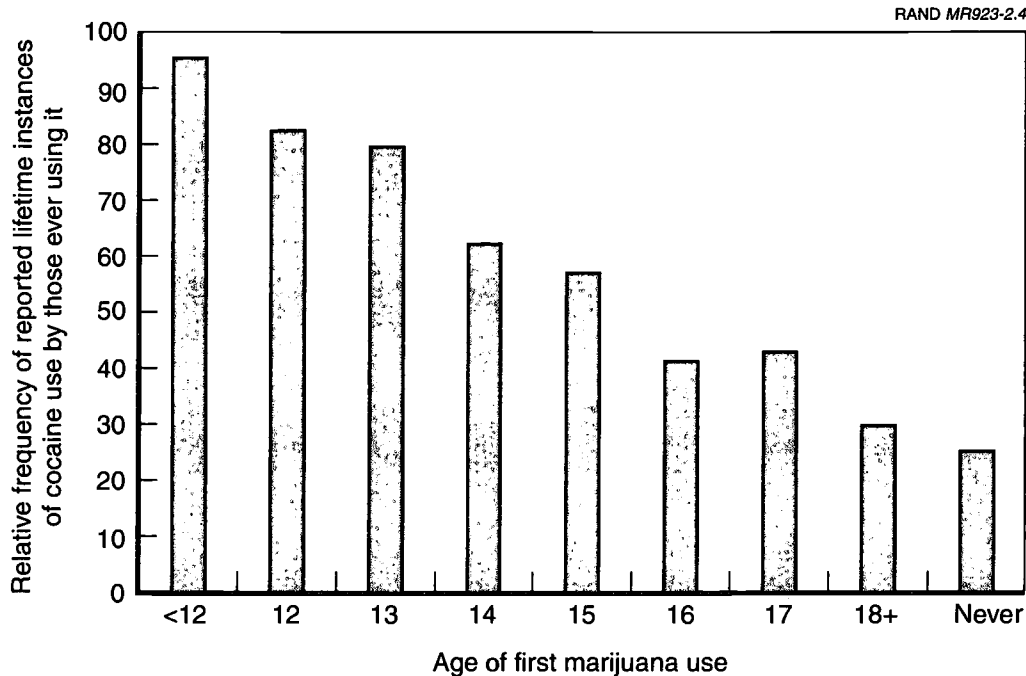
NOTE: Calculated from 1991–1993 NHSDA data.

**Figure 2.4—Later Marijuana Initiation Is Associated With Reduced
Consumption of Cocaine by Those Who Ever Use It**

do not use the NHSDA to estimate directly prevention's effect on the
quantity consumed. Instead, we use the NHSDA to estimate preven-
tion's proportionate (percentage) effect on consumption. We trans-
late that into a reduction in weight consumed by multiplying by the
average quantity consumed over the course of a use career which, as
we have discussed, is estimated from other sources.

Thus, we are not assuming that NHSDA respondents account for
most of their drug use or that respondents accurately represent the
cocaine-consuming population in terms of lifetime use. We are only
assuming that prevention has the same percentage impact on total
lifetime use as it does on use reported in the NHSDA,[8] in other
words, that prevention has the same proportionate effect on hidden

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[8]This position has something in common with the Kreitman (1986) hypothesis that
shifting heavy use can be done by shifting the entire distribution of use.

use as on observable use. (However, to the extent that the behavior of those not adequately represented in the NHSDA sample differs from the behavior of those in the sample, this assumption may be faulty.)

Best-Practice Prevention Programs. As discussed in Chapter One, we are not interested in the cost-effectiveness of the average or typical prevention program but in that of the "best-practice" program— not in what prevention *has been* doing but in what it *could* do. Neither are we interested in comparing the cost-effectiveness of one prevention program with another. We want to compare best-practice prevention with enforcement and treatment. Hence, we want to synthesize information about different prevention programs, not carry through the calculations separately with data on different programs.

In particular, we draw on evaluations of two best-practice school-based programs: the Life Skills Training Program and Project ALERT. These evaluations include control groups monitored in parallel with treatment groups. Their publications have been clear about program content and participants. And the programs evaluated are widely recognized as excellent. For example, Project ALERT is the only program that received "A" ratings in every category from the Drug Strategies review (1996). The Life Skills program has been demonstrated not only to have substantial effects but also to maintain those effects through the twelfth grade; further, it has been selected by the Blueprints Project led by Delbert Elliot of the University of Colorado as one of ten model violence reduction programs that are worthy of replication.

Life Skills. The Life Skills Training Program is delivered to junior high school students.[9] According to Botvin et al. (1995), it focuses on

> teaching information and skills for resisting social influences to use drugs and generic personal and social skills for increasing overall

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[9]Both model programs and thus our analysis focus on this age group. This is consistent with recent findings that the age-12-to-13 transition is marked by dramatic increases in access to drugs, use of drugs, and reliance on friends rather than parents for input to decisionmaking (CASA, 1998).

competence and promoting the development of characteristics as-
sociated with decreased risk of using drugs.

In this analysis, we incorporate data from a recent evaluation of Life
Skills (Botvin et al., 1995). Starting in 1985, Life Skills was imple-
mented in 56 suburban and rural schools in Syracuse, Albany, and
Long Island, New York. Program participants were 91 percent white.
Most of the students in the study were from two-parent families.
Twenty-two of the schools were assigned to the control group. Eigh-
teen were assigned to an experimental group in which instructor
training was provided through a formal workshop supplemented
with implementation feedback. Sixteen were assigned to an experi-
mental group in which training was provided by videotape and no
implementation feedback. The curriculum was taught to students in
the treatment groups during 15 classes in seventh grade, with 10 ad-
ditional sessions in eighth grade, and 5 in ninth.

Measures of drug use in twelfth grade were as much as one-third
lower among students in the experimental groups as among those in
the control group. (Even greater reductions were observed in a
"high-fidelity" subset of the experimental groups. However, because
of concern that schools capable of delivering the program with high
fidelity might differ along unmeasured dimensions from schools that
could not (Gorman, 1998), we base our estimates on all students who
were in the program.)

Project ALERT. Project ALERT was originally developed with support
from the Conrad N. Hilton Foundation. The curriculum is based on
the social influence model. It helps students develop reasons not to
use drugs, identify and counter pressures to use them, understand
that most people do not use drugs, and recognize the benefits of re-
sistance (Ellickson and Bell, 1990a, 1990b; Ellickson, Bell, and Harri-
son, 1993; and Ellickson, Bell, and McGuigan, 1993).

Our data are based upon a trial conducted from 1983 to 1986. Thirty
schools in California and Oregon participated. They collectively
cover urban, suburban, and rural settings. Nine have a minority
population of 50 percent or more, and 18 are in neighborhoods with
household incomes below the state median. Ten schools were moni-
tored as controls. In the 20 treatment schools, an eight-session cur-

riculum was given to seventh-graders. Then, the following year, three booster sessions were given to the students as eighth-graders.

Project ALERT was successful in decreasing the level of marijuana use. For students who had not tried marijuana or cigarettes at baseline, it curbed marijuana initiation during junior high years by one-third and reduced the number of current users by one-half (Ellickson and Bell, 1990b, p. 16). The program also reduced cigarette use. However, by the end of high school, the treatment program's effect on drug use had eroded. In particular, its earlier positive effect on marijuana use had disappeared (Ellickson, Bell, and McGuigan, 1993, p. 859). This lack of permanence, however, does not mean the prevention program "did not work." Delayed initiation of drug use and the resulting reduced drug use during early adolescence yield immediate benefits (fewer accidents, better health). Delayed initiation into marijuana use is also correlated with reduced probabilities of initiating cocaine and, for initiators, reduced lifetime cocaine consumption. In other words, the measure of interest in this book can be affected by a prevention program even if the program's effects on other drugs such as marijuana disappear by the end of high school. Moreover, it is possible that just as the eighth grade booster sessions improved immediate program performance, high school sessions could improve long-term performance. Evaluation of a version of Project ALERT ("ALERT Plus") adapted for high school is now under way.

Estimates of Effectiveness. Both Project ALERT and the Life Skills program provide evidence on decreases in marijuana initiation during junior and senior high school, but most of it is indirect. The one direct estimate, from the evaluation of Project ALERT through eighth grade, is of a 4.9 percent reduction.[10] We prefer not to use this estimate alone, because it is for a program that extends only into eighth grade. The Life Skills program extended further, but the evidence in that program is indirect, derived from reduced levels of marijuana

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[10]Among students who had tried neither marijuana nor cigarettes as of the program's start, 8.3 percent of participants had smoked marijuana as of a year later—a percentage a third less than that in the control group. But among students who had tried cigarettes (but not marijuana) as of the program's start, the fraction of the participant group ever having used marijuana by the following year was 12 percent higher. The weighted average of these two differences (the minus one-third and the plus 12 percent) yields the 4.9 percent reduction used here.

use (e.g., percentage of students using marijuana monthly or weekly) from which reductions in initiation must be inferred.[11] Since we use these indirect Life Skills measures, we also take into account analogous indirect Project ALERT measures. The average across all of these measures from Project ALERT and Life Skills is a 16.9 percent reduction.[12] We round these figures off to 5 percent and 17 percent reductions and use those as our low and high estimates, respectively. We take the average of the two (11 percent) as our base estimate. (Details are in Appendix B.)

The next question concerns the permanence of these reductions. Neither Project ALERT nor the Life Skills program provides evidence on what happens after high school. Suppose we know from the data that someone who would have initiated in eighth grade hasn't by the time of the senior-year follow-up survey. We still do not know what happens to that person after that last follow-up survey. The worst thing that could happen is that the next day the person starts using marijuana (zero permanence of effect beyond the follow-up survey). The best thing that could happen is that the person never uses marijuana (100 percent permanence).

Our analysis takes as its middle estimate the intermediate position of 50 percent permanency, and uses the extreme possibilities in the low and high estimates. (For purposes of calculating timing of use, as we will do below, those whose initiation is merely delayed are assumed to initiate between the ages of 18 and 21, not literally the day after the follow-up survey.)

Figure 2.5 depicts graphically our middle assumption about how a model school-based prevention program affects marijuana initiation. The 11 percent reduction is assumed to apply across ages 12 through 17. The half of that total reduction that is assumed not to be

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[11]Of six indirect drug use reduction measures in common between Project ALERT (eighth grade) and Life Skills (twelfth grade), five were greater in Life Skills. These data support the logic offered here that a model program extending into the high-school years would result in a greater drop in marijuana initiation than that found in Project ALERT.

[12]It could be argued that because we are searching for an upper bound, we should average the measures from the more successful study or even pick the best single measure. However, because these measures are indirect, we think it makes more sense to consider them all in inferring initiation.
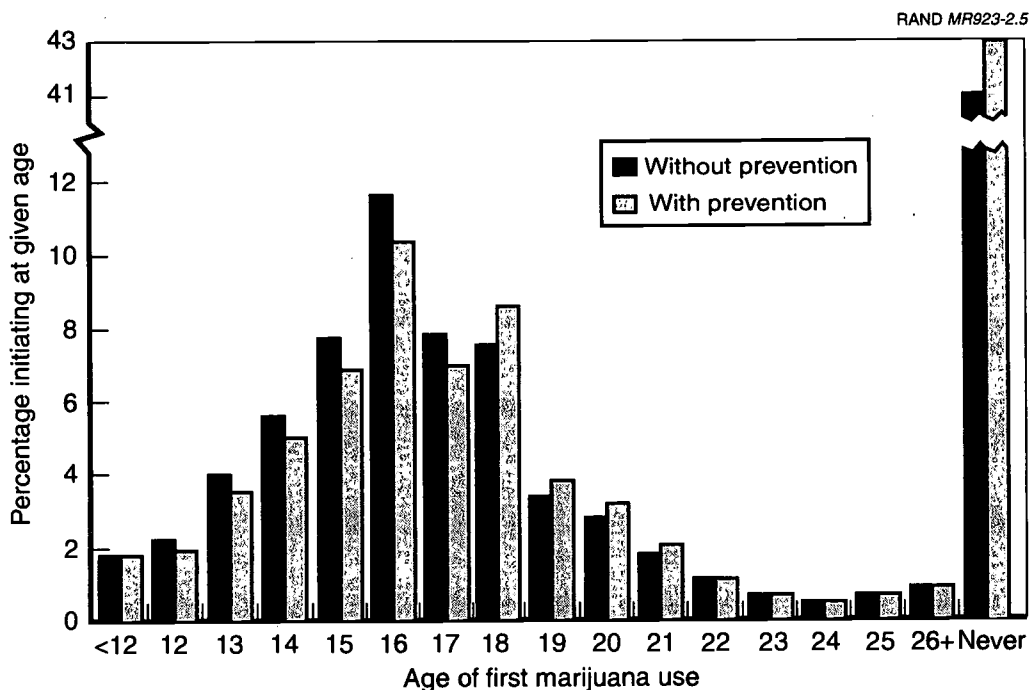
Figure 2.5—Proportion of Cohort First Using Marijuana at Different Ages
(assumed distribution of middle estimate)

permanent is then added back during ages 18 through 21, distributed proportionately according to the "without prevention" values for those ages. The other half is added to the "never" category. The overall effect is modest but definitely noticeable.

We combine these estimates with the NHSDA data on cocaine use as a function of age of marijuana initiation in Figures 2.3 and 2.4 to estimate the proportionate effect on lifetime cocaine use. We have a low, middle, and high estimate of both the reduction in marijuana initiation and its permanence. Combining these yields nine estimates of the effect on cocaine use (see Figure 2.6). (Details are in Appendix C.)

The highest estimate of prevention's effect on a cohort's lifetime cocaine consumption (13.6 percent) occurs with maximum reduction in marijuana initiation, all of which is assumed to be permanent. The lowest estimate of 2.9 percent occurs with minimum reduction

RAND MR923-2.6
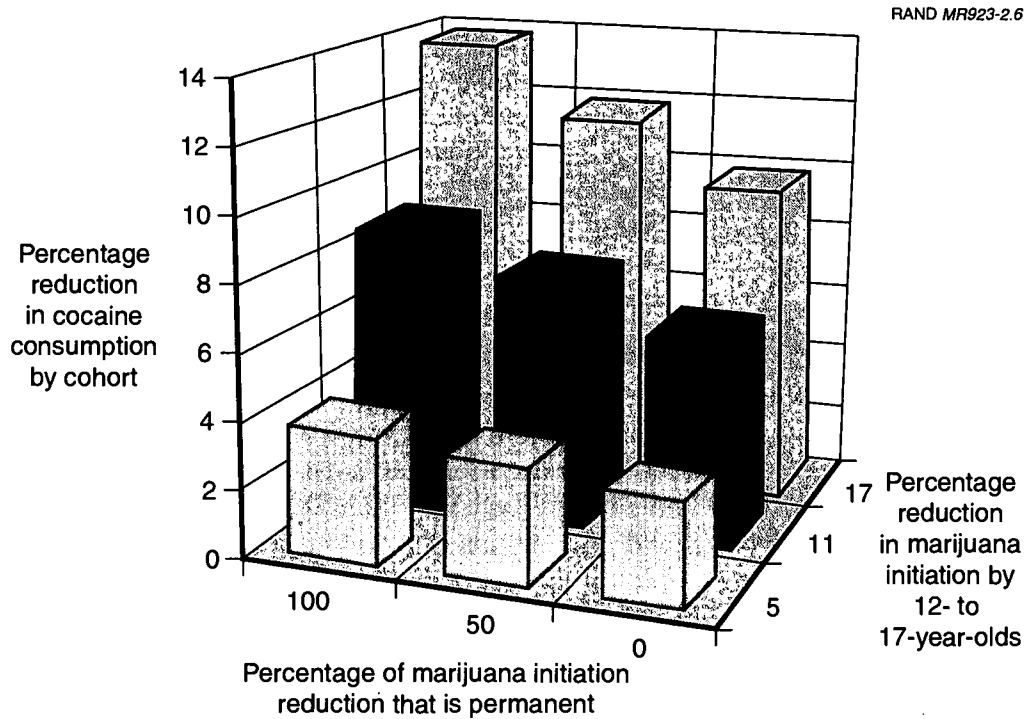
Figure 2.6—Reduction in Cohort's Cocaine Consumption for Different
Assumed Values of Marijuana Initiation Reduction and Permanence

in marijuana initiation, none of which is assumed to be permanent. The middle estimate, resulting from the middle estimates of both dimensions, is 7.6 percent. We take these as our high, low, and middle estimates of prevention's effect on lifetime cocaine use by people in the program (prior to discounting and adjustment through qualifiers).

Note that the estimated decrease in cocaine consumption is considerably more sensitive to the amount of reduction in marijuana initiation than it is to the permanence of that reduction. In particular, the figures in the 0 percent permanent row are about 84 percent of those in the middle row and 72 percent of those in the 100 percent permanent row. This is fortunate, because we have no experimental evidence on the permanence parameter. It also has important implications for evaluations of prevention programs. A program that "only" delays marijuana initiation and does not affect eventual probability of use can be almost as effective at reducing lifetime cocaine

consumption as one whose reductions in marijuana use are perma-
nent.[13]

Discount Factor

Because the drug use averted by a prevention program would have
occurred many years after the resources were spent running the pro-
gram, it is important to consider the time value of those benefits in
estimating cost-effectiveness. Time value is typically taken into ac-
count by discounting future values at a constant rate per year. Dis-
counting costs is a familiar technique in economic analysis. It is
commonly understood that, even with no inflation, it is more costly
to spend a dollar now than in the future. The dollar spent later can
be invested meanwhile and earn interest, thus partially offsetting its
eventual loss. There is not always a similar automatic recognition of
the time value of benefits—that they must also be discounted. How-
ever, Keeler and Cretin (1982) have demonstrated rigorously that
perverse results can occur if one does not discount benefits and costs
at the same rate (see also Gold et al., 1996, p. 309).[14]

Discounting appears in our eight-factor model as the discounted
present value of lifetime cocaine consumption divided by the un-

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[13]This is not license to conclude that every prevention program reducing marijuana
use is effective even if it has no permanent effects. Here, our notion of delay is delay
until ages 18–21. Delaying marijuana initiation only by a year or two would have a
smaller effect. For example, suppose a program reduced marijuana initiation by 11
percent in seventh and eighth grades, and all of that reduction took the form of delay-
ing initiation until ninth grade. Our model estimates that this program would have
only 15 percent as great an effect on lifetime discounted cocaine use as would an 11
percent reduction throughout junior and senior high school, with 50 percent of that
reduction being permanent.

[14]Keeler and Cretin's full argument is somewhat mathematical, but a simple example
makes the necessity of discounting benefits clear. Suppose there are two plans: Plan
A costs $30 million this year and reduces cocaine consumption by 3 metric tons this
year. Plan B costs $29.9 million this year, but does not deliver the 3 metric tons of con-
sumption reduction for 10 years. If the benefit of cocaine consumption is not dis-
counted, Plan B would appear to be more cost-effective. However, by investing $20.2
million this year so that it grows to $30 million in 10 years we would get enough money
to implement Plan A then. Plan A has been transformed into one that delivers the
same benefit as Plan B for two-thirds the cost. Clearly, Plan A is more cost-effective
than Plan B, but unless benefits are discounted B would have been judged to be more
cost-effective.

discounted consumption. We call this ratio of discounted to undis-counted consumption the discount factor.

Because lifetime cocaine consumption varies by age of marijuana initiation, the discount factor is actually the weighted average of a series of discount factors specific to each marijuana initiation age. The weights are in proportion to the fraction of total cocaine con-sumption represented by each group of users having the same mari-juana initiation age.

We do not know how consumption varies over a cocaine use career. Thus, to represent consumption, we rely on number of years of use. We know from NHSDA data how long the average cocaine consumer with a given marijuana initiation age uses cocaine. Some error is likely to result from calculating the discount factor from years of use instead of from the profile of quantity consumed over a career, but that error is likely to be small compared to the other uncertainties in our analysis.

We calculate the discounted and undiscounted number of years of use for each age of cocaine initiation. From these and the relation-ship between the ages of marijuana and cocaine initiates, we calcu-late discount factors for each age of marijuana initiation. These factors are then combined into an aggregate average discount factor in a manner that reflects the amount of cocaine consumption by age of marijuana initiation. We use a 4 percent discount rate because it is typical of the rate used in analyses relating to social welfare and it is the rate we have used in previous analyses of drug control strategies and thus facilitates comparability. We discount to age 12 because that corresponds to the seventh-grade start of the two best-practice prevention programs we use as models.

Note that we calculate the discount factor on the basis of lifetime co-caine consumption, without considering prevention. We will be ap-plying the factor, however, to the reduction in cocaine consumption experienced by prevention program participants, as calculated through the three preceding factors in our paradigm. Should we be using a factor that accounts for prevention? Prevention affects the time profile of cocaine use, and thus affects discounting, in two competing ways. People who start marijuana use later are likely to start cocaine use later; hence, the net present value of their years of

use for any given duration of use will be lower because it started later. However, people who initiate marijuana use later are less likely to progress to heavy cocaine use, and individuals who use cocaine less heavily tend to have shorter cocaine careers. For these people, there is thus a shorter time lag between year of cocaine initiation and the bulk of their cocaine use. Apparently, these two factors more or less offset each other because our estimates of the overall discount factors with and without prevention are fairly close. We get 0.507 without considering prevention, and 0.506 if we take prevention into account with our middle estimate of effectiveness. These variations in discounting due to variations in the effectiveness of prevention are incorporated into the third factor of our eight-factor model, the program effectiveness factor. The fourth factor is defined simply as the discount factor without prevention.

The overall factor of 0.507 is well below 1, so it is an important component in the overall calculation of cost-effectiveness. Although we prefer the 4 percent discount rate, other analysts might reasonably choose different rates, so we calculate an overall factor for 6 percent and 2 percent rates, also. These yield our low and high discount factor values of 0.388 and 0.692, respectively. (The higher the rate, the lower the discount factor.)

We treat these variant factors differently from the way in which we treat variant consumption estimates, program effectiveness values, etc. Most of the uncertainty concerning discount rates and, hence, discount factors is definitional. It does not arise primarily because we do not know what the market rate of return will be in future years, but rather because different people have different beliefs concerning what is the appropriate basis for defining the social discount rate. Therefore, we present the low, medium, and high discount factors in our eight-factor model because different people reading this report will have different opinions about what the social discount rate should be. However, when we compare prevention's cost-effectiveness with that of other drug control programs, we always use the middle discount factor. We do this because the studies of those other programs used a 4 percent discount rate.

It would be an interesting exercise to run the evaluation models for all the drug control programs for different values of the discount rate. Presumably the lower the discount rate, the better prevention would

look relative to treatment and especially enforcement. However, that is beyond the scope of this project; we simply compare prevention's effectiveness to the published estimates for the other programs.

Summary

Eight factors are a lot to keep track of, so it is worth pausing to summarize where we now stand. We have discussed the first four factors, which together determine the estimated effect of a prevention program on the present value of lifetime use by individuals in the program. (See Table 2.2.) There are two reasons why the effectiveness estimates derived from Table 2.2 may be overestimates, and to account for those, we will multiply them by two qualifiers. Before estimating the qualifiers, however, we will consider two effect multipliers—factors five and six.

MULTIPLIERS

Factors five and six consider the indirect effect of a prevention program on people who were not actually in the program. Such effects can occur either through social or market interactions. These interactions work as multipliers, that is, they increase a prevention program's effect on total cocaine consumption.

Social Multiplier

To the extent that initiation into drug use is a contagious or epidemic phenomenon, preventing one person from initiating may reduce

Table 2.2

First Four Factors of the Eight-Factor Model

Factor	Low Estimate	Middle Estimate	High Estimate
Proportion of persons who ever use cocaine	0.13	0.20	0.27
Lifetime consumption per user (grams)	225	350	475
Proportion reduction due to program	0.029	0.076	0.136
Discount factor	0.388	0.507	0.692

future consumption by more than that one individual would have consumed. The first person's initiation might, for example, lead through various personal interactions to, ultimately, two other people initiating. Then the expected amount of consumption averted by preventing the first initiation would be about three times the average lifetime consumption of any one individual. We define the total number of initiations prevented per primary initiation to be the "social multiplier." In the example just mentioned, its value would be three.

Appendix D estimates the magnitude of this social multiplier; the intuition behind the multiplier and our conclusions about its magnitude are given here. If, as appears to be the case, most cocaine users are introduced to the drug by a friend or family member, then, all other things equal, the more current users there are, the higher initiation should be. Light users may be the most dynamic proselytizers because, on average, they are relatively recent converts to drug use themselves and may not yet have experienced many adverse consequences of their use.

Over time, drugs create problems for users, and the drugs acquire a reputation for being dangerous. There is no direct measure of either drug reputation or problematic use, but generally heavy users are more likely to manifest adverse outcomes associated with their use than are light users. Hence, all other things equal, the greater the "history" of heavy use, the lower one would expect initiation to be. The word "history" is used to connote a cumulative process; a reputation is acquired over time. But bad reputations are also not immortal. Musto (1987) hypothesizes that cycles of drug use arise when the current generation of youth no longer remembers the adverse experiences of its forebears.

The basic outlines of how initiation depends on other variables are clear: More light users promote initiation, and more heavy users or a greater accumulated experience with heavy use deters it. What are not clear are the details—how these effects net out, what specific metrics should be used, what mathematical forms the relations should take, and so on. It is not possible to sort this out experimentally, and relevant historical data are scarce. Hence, the simplified models on which we base the social multiplier may well be wrong both in detail (i.e., in their parameter values) and in general (i.e., in

their functional form). Nevertheless, they represent the best esti-mates that can be made at this time; however tenuous, they are probably better than ignoring the social multiplier altogether.

The social multiplier depends on the number of light and heavy drug users, and the numbers of light and heavy users vary over time, so the social multiplier varies as well. Hence, we want to understand both what the social multiplier was in our base year (1992) and how it might have varied, because we want to know the implications for the future. Our analysis suggests the following:

- The magnitude of the social multiplier in 1992 is quite robust with respect to alternative functional forms and parameter val-ues. It is almost always between 1.0 and 2.9. Hence, we take our low, base, and high estimates to be 1.0, 2.0, and 2.9, respectively.

- It is not possible to estimate with any degree of precision the magnitude of the social multiplier in the early years of the co-caine epidemic (the early 1960s). Various functional forms result in substantially different predictions of the social multiplier's magnitude at that time. Even more important, the estimates de-pend strongly on how many heavy users there were then. Since that parameter is largely unknowable, this limits the precision with which the multiplier can be estimated for those early years.

- Nonetheless, all of the functional forms and all of the parameter-izations imply that the social multiplier was substantially larger in those years. What is not clear is whether it was merely much larger or if it was enormously larger. The implications of this are discussed further in Appendix E, which addresses how the cost-effectiveness of prevention likely varies over the course of a drug epidemic.

There is an additional limitation on the spread of an epidemic that we have not accounted for. Besides being limited by bad examples from the growing heavy-user population, initiation should also be limited by a declining number of nonusers susceptible to it.[15] Many persons will not use a newly popular drug even without the deterrent effect of bad examples; some of these persons find drug use distaste-

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[15]For a parallel argument on delinquency, see Weatherburn and Lind (1997).

ful or are philosophically opposed to it, while others are unconnected socially to that portion of the population through which the epidemic of use is spreading. Among the susceptible population, the earliest users will potentially be able to "convert" many others, as hardly anyone they know is already using the drug. Later users will find that more of their acquaintances started before they did. Thus, the social multiplier should decrease with time.

This is an additional argument in favor of a low estimate of 1.0 for the social multiplier at this late stage in the cocaine epidemic. It also provides a reason for believing that the social multiplier is closer to the low end of our range than to the high end. Finally, it could mean that in the early stages of the epidemic, the multiplier may be more appropriately characterized as "enormously larger" than "much larger." Clearly, this presents an interesting avenue for further modeling efforts to follow. However, considering the very uncertain nature of the social-multiplier analysis and the fact that no more than a quarter of any cohort has tried cocaine, great weight should not be ascribed to the omission of the self-limiting effect.

Market Multiplier

The theory of the market multiplier is that when prevention (or treatment) reduces the demand for cocaine, existing enforcement resources get concentrated on a smaller market volume, increasing their effectiveness (Kleiman, 1993). By applying this multiplier to the estimated effect of a prevention program, we account for the enhanced productivity of a given level of enforcement resources resulting from the interaction between the prevention and enforcement programs. Since the increased enforcement productivity is caused by the prevention program, it is appropriate to consider it a prevention program benefit.

Using the market model constructed by Caulkins et al. (1997), we estimate that the most likely value of the market multiplier is 1.38 (see Appendix F). The interaction with enforcement increases prevention's effectiveness by about a third.

The expression of the market multiplier in Caulkins et al. is a function of four parameters. We repeatedly varied each of these four parameters randomly over an assumed distribution suggested by

Caulkins et al. (that is, we performed a Monte Carlo simulation) to determine the likelihood that the multiplier would take different values. The 2.5th and 97.5th percentiles of the resulting distribution of multiplier values were 1.08 and 2.06, respectively.

These values, however, assume that reduced cocaine demand does not lead to reduced law enforcement effort against the drug. This is probably true for marginal changes in demand over the short run (months), but false over the long run (years). Redirection of law enforcement effort toward other drugs would reduce or eliminate the market effect (as far as cocaine control is concerned), driving the market multiplier closer to 1.[16] We have no idea how much closer to 1, so we simply round the low, mid-range, and high values down slightly to 1, 1.3, and 2. This at least allows for the possibility that there is no multiplier effect through the market.[17]

QUALIFIERS

Our estimates of all the factors above rest on an empirical basis. For some of the factors that basis is relatively strong, for others it is weaker, but in all cases we view our low and high estimates as bounding the range of plausible values. Absent some significant empirical evidence that we have overlooked, we do not think values outside these ranges are defensible.

There are two more factors, however, in our eight-factor model concerning which we have next to no empirical evidence. They are no less important; they are simply less studied in the literature. We marshal what evidence we can find concerning their values and report what we subjectively think are reasonable low, base, and high values. However, if a reader were to challenge these values, we would have no real basis for disputing his or her opinion. We refer to these last two factors as "qualifiers." They both have the effect of re-

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[16]Redirection to control of other drugs, however, could result in "bonus" reductions in consumption of those drugs that could be considered a benefit of cocaine prevention. Of course, this does not affect the calculation of cost-effectiveness at reducing cocaine consumption.

[17]The possibility of reduced law enforcement effort with reduced demand was brought to our attention by Weatherburn (1998).

ducing the product of the first four factors, that is, the reduction in cocaine consumption by the participants in a prevention program.

The need for the first qualifier arises from the correlation between age of marijuana initiation and lifetime cocaine consumption. Since we have data on the effect of prevention programs on marijuana initiation but not on cocaine consumption, this correlation is central to our estimate of the effectiveness of prevention on cocaine use. But it is only a correlation; the relationship between age of marijuana initiation and lifetime cocaine consumption may only be coincidental and not reflective of some common underlying causal factor.[18] It may instead reflect some demographic or socioeconomic variable that coincidentally plays out as a difference in cocaine consumption but is unrelated to it (see Appendix G for an example). If so, postponing marijuana initiation may have no effect on cocaine consumption.

Ideally, analysts would investigate this possibility by replicating the cross-tabulations shown in Figures 2.3 and 2.4 for homogeneous subpopulations. We performed such calculations (Appendix G) for the variables in the NHSDA that seem potentially relevant (gender; race; ethnicity of neighborhood; age of first cigarette use, first daily smoking, and first monthly drinking; income and housing characteristics of census tract; and receipt of welfare or food stamps). By and large we see little evidence from these analyses that our aggregate analysis was unduly optimistic because it failed to distinguish important subpopulations. For all but one of the variables we examined, the value of the qualifier fell between 0.89 and 1.00. That is, the unqualified prevention program effectiveness would have to be reduced by at most 11 percent to eliminate the influence of such sources of heterogeneity. (The outlier was in the range of 0.76 to 0.86).

This is of little reassurance, though, if the important forms of heterogeneity are in personality characteristics, not gender, race, ethnicity, or socioeconomic characteristics. Furthermore, the NHSDA mea-

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[18]If an analyst wished to demonstrate that a relationship between marijuana initiation and cocaine consumption was rooted in a common cause, an experimental evaluation would be required. In this evaluation, a model prevention program would have to be implemented in which marijuana initiation rate varied and other factors were held constant. Such an experiment would be (at the least) impractical.

sures have significant limitations even for the attributes that they appear to address.

With these complications in mind, we take as our low, base, and high estimates of this qualifier 0.5, 0.9, and 1.0. Again, those values should be viewed as our opinions, not numbers that are in any sense "derived" from empirical evidence.[19]

The second qualifier accounts for possible degradation in effectiveness if the experimental programs upon which our effectiveness numbers are based were scaled up to the national level. Such scale-up problems can occur for a variety of reasons and are not unique to drug prevention, but the literature (reviewed in Appendix H) gives almost no guidance as to reasonable numerical estimates. We take the only published estimate we found (0.6, by Greenwood et al., 1998) as our middle estimate, but that source explicitly admitted its number rested on little or no empirical basis. For low and high estimates we choose 0.5 and 0.7, respectively. We thus assume a 30 percent to 50 percent degradation in effectiveness when the experimental program is scaled up to national implementation. Again, these are opinions, not scientific estimates.

INTEGRATING THE FACTORS

Table 2.3 summarizes our low, middle, and high estimates of the eight factors that collectively determine the effectiveness of a model school-based drug prevention program. Multiplying together one factor from each of the eight rows gives an estimate of the net present value of the reduction (in grams) in lifetime cocaine consumption per participant in the prevention program.

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[19]Divergence of opinion from ours may be in either direction from the range we have adopted, as we have learned from discussions with other drug policy researchers. Some hold that the correlation between marijuana initiation age and cocaine consumption is coincidental (see Appendix G for further discussion). But we have also encountered an argument (Weatherburn, 1998, personal communication) that, whatever salutary effect prevention may have on marijuana use, an even greater effect should be expected on the use of cocaine. The theory here is that if juveniles inclined to use drugs wish to err on the side of caution, they would be drawn to "less serious" drugs. Conversely, if a program raised their level of inhibition about drugs, the strength of that inhibition would increase with the perceived seriousness of the drug.

Table 2.3

Estimated Effect of Prevention on Cocaine Consumption Per Participant

Factor	Low Estimate	Middle Estimate	High Estimate
Effect on participants			
Proportion of persons who ever use cocaine	0.13	0.20	0.27
Lifetime consumption per user (grams)	225	350	475
Proportion reduction due to program	0.029	0.076	0.136
Discount factor	0.388	0.507	0.692
Effect on nonparticipants			
Social multiplier	1.0	2.0	2.9
Market multiplier	1.0	1.3	2.0
Qualifiers			
Causation/correlation ratio	0.5	0.9	1.0
Scale-up factor	0.5	0.6	0.7
Product of all factors			
Effectiveness with discount factors shown (grams)	0.08	3.77	48.85
Effectiveness with middle discount factor (grams)	0.11	3.77	35.79

The table shows clearly that there is not just one estimate of prevention's effectiveness. Putting together different estimates for various factors will produce different estimates of the overall effectiveness. For example, consider taking the middle estimate for each factor. The product of the first two factors is the base-case lifetime consumption per average person in the population (20 percent of 350 grams per user shows average consumption to be 70 grams).

Multiplying by the proportion reduction due to the prevention program gives us the amount of the reduction (in grams). The discount factor gives an equivalent value of that reduction at the time the program costs occur. Then the market and social multipliers account for effects on other cohorts, and the qualifiers modify the result to give us the bottom-line middle estimate that the program prevents a present value of approximately 3.8 grams of cocaine consumption per program participant.

So, our best estimate is that the expected benefit of putting one person through an exemplary school-based prevention program is a present value of about 3.8 grams less cocaine consumption. (As mentioned above, most participants never initiate cocaine use, so for

them the effect is zero. For some it is much greater. This 3.8-gram figure is an average.) That reduction is fairly small compared with the (undiscounted) base case of 70 grams per person in the program. Clearly, even a model prevention program is not like a vaccine against the measles, which confers virtual immunity on the majority of those inoculated. Prevention's limited effectiveness has implications not only for cost-effectiveness, which we will discuss in the next chapter, but also for nationwide implementation, which we will consider in Chapter Five.

Uncertainty

From Table 2.3, it is easy to calculate what would happen if someone thought that the low or high value for one or a few variables was closer to the truth than our mid-range estimates. For example, if we take the low values for the two multipliers alone, the effectiveness estimate is reduced by about 60 percent. This approach might be preferred by someone who was skeptical of multiplier effects or who was uninterested in effects on persons not participating in the prevention program. Such an individual would infer an effectiveness of 1.45 grams of cocaine consumption per program participant (assuming he or she believed the rest of the mid-range estimates). We will have more to say about the ramifications of selected departures from the mid-range estimates when we discuss cost-effectiveness in Chapter Three.

To illustrate the effect of uncertainty, Table 2.3 also shows the result of multiplying together all of the low estimates to get one extreme, and all of the high estimates to get the other extreme. This is done both while varying the discount factor (yielding 0.08–48.85 grams) and while holding the discount factor fixed at its middle level (0.11–35.79 grams). Holding the discount factor constant is reasonable because, as discussed above, the variation in that factor is not the result of uncertainty about cocaine use and markets.

We can gain a sense of the potential range of effectiveness values by multiplying together all eight low factors and all eight high factors (or all seven of each if we accept the mid-range discount factor). However, because uncertainty about one factor is largely independent of uncertainty about the other factors, it is highly unlikely that *all* factors would take on the low or high values shown in the table. It is

thus highly unlikely that the true effectiveness value lies near the extremes of the effectiveness ranges given in the bottom two lines of the table.

To establish a more reasonable range of variation around the middle estimate, we perform a Monte Carlo simulation. This method is a standard procedure for learning how uncertainty surrounding various factors aggregates into uncertainty about some overall quantity. We hypothesize that each factor (except the discount factor) takes on each possible value over its range with some supposed probability. A computer draws one value randomly from each of the seven probability distributions and multiplies the results together, then multiplies that product by the middle discount factor to come up with an effectiveness number. This draw or "trial" is repeated until there are enough results to come up with a smooth probability distribution for overall effectiveness. For the present analysis, we made 16,000 draws for each of two hypothesized probability distributions for the factors. In the first case ("uniform"), we assume an equal probability that the true value of the factor takes on any value between the low and high estimates (and a zero probability that it falls outside the range). For the second ("triangle"), we assume that the middle estimate is the most likely, and that the low and high (and anything beyond) have a likelihood of zero. The likelihood of any other value is then linearly interpolated between the low or high and the middle (e.g., a value two-thirds of the way from the low to the middle has two-thirds the probability of being true that the middle has).

The results of the simulation are shown in Table 2.4. With the assumption of uniform distributions, 90 percent of the trials fall between 1.01 and 8.82. These may be taken as reasonable low and high estimates for overall effectiveness. (We retain the 3.77 grams from Table 2.3 as our preferred mid-range estimate.) Note that, although this diminishes the uncertainty greatly from that suggested in Table 2.3, the high estimate is still 8.7 times the low one. In Chapter Three, we will see that this is a considerably larger range than we have found in the past for other drug control strategies.

The triangle distribution yields a somewhat narrower range of values for any given percentile deviation from the median. However, the triangle distribution assumes that factor value probabilities ap-

Table 2.4

Probability Distribution of Overall Effectiveness Values from Monte Carlo Simulation

| | Distribution | |
	Uniform	Triangle
Mean	3.84	3.86
Standard Deviation	2.56	1.80
Percentiles		
0.5	0.53	1.01
2.5	0.81	1.34
5	1.01	1.57
10	1.29	1.87
25	1.97	2.55
50	3.20	3.54
75	5.01	4.80
90	7.26	6.25
95	8.82	7.28
97.5	10.47	8.24
99.5	13.99	10.18

NOTE: See text for explanation of distributions.

proach zero as values approach the extremes of the factor distribution. This is unsatisfactory for factors such as program effect, for which we have no particular reason to believe that the low and high values are any less likely than the middle. It is particularly unsatisfactory for the qualifiers, for which we are willing to concede some probability that the true value lies outside the range we assume. For such factors, the triangle distribution underestimates the probabilities of the low and high factor values and thus underestimates the range of values that overall effectiveness might take.

On the other hand, the triangle distribution may be more appropriate where we have more confidence that the middle estimate is more probable than the low or high. This is especially true of the market multiplier, whose range is itself the product of a Monte Carlo simulation.

Sources of Effect

Obviously, there is considerable uncertainty surrounding prevention's ability to reduce cocaine use. There is less uncertainty concerning the sources or nature of whatever reduction would occur among program participants, because the allocation of effects to sources is independent of the qualifiers and multipliers. Recall that the 7.6 percent mid-estimate for program effect is based on reduced cocaine initiation (Figure 2.3) and reduced usage by those who still initiate (Figure 2.4). Recall also from the discussion on the discount factor that the 7.6 percent includes the change in the discount factor when the timing of lifetime cocaine use is altered by prevention. The contribution of each of these three sources to the 7.6 percent is shown in Figure 2.7. The great majority—71 percent—of the reduction is associated with reduced cocaine initiation. A significant amount stems from reducing the average quantity consumed by those who do use some cocaine. Only a little is associated with the delay in consumption associated with the prevention-induced shift in the discount factor.[20]

Figure 2.7 accounts for only the reduction in use by program participants. We can create a parallel graph that apportions the consumption reduction among three groups of people: those in the prevention program; friends, family, and associates of people in the program; and people whose only relationship to those receiving the program is through impersonal market forces. (See Figure 2.8, which is based on calculations in Appendix C.) It shows that 38 percent of a prevention program's effect is achieved by changing the consumption of people in the program. One-sixth is spillover benefits through market interactions, that is, through the increased effectiveness of enforcement against the cocaine market. This is almost always overlooked when counting prevention's benefits. The largest of the three effects comes from short-circuiting the process by which initiation and use by some people (those in the program) prompts others to initiate and use (e.g., their friends and associates and friends and

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[20]Note the difference between "delayed consumption" and delayed initiation. In Figure 2.7, "delayed consumption" refers only to the reduction in the present value of consumption that is shifted further into the future. Delaying initiation results in the "reduced quantity consumed" portion of the pie chart.

RAND MR923-2.7
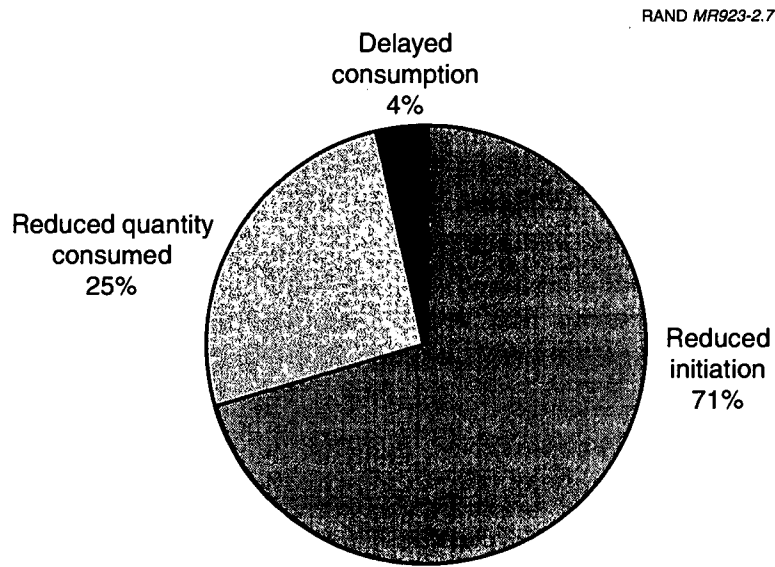
Figure 2.7—Sources of Prevention-Induced Reduction in Cocaine
Consumption by Program Participants

RAND MR923-2.8
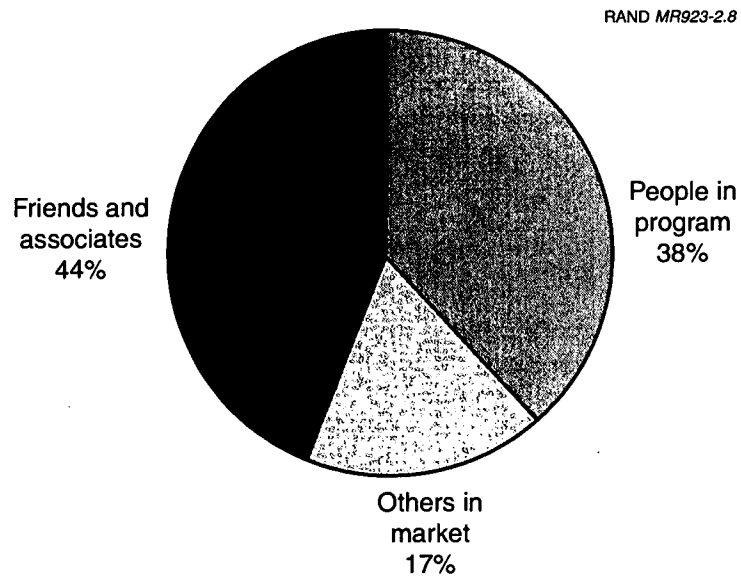
Figure 2.8—Contributions by Different Groups to Total Prevention-
Induced Reduction in Cocaine Consumption

associates of those friends and associates). (In contrast to Figure 2.7, Figure 2.8 is not, of course, independent of multipliers but assumes the mid-range estimates are correct.)[21]

These two figures illustrate the importance of the eight-factor model system's perspective. Without it, someone might be inclined to think that most of a prevention program's effect on cocaine use would be (directly) attributable to its ability to prevent program participants from initiating cocaine. But preventing initiation by program participants accounts for only one-quarter (71 percent × 38 percent) of the effect.

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[21]The percentages in Figure 2.8 do not add to 100 because of rounding error: The numbers are, more precisely, 17.3 percent, 38.5 percent, and 44.2 percent. These percentages are not in the same ratio as the multipliers (1:1.3:2) because friends and associates contribute to the base on which the market multiplier works. Thus, the number of additional persons affected by the multiplier for each program participant is not $(1.3 - 1) + (2 - 1)$ but $(1.3)(2) - 1$. The contribution of program participants is thus $1/(1.3)(2)$. That of others in the market is $[(1.3 - 1)(2 + 1)]/ [2(1.3)(2)]$, and that of friends and associates is $[(2 - 1)(1.3 + 1)]/ [2(1.3)(2)]$.

COST-EFFECTIVENESS AT REDUCING COCAINE CONSUMPTION

We have now developed an estimate of the effectiveness of school-based prevention programs at reducing cocaine consumption. The estimate has been discounted to reflect the time value of money and accounts for effects on people other than program participants. It allows for the possibilities that a key correlation may not be causal and that effectiveness may be lost on scale-up.

To estimate cost-effectiveness, the only thing remaining is to divide effectiveness by cost to determine kilograms of cocaine consumption averted per million program dollars spent. That is what we accomplish in this chapter. We begin by deciding what sorts of costs to include and estimate those costs for our model school-based prevention program. We then perform the division required to obtain cost-effectiveness and compare this estimate with those obtained previously for other drug control approaches. Uncertainty remains an important aspect of our analysis. Therefore, we compare uncertainties of estimates across approaches and examine the sources of uncertainty for the current estimate. Finally, we consider the variation of cost-effectiveness with the passage of time.

DEFINING PROGRAM COST

While the scientific literature on drug use prevention has contributed greatly to our understanding of the relative effectiveness of different approaches, it is uniformly lacking in one element essential for policy formulation: cost data (Lipsey, 1997). The published evaluations of

Project ALERT and the Life Skills Training Program, like those of most prevention programs, do not discuss costs. However, they do report resources used by the programs, making cost estimation possible.

The most difficult aspect of estimating program cost is defining what should be included; given a definition, the estimation is straightforward. Obviously, the costs of course materials must be included. But what about the value of the teacher's time taken up by training and by teaching the prevention classes? And what about the cost of the facilities in which the classes are taught? Some might argue that the only costs that should be considered are those the district is not already paying—the costs of course materials and those related to training teachers in the new curriculum. These are the only extra costs a school district must explicitly include in its budget.

Such an estimate of costs, while sufficient for accounting purposes, shortchanges the full value of the resources committed to drug use prevention. Teachers have been employed to educate children in English, history, math, science, and other subjects the state and the school district believe they must know. If the teacher spends an hour teaching drug use prevention, that is an hour that is not being spent doing what the teacher was originally hired to do. The cost of that hour will not show up on the district's books, but it will show up in what children do not learn. What is the value of an hour of a teacher's time spent on such subjects? One convenient measure is the teacher's salary, expressed in hourly terms. Various objections might be raised to this approach, including these:

- A drug use prevention class may provide as much intellectual challenge to students as any other subject.

- There may be enough slack in the school day to permit teaching other subjects adequately in shorter periods for the relatively brief duration of the prevention course.

- The prevention course could be taught as a supplement to the regular curriculum instead of a replacement for part of it.

- The standard curriculum is itself subject to change. *Current* state or district requirements determine the appropriate use of

teacher time, and drug use prevention could be one of those requirements.

We do not find these arguments persuasive. The first point is essentially an empirical hypothesis, but we are aware of no evidence supporting it. On the second and third points, what is at issue is not whether there may be time available but what is done with it. Regarding any hour that is found either within or outside the current school day, we can ask, What use of this time will best contribute to students' education? In any event, teachers are unlikely to accept an extension of the school day without being paid for it, which would turn the cost of their time spent on drug use prevention into a budgetary cost. Finally, on the last point, what needs to be answered about making any curriculum substitution is, What is the benefit and what is the cost? The benefit is what is gained from the new topic, which, for drug use prevention, we estimated in Chapter Two.[1] The cost is what is lost with the old, which is what we address here. The old topic could, of course, be an ineffective prevention course, but we are not concerned in this book with choosing among prevention strategies. Naturally, if a decision has been made to offer a drug prevention curriculum, the most effective one should be offered. What we are concerned with here, however, is whether prevention should be undertaken at all.

In deference to conflicting views, however, we retain as a low-cost estimate the curriculum cost alone—i.e., the cost of the course materials and of the time the teacher spends in training. For a middle estimate, we add the cost of teachers' time in teaching the drug use prevention classes. However, in contrast to the estimates we gave in the preceding chapter, the middle estimate is not in this case our preferred one. We believe it omits additional important costs—the costs of the school facilities used to house the drug use prevention classes. This may be obtained by determining an hourly share of a district's facilities budget.

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[1]At least as far as reducing cocaine consumption is concerned. Other benefits are discussed in Chapter Four. One benefit that may be of some interest here is that, if successful in reducing drug consumption by students, prevention could contribute to improved classroom behavior and student receptiveness to all subjects that are taught.

It may be argued that these costs should not be counted because they are sunk. A share of facility maintenance might be charged to the prevention program, if teacher time is to be charged, but the buildings are already there. Adding an activity or substituting one for another will not change that.

We are not, however, attempting to estimate the opportunity cost of the real estate, which might well be zero. We are attempting to estimate the cost of the lost learning opportunity. This is difficult to evaluate. One way of doing so, however, is to assume that, when people pay for teachers and for school buildings in their district, they believe the purpose of those resources—education—is worth the cost. Otherwise, they would spend the money on something else. If the district contracts with teachers and builds structures to deliver a certain number of hours of education to a certain number of students, that suggests a value attached to one hour spent by one student in class. It also suggests the value of an hour lost.

The value so obtained could be an underestimate. It may be that students gain much more from their education than the state and the district spend on it. Or it could be an overestimate. People do not actually sit down with data in front of them on input costs and output values to decide what they will spend on schools. But the full costs of the resources devoted to education presumably reflect some sense of education's worth. For our purpose, we prefer those costs to a deliberately partial accounting.

Our purpose is to account for prevention's drug use reduction benefits and as much of its social cost as we can, to allow comparison with previously estimated cost-effectiveness values for other drug control approaches. Without the ability to make such comparisons, estimating a cost-effectiveness number would be of limited value. Aside from our theoretical justification of an inclusive cost estimate, there is thus an important practical one: Our approach here is consistent with that of previous evaluations of drug control strategies, which charged the costs of building prisons, for example, to enforcement strategies (Rydell and Everingham, 1994; Caulkins et al., 1997). It is also consistent with other papers on prevention's cost-effectiveness (e.g., in an evaluation of a school health promotion evaluation reported by Connell, Turner, and Mason, 1985). As suggested above,

more narrowly focused estimates of prevention's costs may be more appropriate for other purposes.

ESTIMATING PROGRAM COST

The cost of the materials for the Life Skills Training Program ($161 per class[2]) is higher than the cost for Project ALERT ($76 per class).[3] The teaching time costs are significantly higher because Project ALERT entails 11 sessions while the Life Skills program is 30 sessions. We utilize the Life Skills program costs in our analysis because this prevention program includes the booster sessions that may be important in sustaining the benefits to the end of high school and beyond.

Estimates of teacher and facilities costs, on average for the United States, are given in Table 3.1. In Table 3.2, we calculate the costs, under the alternative definitions of program cost, of the Life Skills program. For the purposes of this analysis we assume that this one-time cost is averaged over six courses taught by a given teacher, say two times a year for three years, and to calculate the cost per participant we assume that a class has 30 students.

Program costs per participant according to the three cost definitions are estimated to be $1.94, $67.12, and $146.50 per program participant.[4] If we multiply these program costs by 3.75 million children in a national birth cohort, we obtain the estimated annual cost of a full-scale national drug-use prevention program. It ranges from $7 million to $549 million. Although the cost of materials and training is very small compared to the cost of the teacher's time and the essential facilities, even the highest of the three cost figures is not large by

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[2]All costs in this chapter are expressed in 1992 dollars (unless stated otherwise) for comparability with the results of previous studies.

[3]The Project ALERT cost, however, is an underestimate of the true cost, because the program is subsidized by the Conrad N. Hilton Foundation. We do not know whether the Life Skills program is also subsidized.

[4]If the Life Skills program is subsidized, an estimate based on budgetary costs alone could be somewhat higher than the low estimate shown here. That error, however, does not substantially affect the cost-effectiveness estimates we favor, which are based on the high cost estimate.

Table 3.1

Instructional-Staff and Facilities Costs, U.S. K–12 Public Schools, 1992–93 School Year

Cost Category	Total Annual Cost (millions of dollars)	Annual Cost Per Pupil (dollars)	Cost Per Class Session (dollars)
Instructional staff	114,466	2,893	68.88
Facilities	139,393	3,523	83.88
Total	253,859	6,416	152.76

SOURCE: National Center for Education Statistics (1995), Table 38, pp. 50–51, column for 1992–93.

NOTE: Cost of instructional staff is 3.140 million staff times average annual salary of $36,454. Cost per pupil rests on the average daily attendance of 39.586 million pupils. Cost per class session assumes seven classes a day, 180 days per school year, and 30 pupils per class. Costs are in 1993 dollars.

Table 3.2

Cost of Life Skills Training Program

Cost Component	Cost (dollars)
One-time curriculum cost	
Materials (books, videos, posters)[a]	161
Teacher time for training (one day)[b]	197
Total	358
Cost per prevention course (30 classes)	
Curriculum costs[c]	60
Teacher cost[d]	2006
Facilities cost[e]	2443
Total	4509
Net present value per participant per course[f]	
Curriculum cost only	1.94
Curriculum and teacher costs only	67.12
Curriculum, teacher, and facilities costs	146.50

SOURCES: Botvin et al. (1995), Drug Strategies (1996), and Table 3.1 above.

NOTE: All costs are in 1992 dollars.

[a]$175 in 1995 dollars (Drug Strategies, 1996, p. 29).

[b]Annual teacher salary of $36,454 in 1992–93 divided by 180 days per school year is $203; deflation to 1992 gives $197.

[c]Assuming that teachers and materials are replaced every six courses (e.g., every third year, assuming two courses per year).

[d]$68.88 per 1992–93 class times 30 classes gives $2066; deflation yields $2006.

[e]$83.88 per 1992–93 class for 30 classes gives $2516; deflation yields $2443.

[f]Assumes 30 pupils per course. Present value is obtained by discounting second- and third-year costs by 4 percent per year.

national drug control standards. Recall from Chapter One that the United States spends about $40 *billion* per year on drug control.

ESTIMATING COST-EFFECTIVENESS AND THE IMPLICATIONS OF UNCERTAINTY

An estimate of prevention's cost-effectiveness can be found by dividing an estimate of its effectiveness by a measure of program cost. For example, the effectiveness analysis showed that the middle estimate of program effectiveness is 3.8 grams in cocaine consumption averted per program participant. We divide this figure by our $146.50 estimate of program cost per participant (and multiply by 1000 to convert grams per dollar into kilograms per million dollars). This gives a middle estimate of 26 kilograms in cocaine consumption averted per million dollars spent on the prevention program.

However, this ratio of the middle effectiveness estimate to our preferred cost estimate is just one estimate of cost-effectiveness. To get a sense of the range of possible values, Table 3.3 displays nine values that can be obtained by dividing each of the effectiveness estimates in Chapter Two—low, middle, and high—by the three cost estimates. For the low and high effectiveness estimates, we use the 5th and 95th percentiles from Table 2.4. (The effectiveness estimates used here are all based on a discount rate of 4 percent—i.e., a discount factor of 0.507.)

Table 3.3

Cost-Effectiveness at Reducing Cocaine Consumption
(range of estimates in kilograms reduced per million dollars spent)

| Cost | Effectiveness Estimate | | |
Estimate	Low	Middle	High
High	7	26	60
Middle	15	56	131
Low	521	1945	4550

NOTE: Each cost-effectiveness estimate shown is the result of dividing the high, middle, or low effectiveness estimate in Chapter Two by the high, middle, or low cost estimate from Table 3.2.

Estimates in the first row can be compared with the results of previous studies—because they are based on the same costing principles.[5] These three prevention cost-effectiveness estimates are shown in relation to "best estimates" of the cost-effectiveness of other drug control strategies (from Caulkins et al., 1997) in Figure 3.1. The other strategies considered are the following:

- Coca leaf eradication and seizures of coca base, cocaine paste, and cocaine in the source countries.

- Extending sentences of typical cocaine dealers.

- Interdicting cocaine on route from source countries to U.S. markets.

- Expanding the mix of enforcement strategies (arrest, prosecution, and incarceration) used against typical dealers before the advent of mandatory sentencing.

- Extending sentences of dealers prosecuted at the federal level to lengths typical of those in mandatory-minimum laws.

- Expanding the mix of enforcement strategies used against dealers prosecuted at the federal level before the advent of mandatory sentencing.

- Treating heavy users with a program that keeps most (79 percent) off cocaine during treatment and causes a minority (13 percent) to desist from heavy use after treatment.

As the figure shows, the three prevention estimates almost bracket the full range of estimates for the various approaches, with the exception of treatment. The middle estimate of a model prevention program's cost-effectiveness is comparable with that of several enforcement alternatives. It is considerably more cost-effective than source country control or extending sentences of typical dealers, but the low prevention estimate is below both of those. According to the mid-range estimate, prevention is considerably less cost-effective than extended federal enforcement, but if the high estimate is right

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[5]It is worth keeping in mind, however, that previous estimates are for average program performance, while the prevention estimates are for model program performance.

RAND *MR923-3.1*

Model prevention
(low estimate)

Source country control

Longer sentences,
typical dealers

Interdiction

Model prevention
(middle estimate)

Domestic enforcement,
typical dealers

Longer sentences,
federal defendants

Model prevention
(high estimate)

Federal enforcement

Treatment

0   20   40   60   80   100   120

Cost-effectiveness at reducing cocaine consumption
(kilograms averted per million program dollars spent)

**Figure 3.1—Comparison of Prevention With Other Cocaine
Control Programs**

instead, prevention compares favorably. Treating heavy users ap-
pears more effective than prevention, regardless of which prevention
estimate is chosen.

To keep Figure 3.1 simple, we have compared several prevention es-
timates with "best estimates" for other strategies. However, all our
cost-effectiveness estimates for cocaine control strategies are subject
to uncertainty. How do the uncertainty ranges compare? Table 3.4
shows ranges of cost-effectiveness values for prevention and three
other strategies, all derived through the same Monte Carlo method.
Although the enforcement strategies shown are also subject to con-
siderable ranges of uncertainty, those ranges are only about half as

83

wide, when measured by the ratio of high to low estimates, as the range for prevention.

And the range shown for prevention in Table 3.4 does not account for differing cost definitions or beliefs about qualifiers that fall outside our range of estimates for those factors. Such differences help explain why there are such divergent opinions about prevention's cost-effectiveness. This is illustrated in Table 3.5. Here we repeat the base-case, middle estimate, of 26 kilograms reduction in cocaine consumption per million dollars spent on prevention, along with all the factors leading to that estimate. Then we contrast this with the estimates of two (hypothetical) persons of differing opinions. Person 1 believes the middle estimates of all factors but holds that the cost of prevention should include only curriculum cost. That person finds that prevention is enormously cost-effective, more than an order of magnitude better than the next best program shown in Figure 3.1.

Person 2 believes the standard cost estimate, and the middle estimates of cohort effects and market and social multipliers. However, this person is very pessimistic about historical correlation being indicative of future changes, and about experimental performance holding up in full-scale implementation. So Person 2 goes down to 0.1 as an estimate of these qualifying factors and concludes that prevention's cost-effectiveness is near zero.[6]

It is interesting that these two divergent opinions result from disagreement over the qualifiers and the cost estimate, none of which is part of the usual debate over prevention's cost-effectiveness. Most of the attention so far has been given to program effectiveness in the narrow sense (factor 3). And this factor's contribution to uncertainty

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[6]To underscore the range of uncertainty possible, we have chosen to define Person 2 as a skeptic regarding our qualifiers. Those who are skeptical of our mid-range estimates on other variables could not reasonably have gone much below our low estimates. The effects of choosing two low estimates of other variables would not have been as drastic and might be offset by preferences for high estimates on still other variables. See the Monte Carlo uncertainty analysis in Chapter Two. Note also that profound skepticism regarding the causation-to-correlation qualifier suggests disbelief that prevention programs influencing marijuana initiation also influence cocaine use; it may allow for the possibility that prevention has other salutary effects, e.g., on lifetime marijuana use, that are pertinent to a broader (and more favorable) estimate of its cost-effectiveness. See Chapter Four.

is substantial: The ratio of its best to worst estimates is 4.7:1. So collecting additional evidence on how prevention programs affect marijuana initiation (including what happens after age 18) would be helpful.

Table 3.4

Low, Middle, and High Cost-Effectiveness Estimates for Different Cocaine Control Strategies

| Strategy | Cost-Effectiveness (kg. averted per million dollars spent) | | | High:Low Ratio |
	Low Est.	Middle Est.	High Est.	
Prevention	7	26	60	8.7
Mandatory minimums	17	36	75	4.4
Increased federal enforcement	35	63	158	4.5
Treating heavy users	90	104	147	1.6

NOTE: Estimates other than prevention are from Caulkins et al. (1997), pp. 136, 143.

Table 3.5

Illustration of Implications of Different Opinions About Factors Related to Prevention's Cost-Effectiveness

	Base Case	Person 1	Person 2
Effect on cohort			
Proportion of persons who ever use cocaine	0.2	0.2	0.2
Lifetime consumption per user	350	350	350
Proportion reduction due to program	0.076	0.076	0.076
Discount factor	0.507	0.507	0.507
Interaction among cohorts			
Social multiplier	2.0	2.0	2.0
Market multiplier	1.3	1.3	1.3
Qualifiers			
Causation/correlation ratio	0.9	0.9	0.1
Scale-up factor	0.6	0.6	0.1
Results			
Grams reduction per participant	3.77	3.77	0.07
Cost per participant	$146.50	$1.94	$146.50
Kg. reduction per million dollars	26	1943	0.5

However, the other six non–discount-related factors are collectively a far greater source of uncertainty. Together, the ratio of the product of their best to their worst values is over 70:1. Very little research effort or funding is devoted to studying these factors. There might be substantial returns (in terms of reducing uncertainty about prevention's effectiveness) to allocating dollars to funding studies of, for example,

- responsiveness of quantities consumed or supplied to changes in price

- cost components of drug supply

- effect of one person's drug use on that of another

- how drugs accumulate a negative reputation and how that suppresses initiation

- the interaction of personal characteristics (tastes, etc.) with drug use and the mechanisms by which prevention programs affect use

- how large-scale implementations of previous programs fare relative to their demonstrations.

VARIATION WITH THE PASSAGE OF TIME

Our effectiveness estimates are based on program trials in the mid- to late 1980s and on other data from around 1992; cost data are from 1992 or adjusted to that year. As explained in Chapter Two, though, the social multiplier (factor 5) changes over time. Obviously the proportion of the cohort that would initiate cocaine use without prevention (factor 1) does as well, and to a lesser extent other factors may vary, too. This variation is discussed in-depth in Appendix E. That appendix also simulates how the product of the two dominant factors (1 and 5) might have changed over time. This analysis is necessarily rough because the exact form of the feedback behavior that drives the social multiplier is not known. Nevertheless, it suggests that the product of these two factors may have been much higher between 1964 and 1973 than in the mid-1980s—enough, perhaps, to push the cost-effectiveness of prevention programs an order of

magnitude higher.[7] (And the advantage over prevention programs run now would probably be at least as great, because the cocaine epidemic has continued to "mature" or recede since 1992.) Simply put, prevention is more cost-effective when run a number of years before the peak in initiation, when there are relatively few heavy users. (Later in the epidemic, the increased number of heavy users means treatment is more cost-effective than prevention.) This finding has important implications for the prospects of a nationwide prevention program, a topic we discuss in Chapter Five.

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[7]A similar conclusion has been reached by Behrens et al. (1998).

OTHER BENEFITS

So far, we have been concerned with how effective prevention is at reducing cocaine use relative to the resources used by the prevention program. In this chapter we broaden that perspective in two respects. First, we roughly estimate prevention's effect on the use of three drugs other than cocaine: marijuana, alcohol, and cigarettes. Second, we monetize the value of reductions in drug use so we can convert our cost-effectiveness estimates into benefit-cost ratios. We conclude with a brief discussion of some benefits of drug use prevention that are unrelated to drug use.

ESTIMATING THE EFFECT ON USE OF DRUGS OTHER THAN COCAINE

To estimate the effect of model school-based drug use prevention programs on marijuana, alcohol, and cigarette use, we follow the general outline of the cocaine analysis in Chapter Two. That is, we base our estimate of program effectiveness on the program's reduction in a risk factor. We then use NHSDA data to correlate the decrease in the risk factor with a decrease in the outcome of interest—in this case, marijuana, alcohol, or cigarette consumption. We then account for the discount factor, multipliers, and qualifiers to the extent required. We do not attempt, however, to calculate average lifetime consumption. Thus, instead of deriving a reduction in consumption per million dollars spent on prevention, we calculate a percentage reduction. This percentage can then be compared with the percentage reduction that prevention causes in the use of other drugs. In the case of alcohol and cigarettes, it can also be converted

into a social benefit-cost ratio. For cigarettes, we also take a second approach to a comparison of benefits and costs. (For details of the calculations of prevention's effects on alcohol and cigarette use, see Appendix I.)

We view the estimates for these three drugs as much rougher than the estimates for cocaine. Our principal objective was to evaluate prevention's effectiveness at controlling cocaine and we designed our analytical approach around that objective. If we had set out to study prevention's effectiveness on alcohol use, for example, we probably would have approached the problem differently. In particular, we could have drawn on evaluations of more programs besides Project ALERT and Life Skills, and we could have modeled the permanence of initiation delays differently. However, as long as we have constructed the framework for analyzing effects on one drug, it is relatively easy to apply the framework to other drugs. And it can be instructive to compare the magnitudes of the effects estimated with a common methodology.

In the next subsection, we estimate simple program effectiveness, undiscounted and unqualified, on participants. Subsequently, we take the other factors into account.

Simple Program Effectiveness

Marijuana. The analysis with marijuana as the outcome variable is completely parallel to that for cocaine, as described in Appendix C. In particular, the same risk factor (age of marijuana initiation) is used. The resulting low, middle, and high estimates of prevention's effect on (undiscounted) lifetime marijuana use are 1.8 percent, 6.1 percent, and 12.8 percent, respectively (see Figure 4.1, which is constructed in parallel to Figure 2.6 for cocaine). Compare these with the effects on cocaine use of 2.9 percent, 7.6 percent, and 13.6 percent, respectively (Figure 2.6). At first it might seem odd that preventing marijuana initiation is projected to have a smaller effect on lifetime marijuana use than it does on cocaine use. There are two explanations. First, people who initiate marijuana in junior and senior high school use 2.3 times as much marijuana over the course of their lives than those who initiate marijuana later. But early marijuana initiators use 3.8 times as much cocaine. So delaying marijuana initiation is estimated to have a greater effect on a cohort's
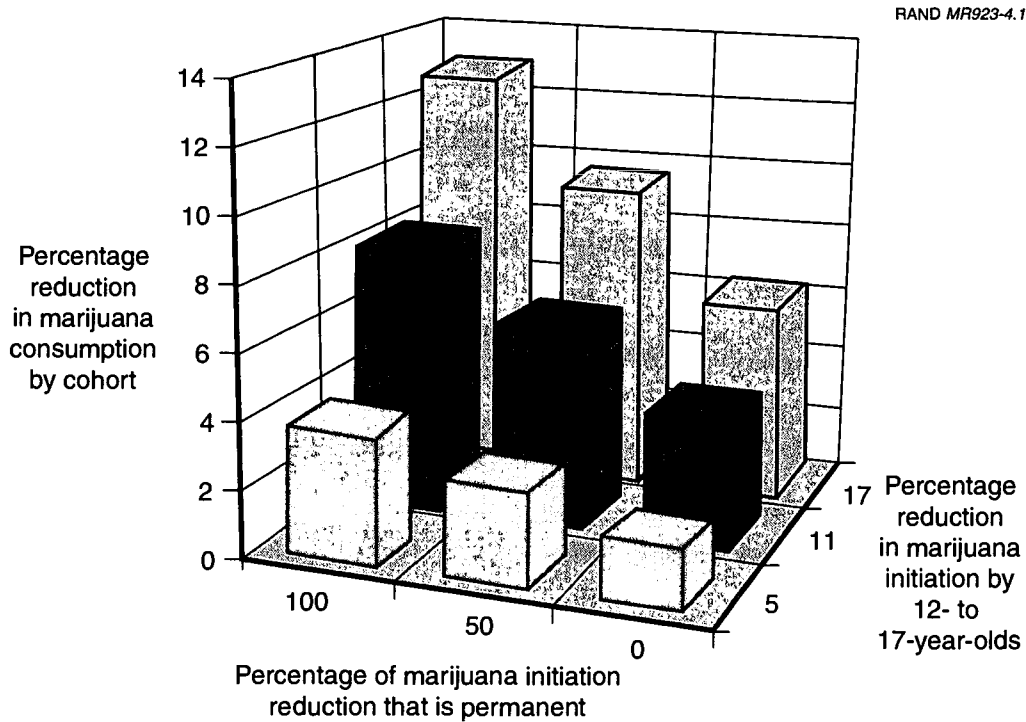
Figure 4.1—Reduction in Cohort's Marijuana Consumption for Different Assumed Values of Marijuana Initiation Reduction and Permanence

cocaine use than on its marijuana use. Second, the 40 percent of people who initiate marijuana use before age 18 account for 80 percent of all marijuana use, but 90 percent of all cocaine use. So permanently preventing a marijuana initiation in junior or senior high school has a slightly greater proportional effect on the cohort's cocaine use than on its marijuana use.[1]

Alcohol. Adolescents consume alcohol, and there are data available on that consumption and on the effect prevention programs have on it. Thus, we do not have to rely on an indirect proxy like marijuana use (as we did for cocaine) but can use an indicator more directly related to the outcome. In particular, we use age of first monthly use.

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[1]Again, the program effectiveness percentages given here do not take into account the causation/correlation qualifier. This might be higher for effectiveness at reducing marijuana consumption than for effectiveness at reducing cocaine consumption, considering that the risk factor we are using is age of marijuana initiation.

For outcome measures—those we would like to see prevention re-
duce—we use two measures of problem drinking: number of occa-
sions on which a person has five or more drinks within a given
month and number of occasions on which a person has gotten drunk
within a given year.

Both the Project ALERT and Life Skills evaluations measured effects
on initiation into monthly use. Thus, we took the smaller of the two
effects as our low estimate (Life Skills, reduction of 1.7 percent) and
the larger as our high estimate (Project ALERT, 5.4 percent) and split
the difference for the mid-range (3.5 percent). Taking the possibility
of 0, 50 percent, or 100 percent permanence, and working through
the risk-factor-to-outcome correlation, we derive a percentage re-
duction in the outcome measure for each of the nine risk-factor re-
duction and permanence combinations. These are graphed in Figure
4.2 for number of occasions on which a person has five or more

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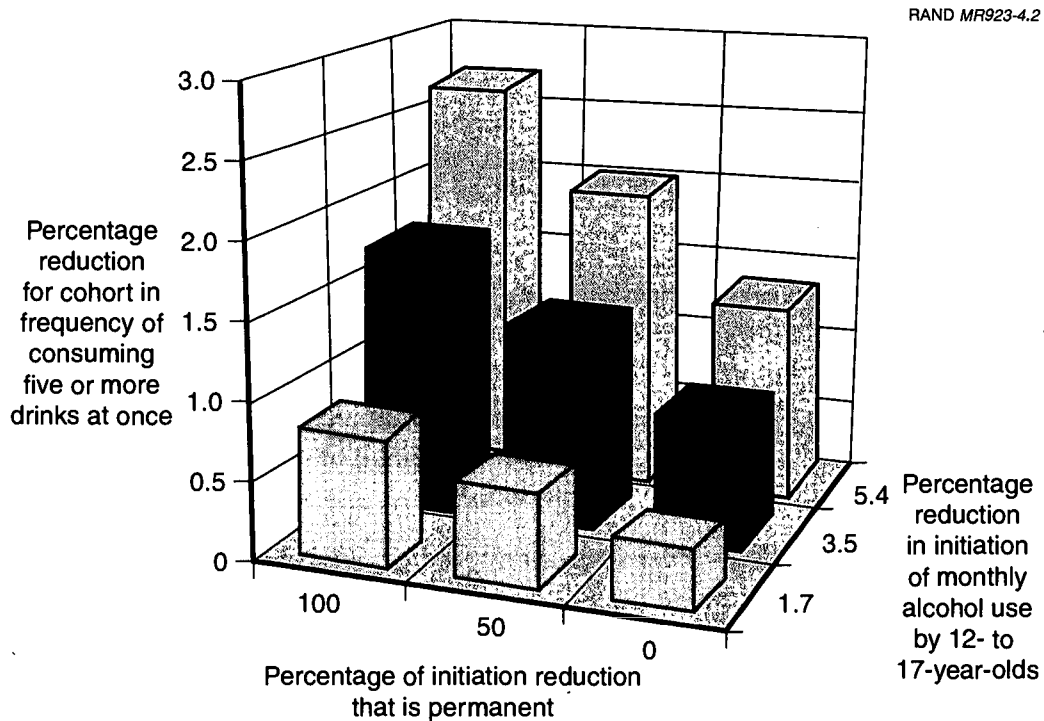
Figure 4.2—Reduction in Cohort's Alcohol Consumption for Different
Assumed Values of Alcohol Initiation Reduction and Permanence

91

drinks within a given month. The low, mid-range, and high estimates are 0.4 percent, 1.3 percent, and 2.6 percent. The values for the second outcome measure are similar: 0.4, 1.1, and 2.3 percent.

Cigarettes. We use the age at which a person first tries cigarettes as a risk factor for lifetime cigarette use. For the outcome measures that we would like to see prevention reduce, we took average number of packs smoked per day and probability of ever smoking five packs (total) in one's lifetime.

As with marijuana, only Project ALERT measured cigarette initiation through eighth grade, so we took the same approach to deriving our range of estimates of program effect on the risk factor as we did with marijuana initiation. We took the Project ALERT estimate (about a 4 percent reduction) as the low, averaged several estimates of weekly and monthly use (approximately 10 percent) as the high, and averaged the two for a mid-range (7 percent). Again, when we combine these three estimates with the three permanence estimates and take into account the correlation of the risk factor and the outcome measures, we derive nine estimates of program effect on the outcome. These are graphed in Figure 4.3 for the probability of ever smoking five packs. The low, mid-range, and high estimates are reductions of 0.2, 2.4, and 6.4 percent. For average number of packs smoked per day, the estimates are 0.8, 3.0, and 6.7 percent.

One striking characteristic of Figure 4.3 is that the estimated effect depends very heavily on the degree of permanence of the reductions in initiation observed through the end of high school. Recall that when age of marijuana initiation was used as a risk factor predicting lifetime cocaine use, it made relatively little difference how permanent the reductions in initiation were. With cigarette use (and, to a lesser extent, alcohol use), there is a greater premium on permanence of effect as opposed simply to delay. Regardless of age of cigarette initiation, about 60 percent of those who try a cigarette will smoke five or more packs in their lifetime. As for average number of packs smoked per day, the gradient along the permanence axis is not as steep as in Figure 4.3, but still steeper than for other drugs. Those who initiate cigarettes at later ages tend to have somewhat lower current rates of cigarette use, but not dramatically lower.
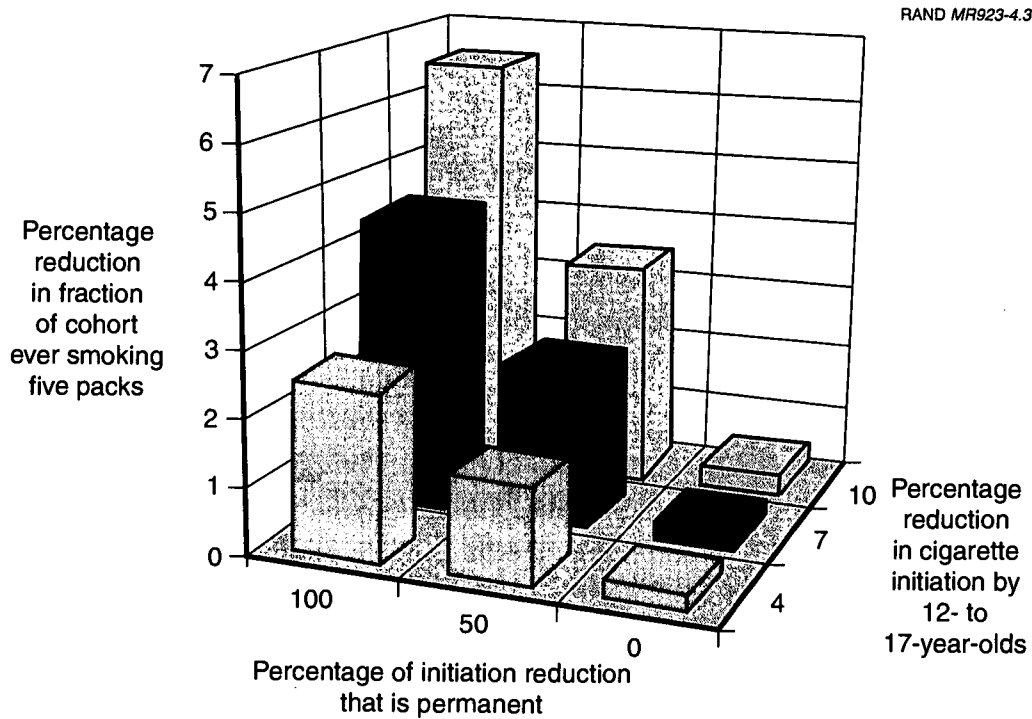
Figure 4.3—Reduction in Cohort's Cigarette Consumption for Different
Assumed Values of Cigarette Initiation Reduction and Permanence

It is problematic that the permanence of the reductions in cigarette
initiation observed through twelfth grade plays such a dominant role.
As a result, there is relatively greater uncertainty in predicting pre-
vention's effect on lifetime cigarette use than on the use of other
drugs. Conversely, a prevention evaluation follow-up around age 25
would do relatively more to reduce the uncertainty for the estimates
of effects on cigarette use than it would for effects on use of the other
substances considered.

Other Factors

As in the case of cocaine, we discount future benefits through the use
of a discount factor whose low, mid-range, and high values are based
on discount rates of 6, 4, and 2 percent, respectively. The discount
factor is highest (0.552, reflecting the least discounting) for mari-
juana because most marijuana is used primarily by people who are

relatively young. Cocaine's discount factor (0.507) is the next largest. Alcohol's (a little under 0.5) is smaller and cigarettes' discount factor (0.385) is the smallest because use of the licit substances persists longer into adulthood.

We set the multipliers at 1. Since adolescents use marijuana, drink alcohol, and smoke cigarettes and peer influences are likely to be felt principally within school, the social-multiplier effects are likely to have been picked up by the evaluation of program effectiveness. Since commerce in alcohol and cigarettes is legal, there is no enforcement that decreasing demand could make more efficient, no consequent rise in unit prices, and no resulting market multiplier effect. The market multiplier for marijuana is probably lower than for cocaine because marginal enforcement risk may play a smaller role in driving up marijuana prices than it does for cocaine prices, but it may well be greater than 1. Nevertheless, we set that multiplier equal to 1 for the sake of comparability with the alcohol and cigarettes results. Hence, the estimates for effect on marijuana may be viewed as being conservative in that respect. We have no basis for estimating a different causation-to-correlation qualifier and no reason to think the scale-up qualifier should be any different from that for cocaine, so we use the same qualifier values we used for cocaine.

Comparison of Effects

If we multiply the base estimate of the undiscounted, unqualified effect on program participants' use by the discount factor and two qualifiers, we obtain the base-case estimate of a model school-based prevention program's effect on the net present value (NPV) of drug use. These factors and their product (the latter in bold) are given in Table 4.1. This table is similar to Table 2.3 but varies in two respects. First, the outcome is in terms of the discounted percentage reduction in the activity, not in grams. As mentioned above, proportion of persons using the drug and lifetime consumption are not taken into account. Second, because we set the multipliers for all drugs other than cocaine at 1, we give estimates for cocaine with the multipliers set to 1 (outside the parentheses) and set to the values derived in Chapter Two (within parentheses).

Table 4.1 suggests that, in percentage terms, school-based drug prevention is most effective against drugs whose use is most deviant. Its effect is smallest on alcohol use, somewhat larger on cigarette use, much greater on marijuana use, and—particularly if the multipliers are included—the greatest on cocaine use.

In Table 4.2, we supplement the discounted base overall-effect estimates with several other representations of our bottom-line results. We give both point estimates and ranges, and we express them both with and without discounting. The indications of ranges are in the form of low and high estimates produced by a Monte Carlo analysis similar to that described in Chapter Two.

The undiscounted numbers are what we would observe over the lifetime of the users. To understand what an undiscounted effect estimate means, assume the estimate is 10 percent and that a group of people exposed to the prevention program would together have consumed 100 grams over the course of their lives. Then, with prevention, they and the people they would influence through the multipli-

Table 4.1

Calculating the Effects on Alcohol and Cigarette Use and Comparing Them With Cocaine and Marijuana Estimates

	Rate of Consuming 5+ Drinks at a Time	Rate of Getting Drunk	Likelihood of Ever Smoking 5 Packs	Rate of Smoking	Marijuana Use	Cocaine Use
Reduction due to program						
Low estimate	0.4%	0.4%	0.2%	0.8%	1.8%	2.9%
Base estimate	1.3%	1.1%	2.4%	3.0%	6.1%	7.6%
High estimate	2.6%	2.3%	6.4%	6.7%	12.8%	13.6%
Discount factor	0.468	0.489	0.385	0.385	0.552	0.507
Causation/correlation ratio	0.9	0.9	0.9	0.9	0.9	0.9
Scale-up factor	0.6	0.6	0.6	0.6	0.6	0.6
Overall effect on NPV of use						
Base estimate	0.3%	0.3%	0.5%	0.6%	1.8%	2.1% (5.4%)[a]

[a]The parenthetical quantity assumes multipliers with values given in Chapter Two; all other quantities in this row assume the multiplier is 1.

Table 4.2

Base Estimates and Ranges of Effect, Discounted and Undiscounted, for All Drugs Considered

	Rate of Consuming 5+ Drinks at a Time	Rate of Getting Drunk	Likelihood of Ever Smoking 5 Packs	Rate of Smoking	Marijuana Use	Cocaine Use
Overall effect (undiscounted)						
Base estimate	0.6%	0.7%	1.3%	1.6%	3.3%	4.1% (10.6%)[a]
Low estimate[b]	0.2%	0.2%	0.2%	0.5%	1.0%	1.4% (3.3%)[a]
High estimate[b]	1.3%	1.1%	3.1%	3.2%	6.2%	6.7% (22.6%)[a]
Discount factor	0.468	0.489	0.385	0.385	0.552	0.507
Overall effect (discounted)						
Base estimate	0.3%	0.3%	0.5%	0.6%	1.8%	2.1% (5.4%)[a]
Low estimate[b]	0.1%	0.1%	0.1%	0.2%	0.6%	0.7% (1.7%)[a]
High estimate[b]	0.6%	0.5%	1.2%	1.2%	3.4%	3.4% (11.5%[c])[a]

[a]Parenthetical quantities assume multipliers with values given in Chapter Two; all other quantities in this row assume the multiplier is 1.

[b]From Monte Carlo simulation analysis.

[c]Rounded from 11.48; thus, 11 when rounded to nearest whole number.

ers would consume 10 grams less. (Because the multipliers are greater than 1 only for the cocaine estimates in parentheses, for all other estimates no one else is influenced and the 10 grams is a reduction in consumption by the program participants themselves.)

Much of the reduction in use, however, occurs many years in the future. Thus, in thinking about the value of that reduction, either in absolute terms or relative to the cost of the program, it is important to include the discount factor. For example, if the discount factor were 0.5, then the 10-gram reduction in use over time would be only as valuable as a 5-gram reduction in use today. Hence, we included the discount factor when we computed cost-effectiveness in Chapter Three, and we will include it in our estimates of the dollar value of the social benefits of the drug use prevented, a topic to which we now turn.

SOCIAL SAVINGS FROM REDUCED DRUG USE

Cost-effectiveness estimates describe how much of a benefit, in this case reduced cocaine use, can be "bought" per dollar spent. They help determine what is the cheapest way to buy a benefit once one has committed to obtaining it—one way or another. Thus, where the purpose is to allocate drug control resources among drug control strategies, the decision can be guided by cost and a single benefit measure like decrease in consumption. This perspective is appropriate for deciding which broad themes should receive emphasis, as the president does through the national drug control strategy and special initiatives like the media-based prevention campaign announced in the summer of 1998.

Cost-effectiveness estimates do *not* help us decide whether the benefits of reduced cocaine use—e.g., lower health care costs, greater productivity, less crime—are worth "purchasing" at all. For various institutional reasons, resource allocation decisions often balance a drug control program with another type of program altogether. For example, police detectives can be assigned to work drug cases or crimes against persons. Military units can focus on drug interdiction or training and readiness. School resources, including classroom time, can be used to teach drug prevention or other subjects. In these cases, citizens and policymakers need to think about how many dollars in total should be spent on drug control as opposed to other potential uses of tax dollars. Knowing how cost-effective one drug control program is relative to another is not irrelevant to such decisions. School districts might be more likely to pursue drug prevention aggressively if they believed it was a singularly effective way to reduce drug use. However, decisionmakers in such circumstances might also appreciate having some other measure of the magnitude of the benefits they are "buying" with the resources they allocate to drug prevention. One way to do that is to convert all benefits generated by a program to monetary terms—i.e., devise benefit-cost ratios.

Within the scope of the current analysis, we have limited ability to derive benefit-cost ratios accounting for all the benefits of prevention. However, it is easy to produce a ballpark estimate of prevention's benefit-cost ratio if we limit it to the benefits from reducing the use of a certain drug.

Cocaine

We calculate a benefit-cost ratio for cocaine by using a published adaptation of what is perhaps the most widely cited set of estimates of the social costs of drug use, those of Rice et al. (1990). (Kim et al. (1995) mention a number of other estimates of the social costs of drug abuse, but they are all fairly close to the Rice et al. estimates.)

Based on Rice et al.'s (1990) figures, Rydell and Everingham (1994, pp. 37–39) estimate that in 1992, the costs of health care, lost productivity, and crime due to cocaine abuse in the United States amounted to about $27 billion. We assume this cost is proportional to the amount of cocaine consumed, which Rydell and Everingham (1994) estimate to be 291 metric tons in that year. Dividing $27 billion by 291 metric tons gives an estimate that the health, productivity, and crime cost per metric ton of cocaine is $93 million. Multiplying this by our point estimate that prevention averts 26 kilograms (0.026 metric tons) of cocaine consumption per million dollars spent yields an estimated savings of $2.40 in these social costs associated with cocaine use per dollar's worth of resources allocated to prevention.

If this estimate were the only plausible estimate, we might safely conclude that school-based drug prevention pays for itself just counting benefits associated with reduced cocaine use; other benefits—not quantified here—would be a "bonus," unneeded to justify investments in prevention. However, the same uncertainties about prevention's cost-effectiveness apply to its benefit-cost ratio. Hence, our low and high estimates of prevention's benefit-cost ratio with this measure of social benefit are $0.64 and $5.60 saved in cocaine-related costs per dollar's worth of resources consumed. Those estimates are both based on our preferred, broad definition of prevention-related costs.

Remember, though, that we are only counting benefits associated with reduced cocaine use. Suppose someone believed that other benefits of drug prevention must account for more than a third of the total. Even if that person gravitated toward the 64 cent-per-dollar benefit-cost ratio when considering only cocaine-related benefits, he or she could safely conclude from this analysis that prevention's

overall benefit-cost ratio is greater than 1. However, people thinking other benefits were smaller would have to be more circumspect.

Other Drugs

We have shown above that school-based prevention is much more effective, in percentage terms, at reducing cocaine use than it is at reducing use of licit substances. That does not mean, however, that the effects on licit substances are of second-order importance. Harwood, Fountain, and Livermore (1998, p. 3) find that the economic costs of alcohol use in the United States in 1992 were on the order of $148 billion, over five times the $27 billion in economic costs associated with cocaine. (Both numbers are estimated by the same basic methodology.) Reducing a $148 billion-per-year problem by a net present value equivalent of 0.3 percent is worth about $450 million per year. That is almost equal to the value of the resources consumed in providing a model school-based prevention program to all students (about $550 million per year).

A comparable calculation for cigarette use is complicated because of the nature of smoking's effects.[2] For example, Manning et al. (1989) find that the external costs smokers impose on those outside their families are fairly small, partly because they die younger than most people and thus receive fewer government benefits during old age. However, Rice et al. (1986) did estimate the economic costs of the health effects of smoking to be $53.7 billion in 1984. (Crime costs are small for smoking.) That is the equivalent of $72.5 billion in 1992 dollars. Reducing an annual cost of that magnitude by a net present value equivalent of 0.5 or 0.6 percent is worth about $400 million per year.

For smoking we can also very roughly estimate the cost per year of life saved, which is a metric often applied to health promotion efforts. Smoking kills about 400,000 Americans a year, reducing life expectancy by the equivalent of about 5 million life-years per year (Kleiman and Caulkins, forthcoming). If prevention cuts smoking by the equivalent of 0.5 or 0.6 percent in net present value, that could be

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[2]We are not aware of any comparable estimates of marijuana's social costs that would permit an analogous calculation for that drug.

taken roughly to imply a total of the equivalent of 25,000 to 30,000 life-years saved annually. Dividing that into the cost of the prevention program ($550 million per year) gives an average cost of about $20,000 per life-year saved. Health interventions are sometimes considered to be cost-justified if they save a life-year at a cost of $50,000 or less (Owens, 1998).

Thus, assuming the mid-range effectiveness estimates, a model school-based prevention program can be or can nearly be cost-justified by focusing just on its smoking reduction benefits or its alcohol consumption reduction benefits alone. And, as found above, it can easily be justified based on its cocaine reduction benefits—again, assuming the mid-range effectiveness estimates. The total benefits in terms of substance abuse reduction is the sum of these three sets of benefits plus the benefits of reduced use of marijuana, heroin, etc.

BENEFITS UNRELATED TO REDUCED DRUG CONSUMPTION

In the preceding sections, we have limited ourselves to reductions in drug consumption or benefits arising from decreased drug use, e.g., lower health costs and crime rates, higher productivity. Drug use prevention programs may have other benefits, however, that are not so closely related to their primary objectives. As mentioned in Chapter Three, a course that strengthens the resistance skills and perceived self-efficacy of adolescents may also dissuade them from associating with gangs, from getting pregnant, and from other behaviors potentially injurious to their health or economic prospects.[3] It might also indirectly promote desirable outcomes such as high school graduation and behaviors promoting health. It is conceivable that such benefits could be at least as valuable to society as the reductions in drug use we estimate in this report. Some (e.g., Lynskey, 1998) have thus advocated that drug prevention messages be embedded within a broader prevention framework.

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[3]In asserting that these behaviors are not the primary targets of drug use prevention programs, we do not mean to suggest that there is no connection between drug use and such behaviors. Drug use has been shown to be associated with violence and with other problem behavior such as getting into trouble with the police or running away from home (Ellickson, Saner, and McGuigan, 1997; Hays and Ellickson, 1996).

NATIONWIDE IMPLEMENTATION

In the preceding chapters we have addressed the effectiveness and cost-effectiveness of drug-use prevention per program participant or per dollar or million dollars spent. Policymakers, of course, would also be interested in the total effect achievable if the program were widely implemented.

By nationwide implementation, we envision that every youth in the United States would receive a prevention program whose effects and costs are those described in Chapters Two and Three. That is, we assume there are neither "diminishing returns" nor "economies of scale" beyond those reflected in the scale-up qualifier (factor 8). Such linear extrapolation makes more sense here, when expansion involves parallel implementation in additional places, than it would for other drug control interventions whose expansion would entail increasing intensity within a given jurisdiction.

Chapter Three noted that giving every youth in the United States a model school-based drug prevention program is not at all cost-prohibitive. Even when we include teacher's salaries and the amortized cost of school facilities, administering a drug prevention program uses only about $150 in resources per program participant. So, assuming birth cohorts of 3.75 million per year, nationwide implementation would cost about $550 million per year—a little less than 1.5 percent of the $40 billion or so spent annually on drug control. In this chapter, we briefly consider some of the implications of such a program—for the current cocaine epidemic, for future drug epidemics, and for the debate over drug legalization.

101

IMPLICATIONS FOR THE CURRENT COCAINE EPIDEMIC

Could a $550 million prevention program dramatically alter the future course of drug use in the United States? We don't think so. According to Table 4.1, our point estimate of prevention's effectiveness is 5.4 percent of what the cohort receiving the prevention program would have consumed, or, without the discounting, 10.6 percent. Certainly a 10.6 percent reduction in U.S. cocaine use would be most welcome, but it would not alter dramatically the complexion of the drug problem as a whole.

Furthermore, the 10.6 percent reduction would not be observed for some time. First of all, there is a lag between the beginning of a prevention program (we discount all costs to participants' age 12) and the time the prevented cocaine use would have occurred. (The median age of cocaine use is almost 30.) The discount factor reflects this lag. Since the discount factor (factor 4) was 0.507, discounting at 4 percent per annum, this 10.6 percent reduction manifests sufficiently far in the future that it is worth only about half as much today.

There is another, even more important, reason, however, why implementing a model prevention program nationwide would not affect national drug consumption quickly. The 10.6 percent reduction is a reduction in consumption attributable to those who received the program. With or without a prevention program, most cocaine consumed in the United States over the next 10 to 20 years will be used by people who are already too old to receive a typical school-based prevention program. The effect of national implementation of prevention on use in 20 years would be the average of a roughly 10 percent reduction in use by those who are 12 or younger today and a 0 percent reduction in use by those who are already over 12.

For a variety of reasons, we cannot calculate this average exactly. The most obvious reason is that we do not know what baseline initiation rates will be in the future, so we do not know how many new users there will be relative to the number of persisting current users. We also do not have good data on how much, in grams, cocaine users consume by year over the course of their careers. Recall that in calculating the discount factor we focused on years of use, not grams used. Likewise, we do not have explicit models of the timing of the

indirect effects of prevention, those mediated through the two multipliers.

However, for any given assumption about what future cocaine initiation rates would be, we can estimate trends in the *number* of cocaine users over time, assuming that the indirect effects manifest alongside the direct effects. To be more precise, we can estimate the number of people over time who would report past-year cocaine use in the NHSDA. From that, we can project the percentage decrease in that NHSDA tally that accumulates as more of the population has gone through a model prevention program. (Methodological details are in Appendix J.) Figure 5.1 shows the results for nationwide implementation of a model prevention program in 1992, assuming baseline cocaine initiation rates of 17.5 percent for birth cohorts beginning in 1970.[1] The three curves correspond to the low, middle, and high estimates of prevention's effectiveness in factor 3.

As suggested, even with the most optimistic estimates of prevention's effectiveness, prevention will not dramatically alter the course of the current cocaine epidemic. Furthermore, even though prevention can eventually reduce the number of users by a nontrivial amount, that effect is far from immediate. With the middle estimate of prevention's effectiveness, it would take a nationwide model prevention program six years to achieve a 1 percent reduction in the number of past-year cocaine users relative to a no-prevention baseline. It would take 10 years to see a 2.5 percent reduction, 20 years to achieve a 5 percent reduction, and 40 years to see a 7.5 percent reduction.

IMPLICATIONS FOR FUTURE EPIDEMICS

One possible response to the preceding analysis is to conclude that America missed its chance to make a big difference via prevention. Perhaps school-based programs should now be implemented nationwide, anyway, because their cost-effectiveness even in terms of reducing cocaine use may not be bad, and, as we saw in Chapter

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[1]This initiation rate assumption is not a prediction or forecast. It is a number arbitrarily chosen out of a range of possibilities, consistent with experience, to help illustrate what nationwide prevention might accomplish.
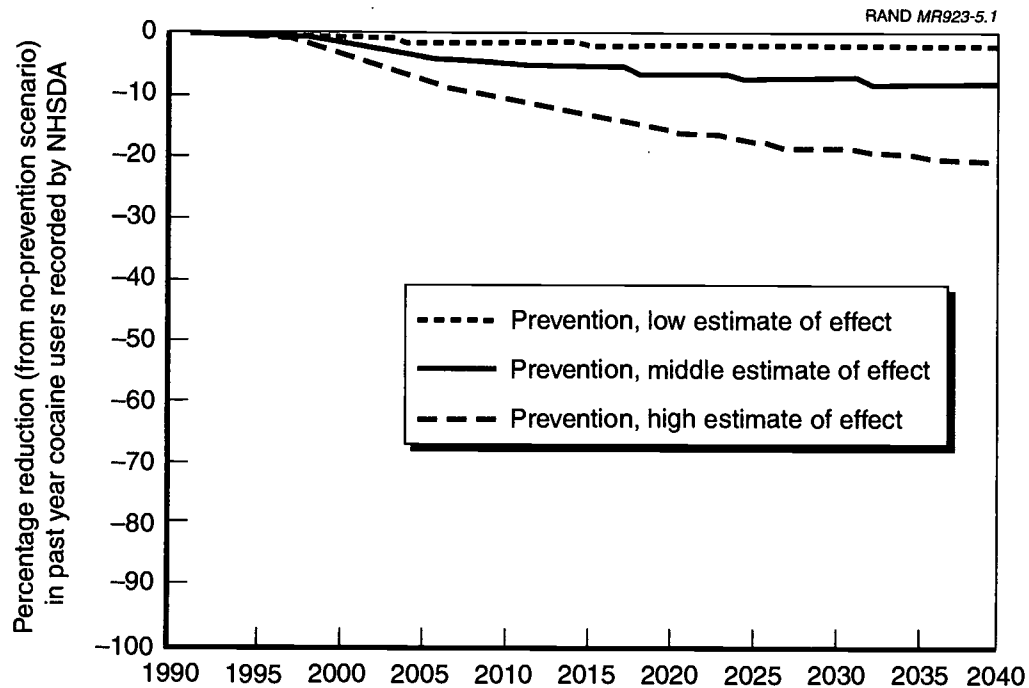
Figure 5.1—Prevention Will Only Modestly Reduce the Number of Cocaine
Users over a Period of Decades

Four, they produce other benefits as well. However, we should not
expect such an expansion to dramatically affect America's drug
abuse problems, even though it might have if it had been done 35 or
40 years ago.

That line of reasoning may sell prevention short, however. As Musto
(1987) points out, drug epidemics are not one-time events. There are
cycles or waves of drug use, with periods of high rates of initiation
separated by periods of low rates. It is too late to prevent the current
cocaine epidemic, which peaked in the late 1980s, but there may well
be another epidemic brewing. Indeed, drug use among youth has
risen substantially since 1992 (see Monitoring the Future statistics in
Johnston, O'Malley, and Bachman, 1996; and in *NIDA Notes,* 1997).

If a prevention program needs to be run ahead of a drug use epi-
demic to be successful, how far ahead does it need to be run? A drug
epidemic may not receive the national recognition required to moti-
vate action against it until some years after the peak in initiation,

when prevention is most needed (U.S. cocaine initiation peaked in 1979; it was recognized as a national crisis around 1984). It would take a few years for a national program to be devised, for funds to be appropriated by lawmakers and to reach agencies that implement prevention programs, and for those programs to be fully implemented nationally. That brings the total lead time to perhaps eight years. But prevention programs must sometimes be administered well ahead of the initiation age. The average age of initiation of the drug of concern may also be eight or so years after the age targeted by prevention programs.[2] This implies that prevention must be done on the order of 15 years or more before a problem is widely recognized.

The ability to forecast trends in drug use, let alone initiation, is limited. This is a problem, because it takes time to develop and implement good prevention programs. So if the prevention program is held back until it is clear that a drug epidemic has started, it will be too late. But running prevention programs constantly, including during the lulls between drug epidemics, is costly; money will be wasted administering prevention programs to people who would not have initiated drug use in any event. Given that school districts operate on tight budgets, there is also the danger that after a few years of low rates of drug use, the prevention programs would be dismantled and, hence, not be in place when they are needed.

Quantitatively addressing this problem of when and how aggressively to run prevention programs at different stages of a drug epidemic and between epidemics is beyond the scope of this report. Analysts would need, at a minimum,

- more precise estimates of prevention's effectiveness than the broad confidence intervals we are able to provide

- quantitative estimates of the temporal variation of all eight factors in our model

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[2]For the current cocaine epidemic, the average age of initiation is about 21.5; in a future epidemic, people may very well initiate at a different age, particularly if the drug is not cocaine.

- a description of the hazard rate for the arrival of the next drug epidemic (i.e., the probability that the epidemic will strike in the next year, given how long it has been since the last epidemic)

- predictions of the epidemic's magnitude and anticipated social cost.

Qualitatively, however, we can make the following observation. As pointed out in Chapter Four, a program that helps youth resist peer pressure and the temptation to take the risk of trying drugs may also help prevent teen pregnancy, absenteeism, poor health habits generally, gang membership, and other perennial problems. Further evaluations of drug use prevention programs should test this hypothesis. If it turns out to be true, there may thus be good reason to sustain such programs regardless of the current state of drug use—so they will be in place when temptations to use drugs increase.

IMPLICATIONS FOR THE LEGALIZATION DEBATE

This analysis does not directly address drug legalization,[3] but our results do have negative implications for one commonly mentioned pro-legalization argument. A principal concern about legalization is that it would lead to substantially greater use. Some counter that if the money saved by not having to enforce the prohibition were used to fund drug prevention, that could offset any such increase in use.

This study shows that it would certainly be feasible to pay for cutting-edge drug prevention for all youth with the money saved by the enforcement reductions associated with legalization. However, it is very doubtful that such prevention could avert an increase in use.

As shown in Table 4.2, a state-of-the-art prevention program would tend to reduce discounted lifetime cocaine consumption of those in the program by between 2 and 11 percent. That is, the majority of the drug use that would have occurred without the prevention pro-

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[3]By drug legalization, we mean any regime in which the supply and use of cocaine for nonmedical purposes is legal for a significant share of the population, e.g., all those over age 21. Some of these comments apply to a decriminalization regime, as well—that is, one in which currently illicit drugs are still illegal but felony charges and prison sentences are replaced by misdemeanor charges and jail terms or probation.

gram would still occur with it. Indeed, because legalization would result in reduced cocaine prices, we would expect usage to increase. How far would prices have to drop to counter the 2 to 11 percent drop in use caused by prevention? Over the long run, a 1 percent drop in cocaine price is accompanied by an increase in consumption of about 1 percent (see Caulkins and Reuter, 1998). Thus, after legalization, a price reduction greater than \$2 to \$13 from the current \$110/pure gram would be enough to offset the prevention-related consumption drop.[4] And legalization would almost certainly result in a much steeper price reduction than that. Moore (1990) mentions \$5/gram as a postlegalization cocaine price, and even that may be high given prices in Colombia and the low transportation costs for legal commodities. Excise taxes can bring prices up some, but not by 2,000 percent without recreating black markets. Furthermore, there would be declines in the nondollar costs of using, such as the time to search for a willing seller. These would increase consumption further, as would legal marketing tactics and reduced stigmatization.

Moreover, as discussed above, there is a substantial lag between when prevention programs are run and when they affect use. Price changes affect use more quickly. Thus, the prevention programs would have to be run for a decade or more before legalization—not concurrent with it.

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[4]The upper end of the effectiveness range is, more precisely, 11.48 percent; to offset such a drop in use requires an 11.48 percent rise in price, and 11.48 percent of \$110 is \$12.63.

CONCLUSIONS AND POLICY IMPLICATIONS

In this chapter, we restate our central findings regarding the effectiveness of best-practice school-based drug use prevention programs. We then discuss the implications of these findings for drug use prevention as a matter of national drug control policy.

MODEL PREVENTION PROGRAMS APPEAR TO BE COMPETITIVE WITH ENFORCEMENT

The performance benchmarks from our previous studies are summarized in Figure 3.1. The various types of enforcement programs (source country control, interdiction, and domestic enforcement under different sentencing regimes) have cost-effectiveness ratios in the range of 10–60 kilograms (net present value) of cocaine consumption averted per million program dollars spent. Our estimates of a model prevention program's cost-effectiveness bracket much the same range. Treating heavy cocaine users remains the most cost-effective cocaine control strategy we have examined: It can avert a net present value of about 100 kilograms of cocaine consumption per million dollars spent.[1]

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[1]Treatment's calculated cost-effectiveness obviously depends on its performance parameters. The analysis that concludes that it averts about 100 kilograms of consumption per million program dollars spent assumes that most users (about 79 percent) stop using while they are actually in the treatment program. It also assumes that, as a result of treatment, a minority (about 13 percent) continue to abstain from use, or at least use at substantially reduced levels, subsequent to discharge from treatment.

108

Prevention achieves its competitive cost-effectiveness despite bringing about relatively small reductions in future cocaine use. It would reduce future use by only 3 percent to 23 percent of the amount that those receiving the program would have consumed with no intervention. And those reductions occur so far in the future that they are equivalent to a decrease of just 2 to 11 percent beginning today.[2] Thus, to the extent that prevention is cost-effective, it is not because it is so effective but because it is so inexpensive. For a given amount of money, prevention may be able to achieve as much reduction in cocaine use as enforcement.

GREAT UNCERTAINTY REMAINS ABOUT PREVENTION'S COST-EFFECTIVENESS

The uncertainty range for prevention is twice as large as that for enforcement programs and several times as large as that for treatment (see Table 3.4).[3] What is striking is that a large proportion of that uncertainty comes from factors rarely identified as pertinent to prevention's cost-effectiveness. Thus, people can reach dramatically different opinions after reading and trusting the same evaluations of classroom program trials. That is because there is abundant room for them to disagree over the following: the indirect effects on nonparticipants and enforcement, the meaningfulness of the correlation between marijuana initiation and cocaine consumption, the degree to which program efficacy is reduced with scale-up, and what definition of program cost is used. This suggests that greater reductions in the uncertainty about prevention's cost-effectiveness might be achieved if some of the research emphasis were switched to these often ignored factors.

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[2]The reductions are expressed as a percentage of what those receiving the program would have consumed even though some of the reduction is in use by those not in the program. These results assume current prevention programs are no better than those that were in place in the years we used to estimate our baseline initiation rates. That is probably a little too pessimistic about the quality of prevention programs in place today. Hence, the ranges given are a little too optimistic about what national implementation of a cutting-edge prevention program would accomplish.

[3]"Uncertainty range" here is the ratio between the 95th percentile and 5th percentile of a large number of cost-effectiveness estimates, each based on random draws from probability distributions of factors contributing to the cost-effectiveness calculation.

THE SOURCE OF BENEFITS IS NOT WHAT MIGHT BE EXPECTED

Some people might think that the drug-reducing benefits of prevention programs are primarily attributable to the programs' ability to prevent young participants from initiating drug use. Our research shows that this is a misleadingly narrow view. Only 38 percent of the reduction in cocaine consumption is in the form of reduced consumption by program participants. Forty-four percent comes from positive spillover to friends and associates of those in the program, and 17 percent comes about because reduced use by all these people shrinks the market, making enforcement against those who remain in the market more effective.

Furthermore, not all of the reduction in quantity consumed by people in the program is caused by reduced initiation. A substantial portion is associated with reductions in quantity consumed by those who do start using cocaine at some point. Thus, a prevention program that succeeds "only" in delaying initiation need not be considered a failed program.

And a program need not work for every participant for it to be effective in total. Our middle estimate of prevention's effectiveness is generated from reduction of merely 4.1 percent[4] in program participants' cocaine use. Assuming a baseline initiation rate of 20 percent and a class size of 30 students, this is equivalent to persuading one student in every four classes not to try cocaine—or getting four to reduce their consumption by 25 percent. Yet that middle effectiveness estimate results in a cost-effectiveness on the same order as that of most enforcement programs, because prevention programs are so much cheaper (even with the costs of teachers' salaries and amortized facility costs included).

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[4]This result comes about by multiplying 7.6 percent from factor 3 times the two qualifiers.

DRUG USE PREVENTION HAS BENEFITS OTHER THAN REDUCED COCAINE USE

We have addressed the question of whether school-based prevention programs are effective when viewed as cocaine control programs. This is an important question from the perspective of allocating cocaine control resources. Nonetheless, viewing such prevention programs strictly as cocaine control programs underestimates their total value. Prevention programs can yield many benefits other than reduction in cocaine use, including reductions in use of other substances. For example, we estimate that school-based prevention could reduce marijuana use among program participants by about 1.8 percent (with a 90-percent confidence range extending from 0.6 to 3.4 percent). We estimate that prevention has a smaller proportionate effect on cigarette and alcohol use (0.5 percent and 0.3 percent, respectively), but because problems with those drugs are so severe, even small reductions can be valuable. Furthermore, prevention may also have benefits ancillary to reduced drug use.

A NATIONAL PROGRAM IS AFFORDABLE BUT WILL NOT END THE COCAINE EPIDEMIC

In addition to the marginal cost-effectiveness of school-based prevention, we are also interested in the characteristics of a full-scale program: What would happen if every child in the United States received cutting-edge prevention classes? First, it is clear what would not happen: It would not bankrupt the country. National-scale drug prevention would cost only about $550 million per year, about 1.5 percent of current national drug control spending.[5]

Second, it is clear that even full-scale implementation of a model drug prevention program would not eliminate or even greatly reduce the cocaine problem in the United States. Even the undiscounted percentage reductions in future use would be just 3 to 23 percent.

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[5]We are not suggesting here that 1.5 percent of current drug spending might be reallocated to prevention. We are primarily interested in affordability relative to total drug control spending and the effects of a national prevention program. It is pertinent here, however, that from time to time new money becomes available, e.g., the billion dollars to be spent over the next five years on the media initiative announced by President Clinton in July 1998.

Reductions in use of marijuana, alcohol, and cigarettes would be smaller still, in percentage terms.

Finally, for the next few decades, much of the nation's cocaine use will inevitably be by people who are already too old for school-based prevention programs. It will thus take many years for the reductions in consumption generated by school-based prevention programs to show up in national drug use figures. With our middle estimates of prevention's effectiveness, nationwide implementation would take 10 years to reduce the number of past-year cocaine users by 2.5 percent, 20 years to reduce it by 5 percent, and 40 years to achieve a 7.5 percent reduction, all relative to a no-prevention baseline.

Thus, prevention cannot be "the" solution to our current drug problem. Indeed, the notion that perhaps enforcement merely needs to "hold the line" until prevention can "cut the legs out from under the epidemic" does not seem to be a realistic strategy for dealing with a problem that is, at this point, more endemic than epidemic.

However, prevention should not be dismissed because it cannot "do it all." Prior research has found that other interventions, such as treatment and interdiction, cannot individually solve America's cocaine problem, either (see Rydell and Everingham, 1994, and Reuter, 1992, respectively). Indeed, frustration may be the only result of attempts to "solve" the problem of illicit drugs if solve is defined as eradicate. Concerted efforts have failed to eradicate other forms of criminal behavior (robbery, assault, etc.). An alternative would be to construct policies that attempt to contain and manage the problem. A medical analogy may be apt here. Smallpox was a problem that could be (and was) eradicated; heart disease will never be eradicated but it can be managed. The drug problem seems more like that of heart disease in this respect, and prevention can play a role in managing the drug problem even if it cannot "solve" it.

DRUG PREVENTION SHOULD BE CONDUCTED BEFORE IT IS PERCEIVED NECESSARY

Another reason for the lag in prevention's effect is the long delay between when a prevention program is run and when it begins to affect initiation and use. This, coupled with the fact that drug epidemics are not always identified as being severe until after initiation has

peaked, has implications for using prevention effectively to stave off a brewing drug epidemic. In particular, it suggests that it is best to begin prevention programs about 15 years before it is clear that there is a serious drug problem that should be, or should have been, prevented. One possible response to this problem is to run prevention programs continually, even when there is no evidence of a pending epidemic. Another alternative would be to invest in improving the early warning signs of an impending epidemic, thus reducing the delay factor (but not fully eliminating it).

PREVENTION CANNOT SUBSTITUTE FOR ENFORCEMENT IN A LEGALIZATION REGIME

While we have not conducted a study of drug legalization, our findings bear on this issue. A principal concern about legalization is that, because it would reduce the costs of drug use, greater consumption would ensue. Some counter that these increases in use could be offset if the money saved by not having to enforce prohibition were used to fund drug prevention. Our results suggest that hope regarding such an offset is unrealistic, even if our most generous, undiscounted estimates of prevention's effectiveness should prove true.

THE BOTTOM LINE

As a broad approach to addressing problems of all types, prevention has such intrinsic appeal that it is with some disappointment that we arrive at this conclusion: In the case of cocaine control, an ounce of prevention may not be worth a pound of cure, or at least we won't know until a pound of uncertainty is cleared away. We hope that by providing an analytical structure and by highlighting the unknowns, we have brought that eventuality a little closer.

Meanwhile, some conclusions can be drawn on the basis of our current knowledge. The bad news for prevention enthusiasts is that prevention does not appear to be the hoped-for silver bullet. It is not likely that with current technology, prevention can play a decisive role in eradicating our current drug problems.

On the positive side, national implementation of cutting-edge prevention programs is clearly affordable. And it appears that preven-

tion is both cost-competitive with enforcement as a way for the United States to deal with the cocaine problem and cost-justified in the sense that the social benefits obtained exceed the program costs.

Finally, decisions about prevention should not be made only with an eye toward ameliorating the current cocaine epidemic. Prevention could also be viewed as "lending a hand" with the current drug epi- demic and possibly with other undesirable social trends, while simultaneously serving as a form of "cheap insurance" against possible future epidemics.

114

ESTIMATING AVERAGE LIFETIME COCAINE CONSUMPTION

The principal performance metric in this research is the *weight* (e.g., in kilograms) of cocaine consumption averted per million program dollars. The prevention literature allows us to estimate reductions and delays in the *number* of initiations into cocaine use and, hence, the *number* of cocaine use careers. To bridge the weight and number, in this appendix we estimate the average weight of cocaine consumed over the course of one typical career of cocaine use. There is no one best way of estimating average lifetime cocaine consumption, so a variety of different methods are employed, yielding a range of estimates.

HISTORICAL CONSUMPTION DIVIDED BY NUMBER OF USERS

Conceptually the correct way to estimate average lifetime consumption would be to follow a large number of users throughout their entire history of use, sum their total consumption, and divide by the number of users. Something like this can be done by dividing Rydell and Everingham's (1994) estimates of national cocaine consumption from 1962 through the end of 1991 (4,116.3 metric tons) by their estimate of initiation over the same period (24.34 million). The result is 169.1 grams.

However, the lifetime consumption of all people who initiated between 1962 and 1991 is equal to the total quantity of cocaine consumed in those years only after making two adjustments to the latter

number. One must subtract the consumption between 1962 and 1991 of people who initiated before 1962 and add the consumption after 1991 of people who had initiated by then and who had not yet quit. In other words, if we let X = average lifetime consumption:

$$X = \frac{\text{Consumption Between 1962 and 1991} - \text{Adjustment \#1} + \text{Adjustment \#2}}{\text{Number of People Who Initiated Between 1962 and 1991}}.$$

The first adjustment is easy to make if we assume that everyone who was already using cocaine in 1962 completes their lifetime consumption by 1991. Under that assumption, every individual who was a light user at the beginning of 1962 consumes X grams between 1962 and 1991. (Because the Everingham and Rydell demand model is Markovian, expected lifetime consumption and the expected subsequent lifetime consumption of a current light user are the same.)

As we shall soon see, the subsequent lifetime consumption of a heavy user is 5.56 times that of a light user. Hence, if L_{1962} and H_{1962} represent the numbers of light and heavy users at the beginning of 1962, then the quantity in Adjustment #1 is $(L_{1962} + 5.56H_{1962})X$.[1]

By parallel reasoning, if L_{1992} and H_{1992} represent the numbers of light and heavy users at the beginning of 1992, then the quantity in Adjustment #2 is $(L_{1992} + 5.56H_{1992})X$. Inserting these expressions into the equation above and using the notation,

C_t =   consumption in year t and

I_t =   initiation in year t

yields

$$X = \frac{\sum\limits_{t=1962}^{1991} C_t}{\sum\limits_{t=1962}^{1991} I_t + \left(L_{1962} + 5.56H_{1962}\right) - \left(L_{1992} + 5.56H_{1992}\right)}.$$

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[1]Because the consumption of light and heavy users differs so greatly, it will be useful to differentiate them, as Rydell and Everingham did.

Everingham and Rydell (1994) estimate that $L_{1962} = 0.33$, $H_{1962} = 0$, $L_{1992} = 5.6$, $H_{1992} = 1.72$,

$$\sum_{t=1962}^{1991} C_t = 4{,}116.3 \text{ metric tons,}$$

and

$$\sum_{t=1962}^{1991} I_t = 24.34 \text{ metric tons}$$

—all in millions—and, thus, that X is 433.0 grams.

Parallel reasoning can be applied to numbers of years of light and heavy use. However, before doing so, it will be useful to work up expressions for the needed variables by trying another approach to estimating lifetime cocaine consumption.

CONSUMPTION PROJECTED THROUGH A MODEL OVER AN INFINITE HORIZON

Everingham and Rydell (1994) posit a Markov model of cocaine use, from which we can calculate the expected number of years of light and heavy use over an individual cocaine consumer's career. Using the notation

$j(S)$ = expected number of future years of light use for someone who is now in state S,

$h(S)$ = expected number of future years of heavy use for someone who is now in state S,

where $S \in \{L, H\}$ is the state indicator, with L and H indicating light and heavy use, respectively, and

a = annual rate of flow of light users into nonuse,
b = annual rate of flow of light users into heavy use,
f = annual rate of flow of heavy users into light use, and
g = annual rate of flow of heavy users into nonuse,

we can write the following equations:

$$j(L) = 1 + (1 - a - b)j(L) + b\,j(H)$$
$$j(H) = (1 - f - g)j(H) + f\,j(L)$$
$$h(L) = (1 - a - b)h(L) + b\,h(H)$$
$$h(H) = 1 + (1 - f - g)h(H) + f\,h(L).$$

If we solve these algebraically using the parameter values from Everingham and Rydell of $a = 0.15$, $b = 0.024$, $f = 0.04$, and $g = 0.02$, we get

$$j(L) = (f + g)/(af + ag + bg) = 6.329$$
$$j(H) = f/(af + ag + bg) = 4.219$$
$$h(L) = b/(af + ag + bg) = 2.532$$
$$h(H) = (a + b)/(af + ag + bg) = 18.354.$$

Since initiates are assumed to start as light users, this means that the expected career involves 6.329 years of light use and 2.532 years of heavy use. Consumption per year under base rate conditions is estimated to be 16.42 grams and 118.93 grams for light and heavy users, respectively (Everingham and Rydell, 1994), implying an average career of cocaine use involves 405.0 grams of cocaine consumption.

A side calculation allows us to estimate the ratio of future consumption for a current heavy user to the future consumption for a current light user as

$$(16.42\,j(H) + 118.93\,h(H)) \div (16.42\,j(L) + 118.93\,h(L)) = 5.56.$$

CONSUMPTION PROJECTED THROUGH A MODEL OVER A FINITE HORIZON

The previous estimate is too high because it extrapolates the Markov model out into the infinite future. In reality, as users get older, the Markov model breaks down. Past work (such as Rydell and Everingham, 1994 and Caulkins et al., 1997) used a 15-year evaluation horizon. For treatment programs, that meant extrapolating the Markov model 15 years beyond the age at entrance into the program—which

occurred only after commencement of heavy use. For enforcement programs, it was 15 years whether use was initially light or heavy, but since the majority of use was attributable to heavy users, the implicit assumption was not very different from that for treatment. In the case of prevention, benefits start at the age of cocaine initiation. We know the latter is 21.5 years, while the average of entrance into treatment is 32 years. Thus, extrapolating to an age 15 years beyond entrance into treatment is similar to extrapolating to an age that is 15 + 32 − 21.6 = 25.4 years beyond initiation. If we numerically project numbers of light and heavy use years over a 25- or 26-year horizon and linearly interpolate between the two to get an estimate for 25.4 years, we get the figures shown in Table A.1, including an estimate of average lifetime cocaine consumption of 291.93 grams.

We need to keep in mind that all the calculations based on the Everingham and Rydell model have converted years of light and heavy use to grams consumed using the average consumption rates in 1992. Those rates were influenced by the price of cocaine at the time. Prices had been much higher before the 1980s. Since consumption responds to price, cocaine consumption careers that included those earlier years most likely involved less consumption, on average, than these calculations suggest.

However, it is appropriate to use the figures we do—16.42 and 118.93 grams per year of light and heavy use—for two reasons. First, we are most interested in estimating the quantity of cocaine that would be consumed by someone starting to use it today. Is it appropriate to assume that the current situation will prevail? We certainly do not know what cocaine prices will be in the next few decades, so price effects cannot be factored in directly. But, particularly given the gen-

Table A.1

Finite-Horizon Lifetime Consumption Estimates from the Markov Model

Time Horizon (years)	Years of Light Use	Years of Heavy Use	Lifetime Consumption (grams)
25	5.99	1.61	289.54
26	6.01	1.65	295.52
25.4	6.00	1.63	291.93

eral stability of prices in the 1990s, projecting a future like the present is not altogether unreasonable. Second, a central goal of this research is to compare prevention's effectiveness to that of other cocaine control programs, and those programs were evaluated as they operated in 1992. Thus, we should base our estimates of prevention's effectiveness on these same assumed rates of consumption per year.

ADJUSTING THE HISTORICAL APPROACH FOR YEARS OF USE

Let's return to the historical approach. We adjusted it for quantities consumed before 1962 and after 1991, expressed in grams. Now, using the equations from the Markov model, we will adjust it for quantities consumed outside the period, expressed in years of use. We begin with

$$\sum_{t=1962}^{1991} I_t j(L) + \left(L_{1962} - L_{1992}\right)j(L) + \left(H_{1962} - H_{1992}\right)j(H) = \sum_{t=1962}^{1991} L_t$$

and

$$\sum_{t=1962}^{1991} I_t h(L) + \left(L_{1962} - L_{1992}\right)h(L) + \left(H_{1962} - H_{1992}\right)h(H) = \sum_{t=1962}^{1991} H_t.$$

The ratios of $j(L)$ to $j(H)$ and $h(L)$ to $h(H)$ are $(f + g)/f = 1.5$ and $b/(a + b) = 0.1379$, respectively, from the infinite horizon equations above. Everingham and Rydell (1994) estimate that

$$\sum_{t=1962}^{1991} L_t = 119.73$$

and

$$\sum_{t=1962}^{1991} H_t = 19.25,$$

yielding estimates that $j(L) = 6.68$, $h(L) = 2.92$, and lifetime cocaine consumption averages 456.9 grams.

STEADY-STATE ESTIMATE

An alternative approach is more empirical. We could observe both consumption and initiation in one year. If the cocaine system were in a steady state, then dividing the first of those two numbers by the second would give the expected lifetime consumption. Obviously the cocaine system is not in a steady state; indeed, drug epidemics are called epidemics in part because they are dynamic. Nevertheless, this calculation is easy to make and may offer circumstantial evidence about average lifetime consumption.

Rydell and Everingham (1994) estimate that in 1992, U.S. national cocaine consumption was 291 metric tons. Johnson et al. (1996, p. 36) estimate that 547,000 people started using cocaine in 1992. So the steady-state assumption would yield an estimate of 532.0 grams consumed over a lifetime of use.

SUMMARY

Table A.2 summarizes the estimates derived above. Considering the strengths and limitations of these approaches, we set our low, mid-range, and high estimates as follows. Given that 169 grams is almost certainly too low but that we have a 292-gram estimate that we think is unbiased, we set a low plausible estimate at 225 grams. We have three estimates falling into a fairly narrow band from 405 to 457 grams, so those must carry some weight in deciding on a mid-range "best" estimate. Thus, we set the mid-range estimate at 350 grams, roughly halfway between the no-known-bias 292-gram figure and the cluster of three estimates over 400 grams. Because the highest figure of 532 grams is methodologically the weakest, we set the high estimate just above the cluster of three at 475 grams.

Table A.2

Alternative Estimates of Average Lifetime Cocaine Consumption

Method	Estimate of Average Lifetime Consumption (in grams)
Consumption over historical period, based on grams, unadjusted	169
Consumption over historical period, based on grams, adjusted for use outside period	433
Consumption over historical period, based on years of use, adjusted for use outside period	457
Infinite-horizon Everingham & Rydell model	405
Finite-horizon Everingham & Rydell model	292
Steady-state assumption	532

122

PREVENTION'S EFFECTIVENESS AT REDUCING
MARIJUANA INITIATION

This appendix estimates the effect of generic, best-practice school-based prevention programs on initiation into marijuana, cigarette, and alcohol use by drawing on information about two state-of-the-art school-based drug prevention programs: Project ALERT (Ellickson and Bell, 1990a) and the Life Skills Training Program (Botvin et al., 1995). Project ALERT provided 11 classroom lessons, 8 primary lessons in the seventh grade, and 3 booster lessons in the eighth grade. The program reduced drug use in junior high (Ellickson and Bell, 1990a), but its effect decreased over time. By the end of high school, drug use by the prevention group was as high (or higher) than in the control group (Ellickson, Bell, and McGuigan, 1993). In contrast, the Life Skills program provided almost three times as many classroom lessons, over a longer period of time. Of the 30 total lessons, 15 were in the seventh grade, 10 in the eighth grade, and 5 in the ninth grade. The greater effort contributed to drug use reductions lasting to the end of high school (Botvin et al., 1995).

The effect of these programs is large enough, and the sample sizes are large enough, for the studies to conclude that the programs have statistically significant effects on drug use. However, the variance in people's behavior is great enough, and the sample sizes are small enough, for the uncertainty associated with point estimates to be very large. We therefore base our estimates on data from a variety of measures of use.

123

Our goal is to discover how much a prevention program reduces initiation into marijuana use during junior and senior high school years. Ideally, we would simply obtain original data on prevalence rates by year. Unfortunately, Project ALERT did not collect such data for the ninth through eleventh grades, and we were unable to obtain the data for the Life Skills program. Hence, we need to interpolate effects on initiation rates from published data on Project ALERT's effects through eighth grade (Ellickson and Bell, 1990a, Tables 2–4) and Life Skills' effects through twelfth grade (Botvin et al., 1995, Table 2).[1] Data on Project ALERT's effects through twelfth grade (Ellickson, Bell, and McGuigan, 1993, Table 1) are included for comparison, but are not used in our calculations because Project ALERT lacked the booster sessions necessary for effects to persist. Life Skills data for outcomes through eighth grade are reported only in terms of an average of an ordinal scale and so cannot be used here.

Table B.1 reports on several measures of use (e.g., lifetime, monthly, weekly) for each intervention and, in the case of Project ALERT, for three risk groups defined by experience with the substances at baseline. The Life Skills evaluation had two experimental groups; we take the experimental effect to be the simple average of the effects in the two groups, relative to the control group. For example, 27 percent of the control group, 23 percent of the first experimental group, and 21 percent of the second experimental group smoked cigarettes on a weekly basis, so we take the measure of reduction in weekly cigarette use to be –18.5 percent ((22 percent – 27 percent)/27 percent). For monthly marijuana use, the reduction is 1/14 (from 0.14 for controls to 0.13 for the experimental groups). For weekly marijuana use, the reduction is 1/3 (from 0.09 to 0.06).

The Project ALERT data are reported by risk category. For example, the marijuana use risk categories are having already used marijuana at baseline (category 3), having used cigarettes but not marijuana at baseline (category 2), and having not used either at baseline

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[1]We use the "Full Sample" not the "High-Fidelity Sample" from the Life Skills program for two reasons. First, it might be more representative of how large-scale implementation of prevention programs would perform. Second, it is possible that unobserved variables (e.g., quality of school administration) could be causally related both to high fidelity in program implementation and to lower rates of drug use for reasons beyond the drug prevention program itself.

Table B.1

Percentage Difference Between Program Recipients and Controls[a] for Various Measures of Drug Use in Eighth Grade

| | Percentage Differences | | | Risk Groups Counted[b] | |
	Life Skills	ALERT, 8th Grade	ALERT, 12th Grade	ALERT, 8th Grade	ALERT, 12th Grade
Used marijuana					
Daily			11.3%		2, 3
Weekly	−33.3%	−18.0%	6.7%	3	1, 2, 3
Monthly	−7.1%	−5.8%	−3.3%	2, 3	1, 2, 3
Past month		−20.3%	1.6%	1, 2, 3	1, 2, 3
Past year			0.2%		1, 2, 3
Ever		−4.9%	0.0%	1, 2	1, 2
Used cigarettes					
Pack/day	−20.8%				
Daily		−1.6%	6.8%	2, 3	1, 2, 3
Weekly	−18.5%	−7.9%	7.1%	2, 3	1, 2, 3
Monthly	−19.7%	−0.7%	6.3%	2, 3	1, 2, 3
Past month		−2.0%	8.0%	1, 2, 3	1, 2, 3
Past year			5.7%		1, 2, 3
Ever		−4.3%	4.3%	1	1
Used alcohol					
3+ drinks per occasion	−5.1%				
Drunk	−16.3%				
Weekly	−8.6%	2.2%	−7.0%	2, 3	1, 2, 3
Monthly	−1.7%	−5.4%	−4.8%	2, 3	1, 2, 3
Past month		−2.1%	−0.7%	1, 2, 3	1, 2, 3
Past year			1.6%		1, 2, 3
Ever		−4.1%	1.6%	1	1

[a]Percentage of program recipients using, minus percentage of controls using, divided by percentage of controls using, times 100 percent.

[b]For marijuana use, risk groups are described by the column heads in Table B.2, left to right; for cigarettes and alcohol, groups 1, 2, and 3 are nonusers, experimenters, and users at baseline, respectively. Those who had initiated at baseline are omitted from the estimation of reduction in percentage having ever used. Omissions for other variables result mainly from Project ALERT's not reporting use percentages when they fell below a specified threshold (2.5 percent for eighth graders and 2 percent for twelfth graders).

(category 1). For measures of any use to date, we excluded individuals who had already initiated at baseline from the calculations because it would be impossible for a prevention program to affect their lifetime prevalence. For other measures, we computed a simple av-

erage of the results for the program's two experimental variants and weighted the risk categories in proportion to their share of the population. Table B.2 illustrates the calculations for marijuana use in the previous month in the eighth grade.

Table B.1 shows that the effects of Project ALERT, which did not have as many booster sessions as the Life Skills program, had eroded by twelfth grade. Hence, we estimate program effects through eighth grade based on Project ALERT and through twelfth grade on the Life Skills data. Even omitting the twelfth grade program effect column for Project ALERT, however, the data are anything but consistent.

These measures of use do not, for the most part, pertain directly to initiation, which is the risk factor we are using.[2] Neither can we derive a complete grade-specific profile of changes in initiation from the use data shown. This is because there are many patterns of

Table B.2

Estimation of Project ALERT Effect on Marijuana Use in Past Month (in eighth grade)

| | Risk Group | | | |
	Nonusers at Baseline	Cigarette Users at Baseline	Marijuana Users at Baseline	Total
Size of group	1976	1344	554	3874
Control prevalence	3.7%	13.6%	43.8%	
Treatment group #1 (teen leader) prevalence	2.9%	11.1%	37.6%	
Treatment group #2 (health educator) prevalence	1.4%	10.1%	39.1%	
Average treatment prevalence	2.15%	10.6%	38.35%	
Number who would be using under experimental conditions	42.48	142.46	212.46	397.41
Number who would be using under control conditions	73.11	182.78	242.65	498.55
Percentage change	–41.9%	–22.1%	–12.4%	–20.3%

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[2]We use age of initiation because that is available for all respondents to the NHSDA. If we focused on other aspects of adolescent use, we would be able to use data only on NHSDA respondents who were adolescents at the time of the survey. This would yield a much smaller sample size and, even more important, make it impossible to know those individuals' lifetime cocaine consumption.

changed initiation that would be consistent with the observed changes in use in eighth and twelfth grades. So, we make the considerable simplifying assumption that the effect on initiation is the same in all grades.

The only direct estimate of the reduction in marijuana initiation is the 4.9 percent reduction in lifetime prevalence in the Project ALERT data from eighth grade among those who had not already used marijuana before they received the prevention program. (Marked in bold on Table B.1.) We take that as one of our effectiveness estimates.

For our second estimate, we take the average effect across evidence from both studies and as many measures of marijuana use as possible (other than the 4.9 percent above). In particular, we want to use both Project ALERT (through eighth grade) and Life Skills data. The average of the resulting five measures (marked in bold in Table B.1) is –16.9 percent.[3]

We round these two estimates off to get our low and high estimates of effectiveness and take their average as our middle estimate. Thus, for our low, base, and high estimates of the effect of a cutting-edge prevention program on marijuana initiation in junior and senior high school, we use –5 percent, –11 percent, and –17 percent, respectively.

We have not yet addressed what happens to initiation beyond the time for which there is evaluation data. There is no empirical information on what effect the school-based prevention programs might have on initiation after high school. The two extreme possibilities are

- that the initiation reductions during junior and senior high school are merely postponements of initiation to the post–high school years

- that the initiation reductions in the junior and senior high school years are permanent.

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[3]It could be argued that because we are searching for an upper bound, we should average the measures from the more successful study or even pick the best single measure. However, because these measures are indirect, we think it makes more sense to consider them all in inferring initiation.

For the first possibility, we assume that the reductions in marijuana initiation during ages 12–17 are delayed until ages 18–21. We assume that initiation in each of the four post–high school years increases by a common percentage that is sufficient to make the cumulative initiation through age 21 the same as it would have been without the prevention program. Under the second possibility, the reductions are permanent, so that each year after high school the initiation proportions are the same with prevention and without. An intermediate possibility is that one-half the initiation reductions result in initiation postponement until after high school and one-half are permanent.

To summarize, our image of a model school-based prevention program's effect on marijuana initiation is as follows:

- It reduces marijuana initiation by 11 percent during the junior and senior high school years (with 5 to 17 percent being the range for sensitivity analysis).

- 50 percent of these reductions are permanent, as opposed to being offset after high school (with 0 to 100 percent being the range for sensitivity analysis).

Figure B.1 shows the result of these assumptions for the middle estimate. It shows the percentage of a cohort that starts using marijuana at each age. The light bars show initiation with school-based prevention. The dark bars show initiation without school-based prevention. The difference between the light and dark bars in Figure B.1 is graphed in Figure B.2.
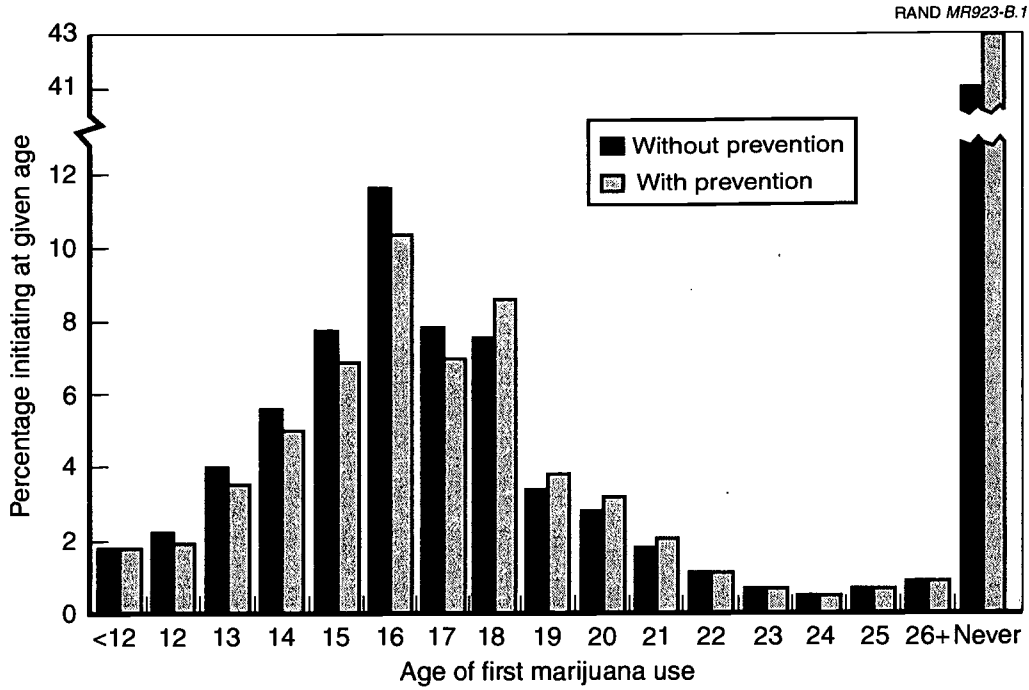
Figure B.1—Proportion of Cohort First Using Marijuana at Different Ages
(assumed distribution of middle estimate)
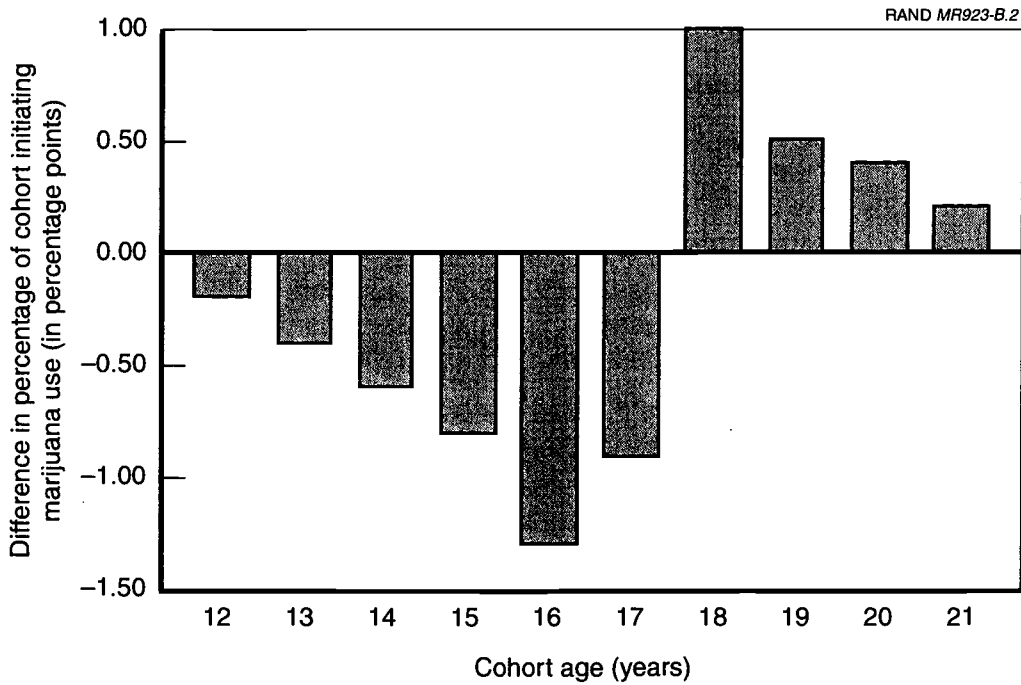
Figure B.2—Difference Between Percentage Initiating Marijuana Use With
and Without Prevention

RELATIONSHIP BETWEEN AGE OF MARIJUANA INITIATION AND LIFETIME CONSUMPTION OF COCAINE AND MARIJUANA

In this appendix, we show how we calculate the reduction of lifetime cocaine consumption associated with delaying (or preventing) marijuana initiation. We do not take into account here the two qualifiers (factored in later). We also sketch the parallel calculations for reduction of marijuana consumption.

The first steps in the calculations for cocaine consumption are embodied in Table C.1. The table's first column (the column of row labels) identifies the value of the risk factor—age of marijuana initiation. The second column gives the NHSDA's historical distribution of people across this risk factor. It is computed for people who were 30–34 in 1992, old enough to have initiated marijuana if they were going to, but young enough to have grown up in times when marijuana use was relatively common. (See Chapter Two for further explanation of this choice of age range.) The third column shows how this distribution would change if the cohort were exposed to a prevention program that reduced initiation in junior and senior high school by 11 percent, with half of that reduction being permanent.

Columns four and five show the 1991–1993 NHSDA data on the proportion of people who ever use cocaine and how often the average user used it in his or her life. The number of times used was taken from the NHSDA's lifetime cocaine consumption variable, coded as described in Table C.2.

130

Table C.1

Lifetime Cocaine Consumption by Age of Marijuana Initiation

Age of Marijuana Initiation	Proportion of Cohort Starting Marijuana		Proportion of Cohort Ever Using Cocaine	Mean Number of Times Used, Undiscounted	Discount Factor	Mean Number of Times Used, Discounted
	Without Prevention	With Prevention				
11 or younger	0.018	0.018	0.583	55.840	0.559	31.215
12	0.022	0.019	0.597	49.440	0.549	27.133
13	0.040	0.035	0.550	43.860	0.532	23.341
14	0.056	0.050	0.460	28.700	0.523	15.018
15	0.077	0.068	0.427	24.650	0.502	12.365
16	0.116	0.103	0.359	14.920	0.497	7.410
17	0.077	0.069	0.343	14.740	0.472	6.950
18	0.075	0.086	0.299	7.470	0.464	3.468
19	0.033	0.038	0.270	8.390	0.458	3.847
20	0.027	0.031	0.197	5.740	0.413	2.369
21	0.017	0.020	0.204	9.250	0.396	3.662
22	0.011	0.011	0.183	8.880	0.382	3.397
23	0.006	0.006	0.226	3.230	0.432	1.395
24	0.004	0.004	0.188	4.650	0.362	1.681
25	0.006	0.006	0.232	6.560	0.351	2.303
26 or older	0.008	0.008	0.132	3.790	0.339	1.286
Never	0.407	0.428	0.004	0.100	0.466	0.047
Total (all ages)						
Without[a] prevention	1.000		0.220	52.6		26.7
With[a] prevention		1.000	0.208	51.6		26.1

[a]Numbers in columns 2 and 3 are sums. Numbers in other columns are averages weighted according to distribution in column 2 (without prevention) or 3 (with prevention).

Table C.2

Coding of the NHSDA's Lifetime Cocaine Consumption Variable (COCTOT)

COCTOT Value in NHSDA	Meaning in NHSDA	Recoded to Be
1	1 or 2 times	1.5
2	3 to 5 times	4
3	6 to 10 times	8
4	11 to 49 times	25
5	50 to 99 times	74.5
6	100 to 199 times	149.5
7	200 or more times	300
81–98	Bad data, imputed, etc.	0

In principle, since we are investigating lifetime use, we should restrict our attention to people who have completed their cocaine using careers. Last use can never be known for certain before death, but a reasonable proxy might be to count people who have ever used cocaine but not in the last 12 months. Unfortunately, that restriction substantially reduces the sample size.

Another approach would be to restrict attention to those who initiated cocaine use at least 12 years earlier. The reasoning here is that if they were ever going to consume quantities of real significance they would have done so within 12 years of initiation. That approach also reduces the sample size.

We compared estimates of the relation between marijuana initiation and cocaine consumption based on these two approaches and on simply including all people between 30 and 34 in 1992 who had ever used cocaine, even if they had begun use recently. The results were quite similar and highly consistent. We use the results for all who ever used cocaine because those results are the least variable.

Of course, including people who are early in their cocaine use careers would bias downward estimates of lifetime consumption. However, it is not obvious that it affects the relationship between age of marijuana initiation and lifetime cocaine use. We would expect such an effect in two circumstances. The first circumstance is if, by a certain age, the percentage of lifetime cocaine consumed varied with lifetime cocaine consumption and initiation age. For example, suppose that earlier initiators with higher lifetime use consumed a lower per-

centage of their cocaine by age 30 than did later initiators with lower lifetime use. Truncating careers would then omit more of early initiators' lifetime consumption and result in an underestimate of the correlation between marijuana initiation age and cocaine use. However, data on lifetime cocaine use profiles are available and do not vary sufficiently with marijuana initiation and total lifetime cocaine consumption for career truncation to much affect the correlation.

The second circumstance is if the distribution of users over initiation ages were different in recent years than it was earlier. If ages of initiation have been getting younger, then estimates of lifetime use will be underestimated the most for younger ages of initiation. But there is little evidence that has happened to any appreciable degree. If we regress the average age of cocaine initiates on time for 1973–1992 data reported by Johnson et al. (1996), the slope is only –0.01; the difference between that number and 0 is not statistically significant. There is also not a great deal of variability in the average age of cocaine initiates. Over this period, the minimum average age was 20; the maximum was 23.

Returning to Table C.1, the sixth column is the discount factor. It estimates the ratio of the net present value of lifetime cocaine consumption (discounting at 4 percent back to age 12, i.e., seventh grade) to undiscounted lifetime consumption, by age of marijuana initiation. These discount factors were computed based on years of cocaine use as follows. We created a table of the currently consuming population by current age and marijuana initiation age. That is, for each marijuana initiation age (e.g., 17), we put down the number using cocaine at each subsequent age (i.e., at 18, 19, 20, etc., for initiation age 17). Then, we created a table of population ever having consumed cocaine by current age and marijuana initiation age. We divided the numbers in the first table by those in analogous cells from the second table. This yielded, for each marijuana initiation age, a retention rate—i.e., the percentage of those ever using cocaine who are still using it, at each subsequent age. We then discounted each of those percentages to age 12. By adding up these discounted percentages for each marijuana initiation age and dividing them by the undiscounted sum for that age, we got our discount factor for that age. Properly speaking, this is the discount rate for *years* of cocaine use, not for the *quantity* of cocaine used. If heavy use tended

to cluster early or late in a cocaine career, that would bias the discount factor; however, this bias is unlikely to be significant.

The seventh column in Table C.1, the discounted consumption, is just the product of the undiscounted consumption and the discount rate. (Note that the age-specific discount factors and the aggregate factor given in Table C.1 are based on a 4 percent discount rate; as explained in Chapter Two, 2 percent and 6 percent rates were also used to explore the sensitivity of our results to variations in the rate.)

We next sum the entries in the prevalence and in the undiscounted- and discounted-consumption columns (columns four, five, and seven), weighted by the appropriate risk factor distribution (column two for no prevention, column three for prevention). This gives the changes in the key variables of interest. This information is summarized in Table C.3. Ultimately, what we are looking for is the discounted lifetime cocaine consumption, which is the change in the discounted consumption per person (–0.445 gram) divided by the discounted consumption per person in the absence of prevention (5.879 grams). It is the bold number in Table C.3 (–7.57 percent).

This change comes about from three factors: The change in prevalence of cocaine use, the change in users' average lifetime consump-

Table C.3

Effect of Prevention on Lifetime Cocaine Consumption Measures

	Without Prevention	With Prevention	Difference[a]	Percentage Effect[b]
1. Proportion who ever use cocaine	0.220	0.208	–0.012	–5.47%
2. Lifetime consumption per user	52.6	51.6	–1.0	–1.95%
3. Discounted lifetime consumption per user	26.67	26.08	–0.59	–2.22%
4. Implied average discount factor	0.5070[c]	0.5056[c]	–0.0014	–0.28%
5. Discounted lifetime consumption per person	5.879[d]	5.434[d]	–0.445	–7.57%

[a]Equals data column 2 minus data column 1.

[b]Equals data column 3 divided by data column 1, times 100 percent.

[c]Equals row 3 divided by row 2.

[d]Equals row 1 times row 2 times row 4.

tion, and the change in the ratio of discounted to undiscounted consumption (i.e., the effects of delaying consumption further into the future).   The contributions of these three factors are given in the rightmost column of Table C.3 and are displayed graphically in Figure C.1.

All of the numbers in Table C.1, Table C.3, and Figure C.1 pertain to a prevention program that reduces marijuana initiation by 11 percent in junior and senior high school, with 50 percent of that reduction being permanent. Changing either or both of those characteristics of the prevention program (the 11 percent and 50 percent) to their low or high estimates affects the third column in Table C.1 and, hence, all the numbers calculated directly or indirectly from that column. Figure C.2 summarizes the bottom-line effect on discounted lifetime cocaine use (number in bold in Table C.3) for all nine combinations of low, middle, and high estimates of both the reduction in marijuana initiation and its permanence.

The highest estimate of prevention's effect on a cohort's lifetime cocaine consumption (13.6 percent) occurs with maximum reduction

RAND *MR923-C.1*
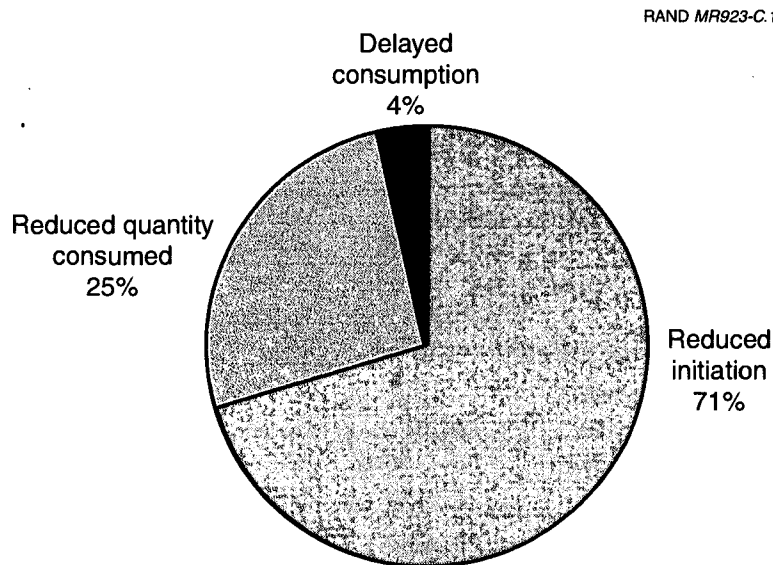
Figure C.1—Sources of Prevention-Induced Reduction in
Cocaine Consumption by Program Participants

in marijuana initiation, all of which is assumed to be permanent. The lowest estimate of 2.9 percent occurs with minimum reduction in marijuana initiation, none of which is assumed to be permanent. The middle estimate, resulting from the middle estimates of both dimensions, is 7.6 percent. We take these as our high, low, and middle estimates of prevention's effect (unqualified) on lifetime cocaine use by people in the program.

We were also interested in the effect of prevention on marijuana use. To derive that, we performed calculations similar to those for cocaine. Table C.4 and Figure C.3 are parallel to Table C.1 and Figure C.2. Parallel calculations of the discount factor give low, middle, and high estimates of 0.445, 0.552, and 0.718, respectively.
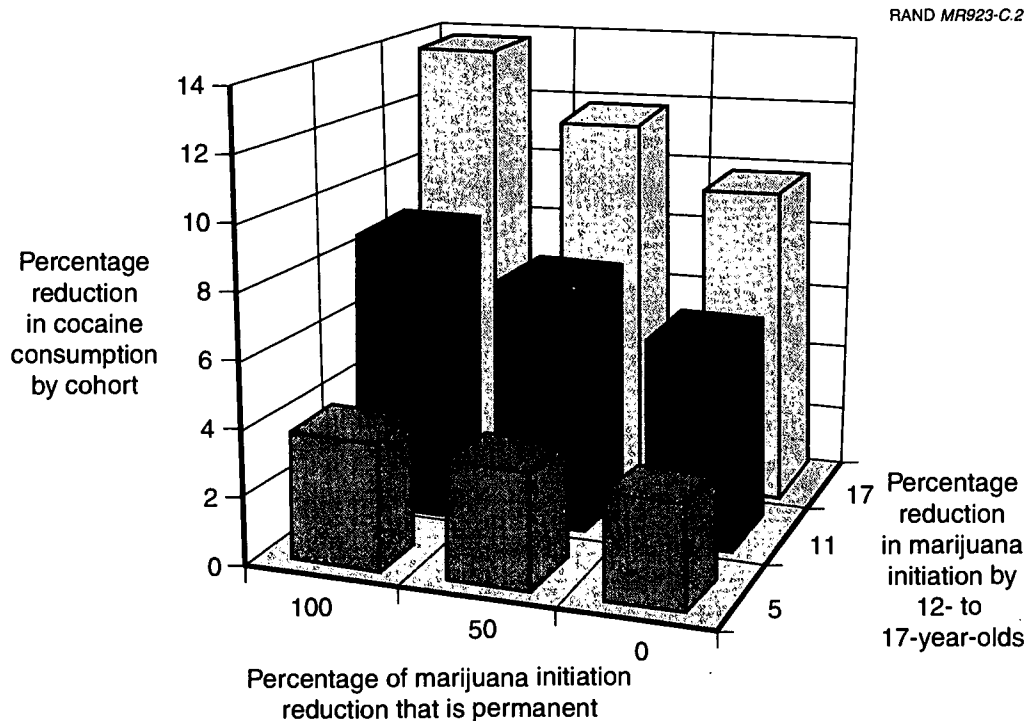
Figure C.2—Reduction in Cohort's Cocaine Consumption for Different Assumed Values of Marijuana Initiation Reduction and Permanence

Table C.4

Lifetime Marijuana Consumption by Age of Marijuana Initiation

Age of Marijuana Initiation	Proportion of Cohort Starting Marijuana		Proportion of Cohort Ever Using Marijuana	Mean Number of Times Used, Undiscounted	Discount Factor	Mean Number of Times Used, Discounted
	Without Prevention	With Prevention				
11 or younger	0.018	0.018	1.000	177.530	0.590	104.659
12	0.022	0.019	1.000	155.200	0.616	95.634
13	0.040	0.035	1.000	138.630	0.570	78.959
14	0.056	0.050	1.000	113.170	0.626	70.895
15	0.077	0.068	1.000	100.080	0.516	51.649
16	0.116	0.103	1.000	73.490	0.589	43.271
17	0.077	0.069	1.000	67.100	0.453	30.384
18	0.075	0.086	1.000	62.170	0.504	31.360
19	0.033	0.038	1.000	55.560	0.553	30.714
20	0.027	0.031	1.000	35.940	0.544	19.545
21	0.017	0.020	1.000	39.230	0.438	17.170
22	0.011	0.011	1.000	46.460	0.491	22.795
23	0.006	0.006	1.000	26.580	0.535	14.221
24	0.004	0.004	1.000	42.850	0.385	16.491
25	0.006	0.006	1.000	32.610	0.415	13.525
26 or older	0.008	0.008	1.000	18.970	0.386	7.330
Never	0.407	0.428	0.000	0.000	NA	NA

NA is not applicable.

137
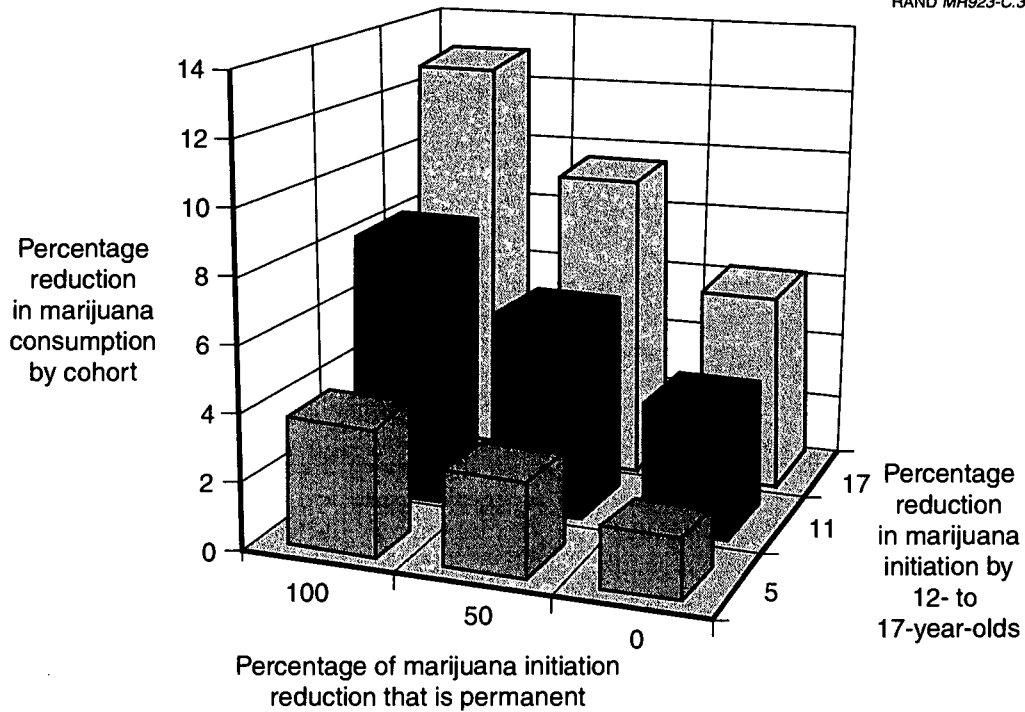
Figure C.3—Reduction in Cohort's Marijuana Consumption for Different
Assumed Values of Marijuana Initiation Reduction and Permanence

ESTIMATING THE MAGNITUDE OF THE SOCIAL MULTIPLIER

To the extent that initiation into drug use is a contagious or epidemic phenomenon, preventing one person from initiating may reduce future consumption by more than that one individual would have consumed. Consider an example in which the first person's initiation would have led through various personal interactions to, ultimately, two other people initiating. The consumption averted by preventing the first initiation would be expected to be about three times the average lifetime consumption of any one individual. We call this ratio of total initiations prevented to primary initiations prevented the "social multiplier." In the example just mentioned, its value would be three. In this appendix, we show how we estimated the low, middle, and high values given in Chapter Two.

COMPUTING THE SOCIAL MULTIPLIER FOR A GIVEN INITIATION FUNCTION

Assume that, within a model of the dynamics of cocaine use (e.g., Everingham and Rydell, 1994), an expression has been established that determines initiation as a function of other model variables, such as the numbers of light and heavy users. It is then easy to compute the social multiplier through a four-step process:

1. Run the model into the future.

2. Rerun the model while exogenously reducing initiation (i.e., forcing it down from outside the model) in a particular year, call it

year 0, by one person.[1] Initiation is a function of model variables that are themselves influenced by past initiation rates; among those are interactions allowing for an influence by program participants on nonparticipants. Thus, we would expect a change in initiation in all years following year 0. Record the change in the outcome measure of interest (e.g., discounted quantity consumed, and discounted number of user-years) from the analogous measure obtained in step 1.

3. Rerun the model scripting initiation to be exactly as in step 1 except that initiation in year 0 is reduced by one person. Initiation in all other years is the same as in step 1 because the endogenous initiation function (i.e., that calculated within the model) has been replaced with a fixed, scripted initiation series. Interactions between the subtracted initiate and anyone else considering initiation are thus ignored. Record the change in the outcome measure of interest from that obtained in step 1.

4. The social multiplier is the change in the outcome measure in step 2 divided by that in step 3.

These calculations are straightforward. The challenge lies in trying to model how the variables affect initiation, a topic that takes up the rest of this appendix.

CAVEATS TO THE INTERPRETATION OF THE SOCIAL MULTIPLIER

Three comments should be made before proceeding with the estimation of the initiation functions. First, since initiation is, in general, a function of other model variables, the magnitude of the social multiplier also depends on the values of those variables in the year in which an initiation is exogenously prevented. Hence, the social multiplier's value will be different at different points in the epidemic, as the values of the variables upon which initiation depends change. For purposes of developing a point estimate of prevention's cost-effectiveness, we focus on the value of the social multiplier in 1992,

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[1]Initiation rates are typically in the hundreds of thousands to millions per year. To avoid problems with numerical precision, the calculations were actually done with an exogenous reduction of 10,000 initiations in year 0, not one initiation.

but we also investigate the relative magnitude of the multiplier at different points in the cocaine epidemic. In Appendix E, we combine the latter information with data on how cohort sizes have varied to estimate how the cost-effectiveness of prevention interventions might have changed over the course of the epidemic.

Second, in estimating prevention's effects on cocaine consumption, we use demonstrated program effects on marijuana initiation to estimate differences in cocaine consumption, then apply a social multiplier to the latter. That is, the multiplier pertains to interactions among cocaine users only. We ignore any extramural multiplier effects from the program-induced reduction in marijuana initiation; we assume that programs reducing marijuana initiation have no unmeasured indirect effect on marijuana initiation by nonparticipants. This seems reasonable since entire cohorts of students in schools received the prevention program, and presumably a large number of the social contacts of those who received the program are with others within that school and cohort. Say the prevention program convinced one youth participating in the prevention program not to use marijuana. Two other classmates were not influenced by the prevention program but decided to abstain because of the first youth's abstinence. The program evaluation would detect the abstinence of all three youths, not just the one who was directly affected by the prevention program. To the extent that some of the marijuana multiplier "spills over" outside the school, ignoring the marijuana social multiplier has led us to underestimate prevention's cost-effectiveness. If, for example, there were a marijuana multiplier of 1.25, our estimate of prevention's cost-effectiveness is only 80 percent (1/1.25) of the true value (assuming all other factors are correctly estimated).

Finally, we couch our discussion of the social multiplier in terms of prevented initiations averting other initiations. It is also possible that exogenously preventing an initiation will reduce the length or intensity of someone else's career of drug use through these same social interaction mechanisms. That could be true even if it does not prevent the second person from initiating. We have no way of estimating such effects and are forced to assume that they are reflected in the social multiplier calculated on the basis of initiation.

INTRODUCTION TO MODELING ENDOGENOUS INITIATION

Modeling of endogenous initiation functions is discussed in Behrens et al. (1998). The two key dynamics are identified by Musto (1987) and elaborated by Kleiman (1992). First, most people do not start using a drug spontaneously. Instead, they are introduced to the drug by a friend or family member. Hence, all other things equal, the more current users there are, the higher one would expect initiation to be. Light users may be the most dynamic proselytizers because, on average, they are relatively recent converts to drug use themselves and may not yet have experienced many adverse consequences of their use.

Second, over time, drugs create problems for users, and the drugs acquire a reputation for being dangerous. There is no direct measure of either drug reputation or problematic use in the Everingham and Rydell model, but generally speaking heavy users are more likely to manifest adverse outcomes associated with their use than are light users. Hence, all other things equal, the greater the "history" of heavy use, the lower one would expect initiation to be. The word "history" is used to connote a cumulative process; a reputation is acquired over time. But bad reputations are not immortal. Musto hypothesizes that cycles of drug use arise when the current generation of youth no longer remembers the adverse experiences of its forebears.

The basic outlines of how initiation depends on other model variables are thus fairly clear. The details, however, are not. In particular, what functional form best captures the relationship and what are its parameter values?

It is not possible to answer these questions experimentally. There are not even obvious opportunities for quasi- or natural experiments. In fact, the only relevant data are annual data for the current cocaine epidemic for the United States as a whole. Initiation is not measured at the city or state level or in smaller time intervals.

Even under the best of circumstances, the 30 or so data points in the U.S. annual time series would be a meager basis for estimating the endogenous initiation function, but there are other problems as well. In particular, there is good reason to believe that cocaine prices,

patterns of use of other drugs, and the existence and nature of drug prevention programs all influence initiation into cocaine. However, there are not adequate data to control for any of these, except perhaps in the most recent years, and restricting the analysis to those years would sacrifice so many data points that there would be a danger of overfitting. Hence, it must be recognized that the initiation functions estimated here are highly simplified, omitting relevant variables such as cocaine price and use of other drugs. Furthermore, they rest on a very thin empirical basis and may well be wrong both in detail (i.e., the specific parameter values) and in general (i.e., their functional form). Nevertheless, they represent the best estimates that can be made at this time, and the resulting estimates of the social multiplier, however tenuous, are probably better than ignoring its existence altogether.

The basic strategy for identifying the endogenous initiation function begins with hypothesizing functional forms. We then find the parameter values that minimize the sum of the squared errors between the resulting predictions of what initiation would have been and some data describing what initiation is believed actually to have been. Finally, we observe the quality of the fit.

There are two variants of this strategy, depending on what values of the epidemic variables (e.g., numbers of light and heavy users) are used to predict initiation. One approach is to use the Everingham and Rydell (1994) estimates of how the epidemic evolved. The other is to use whatever values are generated by the initial conditions, the endogenous initiation function in question, and the four flow parameters Everingham and Rydell (1994) use to describe transitions between use states. The results obtained with each of these two variants of the basic strategy are described in turn. In both cases, the initial numbers of light and heavy users in 1962 were taken to be 0.33 and 0.01 million, respectively.

FITTING WITH EXOGENOUS DATA ON NUMBERS OF USERS

Functional Forms Evaluated

Starting with the Musto hypothesis of how initiation is influenced by bad experiences with drugs, we hypothesized that initiation might be deterred by any of the five following measures:

1. Number of people who were ever heavy users: E
   $E(1962) = H(1962)$
   $E(y) = E(y-1) + b * L(y-1)$ for $y > 1962$,
   where b is the annual flow rate of light users into heavy use
   and $H(y)$ and $L(y)$ are the number of heavy and light users, respectively, in year y.

2. Decaying number of people who were ever heavy users:[2] ED
   $ED(1962) = H(1962)$
   $ED(y) = d * ED(y-1) + b * L(y-1)$ for $y > 1962$,
   with $d = 0.96$ initially.

3. Decaying number of heavy-user-years accumulated to date: HY
   $HY(1962) = H(1962)/(1-d)$
   $HY(y) = d * HY(y-1) + H(y)$ for $y > 1962$,
   with $d = 0.96$ initially.

4. Bad reputation, which we express as the fraction of consumption accounted for by heavy users: R
   $R(y) = 118.93H(y) \div [118.93H(y) + 16.42L(y)]$

5. Decaying bad reputation: RD
   $RD(1962) = 118.93H(y) \div [118.93H(y) + 16.42L(y)]$
   $RD(y) = (1 - c)RD(y-1) + c * R(y)$ for $y > 1962$,
   with $c = 0.2$ initially.

The parameters c and d determine how quickly memory of past experiences fades. In measure 5, reputation is set equal to a weighted average of the current year's reputation (defined in measure 4) and

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[2] ED and the following variables HY and RD are single variables expressed by two letters (i.e., ED is not $E * D$).

the preceding year's (which itself depends on previous years' reputations). The parameter c determines the weight—80 percent the preceding year's versus 20 percent the current year's, according to our initial assumption. The parameter d has the following interpretation. Assuming the length of a generation in a "generational forgetting" hypothesis is 25 years, we take $1 - d$ to be 0.04. The measure, decaying heavy-user-years, is then equal to the number of heavy users in the current year times 0.96 times the decayed heavy-user-years accumulated through last year (itself the result of an analogous sum).[3]

With these variables so defined, we evaluated two sets of functional forms for the initiation function. The first set were special cases of the following general form:

$$I = \tau + \alpha * L^{\beta_L} * H^{\beta_H} * E^{\beta_E} * ED^{\beta_{ED}} * HY^{\beta_{HY}} * R^{\beta_R} * RD^{\beta_{RD}}.$$

Specifically, we evaluated 42 models created by the following cross product: { $\tau = 0$ or was free to be other than zero} × { $\beta_L = 0$, $\beta_L = 1$, or β_L free} × {zero or one of the other six β's free and all the others zero}. In other words, the constant intercept was either zero or allowed to be free; light use was excluded, forced to enter linearly, or allowed to be free; and no more than one of the terms reflecting heavy use was included. That results in $2 \times 3 \times 7$, or 42, models.

The τ term has an intuitive interpretation in terms of diffusion models from the field of marketing that describe adoption of a new product. Those models often distinguish between "innovators" who adopt on their own and "imitators" whose adoption is influenced by the behavior of others. In this language, τ would reflect initiation by innovators.

For the second set of functions, we started with six factors of the form $(L/H)^{\beta_H}$, where H can stand for any of the measures of heavy use (H, E, ED, HY, R, and RD). For each of these six factors (represented by { }), we created three functions: $\alpha L * \{ \}$, $\tau + \alpha L * \{ \}$, and $(\tau + \alpha L) * \{ \}$. These were tried because with the first set of functions, when β_L was allowed to be free, it often took values around $1 - \beta_H$. These forms

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[3]The qualitative results turned out to be fairly robust with respect to the specific values of c and d for the functional forms and parameter values that produced the best fits.

also have intuitive appeal; they indicate that initiation is proportional to the number of light users modified by some reputation effect. They were labeled models #43a, #43b, #43c, . . . , #48a, #48b, and #48c.

Sets of Initiation Data to Which Models Were Fit

The endogenous initiation functions were fit to three different sets of historical initiation data:

1. The average of the difference estimate reported in Everingham and Rydell (1994) and Johnson et al.'s (1996) estimates based on the retrospective method for the years 1963–1991. Everingham and Rydell argue that an average of estimates generated by the difference and retrospective methods is superior to either method alone and use such an average in their report. However, the retrospective estimates Everingham and Rydell use to create the average estimate were based on fewer and older data than Johnson et al.'s figures. Hence the current estimate.

2. Johnson et al.'s (1996) initiation data for 1962–1992. This series was tried out of concern that the "plateau" in the difference method's initiation estimate (460,000 persons per year for every year from 1963–1971) might make it difficult to get the correct fit.

3. Johnson et al.'s (1996) initiation data for 1966–1992. This series was tried because estimates of initiation in the early to mid-1960s are inevitably rough.[4]

Conclusions Concerning the Endogenous Initiation Function

The parameters yielding the best fit were found with Microsoft Excel's Solver. The summary measures of goodness of fit reported here are the percentage of variation around the initiation mean that is explained (R^2) and the proportion of variation explained adjusting for

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[4]Models #29–#42 performed poorly with the first two sets of initiation data. (These were the models not accounting for the number of light users, i.e., those with the exponent of L set to 0.) We thus did not fit those models to this third set of data.

the number of parameters ($R_a{}^2$). These measures are borrowed from regression analysis. The analysis suggested the following:

- Forcing β_L, the exponent of L, to be zero gives very poor fits to the data.

- When τ is allowed to be nonzero, it generally is negative. That is inconsistent with the interpretation of innovators versus imitators described above and can lead to the nonsensical result of negative initiation for certain values of the other model parameters. Presumably this reflects overfitting of the data, and models for which τ was negative were ignored.

- At least one of the terms measuring heavy use must be included. For any given functional form, when the heavy-use term is omitted, the results are worse.

- The $R_a{}^2$ measures of goodness of fit are consistently higher (by an average of 0.15) when fitting the Johnson et al. initiation data.

- Otherwise the results are fairly similar when fitting to all three sets of initiation data.

Perhaps most important, if we exclude models with $\tau < 0$, quite a few have $R_a{}^2$ values around 0.88 to 0.90. Generally, though, the models with the best fit involve decaying number of heavy-user-years (HY), including (for the Johnson et al. 1962–1992 data) the models numbered as follows:

$$\#9 \qquad I = 0.244 * L * HY^{-0.169} \qquad (R_a{}^2 = 0.896).$$

$$\#23 \qquad I = 0.2065 * L^{1.138} * HY^{-0.2188} \qquad (R_a{}^2 = 0.900).$$

$$\#46a \qquad I = 0.177 * L * (L/HY)^{-0.2196} \qquad (R_a{}^2 = 0.900).$$

Those functions' fits are not terribly sensitive to the memory rate. The fit is about the same with d taking any value between 0.93 and 0.99 (i.e., "generation lengths" of between 14 and 100 years). It likewise is not terribly sensitive to $HY(1962)$; values between 0.01 and 2 (million) give $R_a{}^2$ above 0.89. The Johnson et al. initiation and functionally estimated initiation curves are shown in Figure D.1.
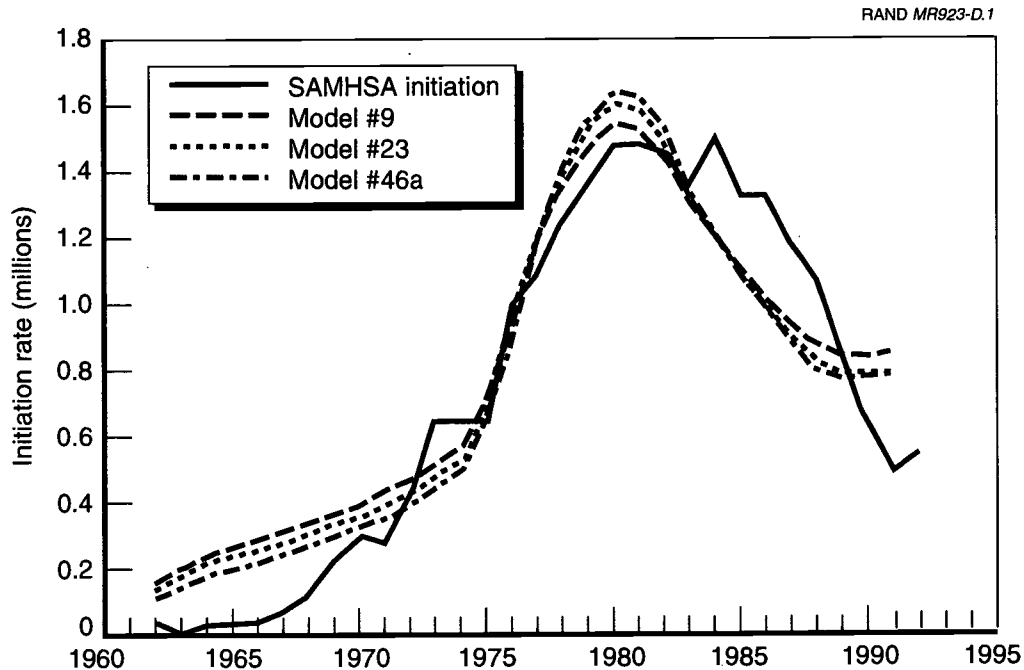
Figure D.1—Modeled Initiation Compared With Initiation Data from the
Substance Abuse and Mental Health Services Administration

There is also theoretical appeal to the decaying heavy-user-years
measure. If we focus on the current number of heavy users, then
when a heavy user exits the population, his or her contribution to
deterring initiation goes to zero immediately. That is, there is no
memory whatsoever of past bad experiences with heavy use, which
seems implausible. The reputation measure has a similar problem
because it is based entirely on the relative number of current light
and heavy users. At the other extreme, if we focus on the number of
people who were ever heavy users, then when people exit the heavy-
user population, it does not in any way diminish their contribution to
deterring initiation, which seems perverse. Decaying heavy-user-
years reflects a more reasonable, intermediate amount of memory.

Notwithstanding the theoretical appeal of focusing on decaying
heavy-user-years, many functions achieve roughly comparable fits.
Hence, it seems prudent to estimate the social multiplier for one of
each "class" of initiation functions, where a class is defined by the
measure of heavy use (i.e., H, E, ED, HY, R, and RD). In particular, we

performed the social multiplier calculations with the function from each class that gave the best fit to all years of the Johnson et al. data, omitting instances in which τ (number of innovators) was negative. That meant models #43a, #44a, #45a, #46a, #11, and #13 (parameterized using SAMHSA initiation, 1962–1992), each of which has just two parameters, as follows:

#11 $\qquad I = 0.131 * L * R^{-0.488}$ $\qquad (R_a^2 = 0.881)$

#13 $\qquad I = 0.117 * L * DR^{-0.566}$ $\qquad (R_a^2 = 0.882)$

#43a $\qquad I = 0.110 * L * (L/H)^{0.263}$ $\qquad (R_a^2 = 0.889)$

#44a $\qquad I = 0.123 * L * (L/E)^{0.245}$ $\qquad (R_a^2 = 0.895)$

#45a $\qquad I = 0.113 * L * (L/ED)^{0.266}$ $\qquad (R_a^2 = 0.893)$

#46a $\qquad I = 0.177 * L * (L/HY)^{0.220}$ $\qquad (R_a^2 = 0.900).$

Conclusions from the Resulting Social-Multiplier Calculations

Social multipliers were calculated from these initiation functions using the scheme laid out at the beginning of this appendix. They were calculated for four cocaine consumption outcome measures: net present value (NPV) of consumption (grams) over a 20-year, 30-year, and 40-year time horizon and NPV of heavy-user-years over a 20-year time horizon. (All discounting was done at 4 percent per year.) These calculations suggest the following conclusions:

- The longer the time horizon, the greater the magnitude of the multiplier. In particular, the 30-year-horizon multipliers are about 1.2 to 1.35 times as large as the 20-year, and the 40-year-horizon multipliers are about 1.35 to 1.6 times as large as the 20 year. That makes sense because multiplier effects accumulate.

- The 20-year multiplier based on heavy-user-years is consistently 15–20 percent smaller than the 20-year multiplier based on consumption.

- The magnitude of the multiplier decreases monotonically as a function of the year in which prevention occurred,[5] but not by an enormous amount. The multiplier in 1963 was 1.5 to 2 times that in 1992. By 1972 it was just 1.2 to 1.5 times that in 1992. Between 1982 and 1997, the multiplier declined by another 5 to 20 percent.

  The values in 1963 are very much a function of the assumption made about the number of heavy users at the beginning of the epidemic, which is a number that is not known with any precision. If it is higher than the assumed value of 10,000, there is less difference between the early and late multiplier values.

- Adding a $\tau = 0.1$ term to the initiation functions has only a modest effect on the multipliers. (The added term enables these endogenous initiation functions to reproduce the magnitude of the epidemic.)

- The 1992 multipliers for 20-year time-horizon effects on consumption for all six models were in the range of 2.0 to 2.5; for the 30-year time-horizon effects, they were in the range 2.3 to 3.1.

FITTING WITH ENDOGENOUSLY DETERMINED DATA ON THE NUMBERS OF USERS

In the second approach to estimating the endogenous initiation function, we took for the numbers of light and heavy users over time whatever values were generated by the initial conditions, the endogenous initiation in question, and the four flow parameters governing transitions between use states (Everingham and Rydell, 1994).

The functional forms evaluated were the first 28 in the first set as defined above (i.e., those in which L's exponent was not 0), except that where τ was allowed to be free before, it was now constrained to be nonnegative. Since τ was essentially never found to be positive, in some cases those models were not estimated.

The data to which they were fit were

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[5]This monotonic decrease is not a general result; it is easy to construct sets of initial conditions for which it would first increase then decrease.

- the average of the difference estimates reported by Everingham and Rydell and Johnson et al.'s retrospective estimates for 1962–1991

- Johnson et al.'s data for 1962–1992

- the average estimates for 1970–1991

- Johnson et al.'s data for 1970–1992.

Results of the analysis were as follows:

- Again τ was essentially never estimated to be positive.

- Fits were much better when the exponent of L was allowed to be free than when it was constrained to be 1.

- When the exponent of L was allowed to be free, the proportionality constant (α) became extremely small and the coefficient on heavy use tended to be very large in absolute value. This suggests overfitting. These results, however, were not observed when the heavy-use indicator was decaying heavy-user-years.

- The model achieving the best fit with all four sets of initiation data was

$$I = \alpha * L^{\beta_L} * HY^{\beta_{HY}}.$$

  However, the parameter values for that model were substantially different in each of the cases.

CONCLUSION

Given the theoretical arguments favoring decaying heavy-user-years as the measure of heavy use that deters initiation years, we are inclined to accept this last functional form as the best one. It is noteworthy that all β_L values are positive and all β_{HY} values are negative. Thus, initiation will be high (and prevention will have its greatest potential for effect) when the number of light users is high and the (decayed) accumulated number of heavy-user-years is low. There does, however, remain some question as to the specific parameter values. The latter are given in Table D.1.

Table D.1

Alternative Parameterizations of Preferred Initiation Model

P Number[a]	Initiation Data	Dates	Source of L & H Data	α	β_L	β_{HY}
P1	average[b]	1962–1991	E&R (1994)[c]	0.1906	1.6898	–0.8480
P2	SAMHSA	1962–1992	E&R (1994)[c]	0.206	1.138	–0.219
P3	SAMHSA	1966–1992	E&R (1994)[c]	0.223	1.100	–0.224
P4	SAMHSA	1962–1992	endogenous	0.1930	1.9093	–0.9901
P5	SAMHSA	1970–1992	endogenous	1.0141	1.4512	–1.2234
P6	average[b]	1962–1991	endogenous	0.1676	2.0246	–1.1980
P7	average[b]	1970–1991	endogenous	1.8279	1.4268	–1.6105

[a]Parameterization number.

[b]Average of difference and retrospective (SAMHSA) approaches.

[c]Everingham and Rydell, 1994.

It is not clear which of these seven parameterizations (referred to as P1 through P7, respectively) to prefer and, hence, which should be used as a basis for estimating the social multiplier. None of the seven gives even a reasonably good fit in all seven situations or contexts described in columns 2 through 4 of Table D.1, so we cannot prefer one over the others on that account.

Hence, we computed social multipliers for each of these parameterizations. The results for P5 and P7 are largely nonsensical: Those parameterizations could reproduce the epidemic starting in 1970, but when started in 1962, produced an explosion in use that is much, much larger and earlier than was actually observed. Two things were made clear by this exercise:

- *It is not possible to estimate the social multiplier at the beginning of the epidemic.* Parameterizations P4, P5, P6, and P7 suggest the social multiplier in the early years of the epidemic (1962, 1963) was huge, over 100. P1 suggests it was initially about 25 to 50; P2 and P3 suggest that it was between 4 and 9.

  There is such disagreement because for all the models, the social multiplier in the early years is extremely sensitive to the initial number of heavy users. Except for parameterizations P2 and P3, the multipliers seem to get arbitrarily large as the number of heavy users approaches zero. Hence, it is pointless to try to pin down the "correct" parameterization; uncertainty about the so-

cial multiplier generated by uncertainty about the number of heavy users in those years swamps that generated by uncertainty about which model is correct. We have only a very rough guess as to the number of heavy users there were in the 1960s, so even if we knew the exact initiation function, we still would not be able to estimate the social multiplier very precisely for those years.

- *The social multiplier in 1992 was probably between 1.0 and 2.9.* All of the parameterizations yield multipliers for 1992 that are between 1.0 and 3.6, regardless of which outcome measure is used.[6] (See Table D.2.) In Appendix A, we use a time horizon extending from the average cocaine initiation age to 15 years beyond the average age of treatment entry. That totals 25.4 years, so a simple average of the 20- and 30-year measures may make the most sense. Focusing on that average measure, the range shrinks slightly to 1.0 to 2.9; the shrinkage is modest because, with respect to the multiplier in 1992, most of the variation is

Table D.2

Social Multipliers in 1992 for Different Model Parameterizations

	Outcome Measure[a]			
	Grams Consumed over			Number of Heavy-User-Years over
P Number[b]	20 Years	30 Years	40 Years[c]	20 Years
P1	1.202	1.219	1.255	1.178
P2	2.520	3.092	3.528	2.148
P3	2.568	3.151	3.594	2.185
P4	1.482	1.516	1.654	1.428
P5	1.000	1.000	1.000	1.000
P6	1.138	1.145	1.187	1.125
P7	1.000	1.000	1.000	1.000

[a]All results are net present value.

[b]Parameterization number. See Table D.1.

[c]Beginning in 1991.

[6]The 40-year-horizon multiplier is for 1991, not 1992, since the epidemic was only projected out to 2031.

across parameterizations, not across definitions of the multiplier. That is the opposite of what is true when trying to estimate the social multiplier for earlier years. This range subsumes the range for the multipliers estimated earlier for these outcome measures. Hence, in the body of this report, we take the low, middle, and high estimates of the social multiplier in 1992 to be 1, 2, and 2.9, respectively.

HOW PREVENTION'S COST-EFFECTIVENESS VARIES OVER TIME

We calculate prevention's cost-effectiveness by dividing the product of the eight-factor, multiplicative model of effectiveness by program cost. Since some of the factors depend on the state of the drug epidemic and the state of the drug epidemic varies with the passage of time, the cost-effectiveness of prevention also varies with time. The data supporting our estimate of the various factors are for 1992, which we chose to facilitate comparison with parallel calculations for other drug control programs (e.g., interdiction) that had already been done using that base year. Our estimate of cost-effectiveness in Chapter Three thus applies to 1992. This appendix, in contrast, discusses how prevention's cost-effectiveness has varied over the course of the cocaine epidemic. We discuss in turn how each of the eight factors in the model has varied.

QUANTITY OF COCAINE CONSUMED OVER A USER'S LIFETIME

Prices influence consumption by influencing the quantity consumed by those who use (as well as the number of users). Prices vary over time (Caulkins, 1993), so the average lifetime quantity consumed presumably varies as well. In particular, cocaine prices collapsed during the 1980s and have remained much lower than they were in the 1970s. Hence, preventing a cocaine initiation in 1969 probably averted fewer grams of cocaine use, on average, than preventing a cocaine initiation in 1989.

The baseline estimate of prevention's cost-effectiveness assumes that the years of cocaine use prevented by a program implemented in 1992 would all have occurred under base year conditions—i.e., with prices at their 1992 levels. This is appropriate for two reasons. First, prices have been relatively stable since 1992, so projecting price stability at 1992 levels is not unreasonable. Second, our parallel work with other interventions evaluated their efficacy under comparable conditions.

BASELINE RATE OF COCAINE INITIATION

The importance of the initiation rate is most easily understood by considering that if no one initiates cocaine use in the absence of a prevention program, running a prevention program cannot possibly reduce cocaine use; the cost-effectiveness must inevitably be zero.[1] Conversely, under the assumption that a prevention program persuades a proportion of people who would have used drugs not to do so, the program will get the biggest bang for the buck if baseline rates of initiation are high. Cocaine initiation rose to a peak around 1980 and has fallen since. In particular, peak rates of initiation in the early 1980s were five times the rates in the early to mid-1960s and were double the rates in the early 1990s. These facts, together with the eight-year lag between prevention program participation and cocaine initiation, imply that, with respect to this factor, a model prevention program would have been substantially more effective if it had been run in the 1970s than it could possibly be now.

DIRECT EFFECTS OF PREVENTION PROGRAMS ON COCAINE CONSUMPTION

The proportional effect of prevention programs on cocaine consumption might have been different had the experimental programs been conducted in different years for at least two categories of reasons. First, a prevention program's effectiveness might be influenced by society's underlying attitudes toward drugs, or even its attitudes toward a method or style of intervention. For example, public

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[1]Theoretically it could even be negative if the prevention program stimulated curiosity about cocaine that induced someone to try cocaine who otherwise would not have.

opinion of the honesty and ethical standards of police has varied somewhat (Maguire and Pastore, 1996, p. 139), and such attitudes might influence the effectiveness of DARE. Similarly, after the Gulf War, prevention programs that featured military personnel may have had a particular power to affect the attitudes and behavior of youth (Caulkins et al., 1994).

Second, it is possible that prevention programs do not influence a given proportion of those who would otherwise have used, but rather a given number. For example, each time a teacher leads a prevention program, he or she might be able to influence some number of youths, regardless of how many would otherwise have used (as long as at least that number would have). However, this would seem more plausible for individual-based interventions than for programs run for entire classes; the literature commonly discusses the effectiveness of school-based programs in terms of proportions of youth whose behavior is changed.

We have no way of estimating the magnitude or even the direction of the effect of any such changes over time and generally do not expect the effects to be large.

DISCOUNT FACTOR

The discount factor could conceivably vary over time if the distribution of consumption over user careers changed. For example, people who are ever going to use cocaine heavily may reach such states more quickly after initiation now than they did 25 years ago—e.g., because prices are lower now or because of crack. If so, then the discount factor could be greater now than it was in the past. However, it does not seem likely that such changes are large. Also, it is not clear whether considering all such changes would tend to increase or decrease the discount factor over time. Finally, to estimate the discount factor, we use a Markov model of cocaine careers that is independent of year of initiation. This approach obviously precludes estimating how the relation between year of use and amount of use affects the discount factor.

SOCIAL MULTIPLIER

The social multiplier reflects ways in which current and past use influence initiation, so the social multiplier can certainly change over time. In particular, it tends to be greater when the ratio of light to heavy users is large, as would typically be the case early in an epidemic. This implies that prevention is more effective early in an epidemic than later.

In Appendix D, we estimated the magnitude of the social multiplier in 1992 with reasonable precision, but could not do the same for the early years of the cocaine epidemic. Hence, it is not possible to say precisely by how much prevention's cost-effectiveness diminishes over time because of decreases in the social multiplier. However, for all of the initiation functions considered, the ratio of the social multiplier in 1962 to 1992 was at least two and a half to one and in some cases was much larger. So this factor could well be quite important.

MARKET MULTIPLIER

The market multiplier is determined by the slope of the supply curve. This is fully explained in Appendix F. For our current purpose, it is sufficient to note here that the relevant supply curve is the long-run supply curve, because eliminating a cocaine using career affects demand over many years. In the long run, two opposing effects tend to keep the supply curve from being flat. The first is the familiar scarcity of factors of production, which—through diminishing returns—means that higher prices are necessary to call forth greater market quantities. The second is industrywide economies of scale through which expansion in the size of the market reduces the per-unit production costs; principal among these is the dilution of enforcement effort and, hence, the component of price per gram that reflects compensation for the risks of enforcement sanctions. Scarce factors tend to make unit production costs higher when market volume is large; industrywide economies do the opposite.

The base case analysis was done for 1992—a time in which

- the cocaine market was stagnant (Rhodes et al., 1995)

- few factors of production were in short supply (Caulkins et al., 1997)

- the market was large enough to have "swamped" enforcement so that punishment capacity constrained severity of punishment (Kleiman, 1993).

Hence, following Caulkins et al. (1997), conditions in 1992 were taken to imply a market multiplier greater than 1; in particular, its base case value was taken to be 1.36.

In all likelihood, the situation was different around 1980. As far as is known, the cocaine market was growing rapidly, so a variety of factors may have been constrained. Hence, the supply curve may have sloped upward, and the market multiplier may well have been less than one. Stronger statements are not possible because no time series describing the slope of the supply curve have been estimated. Nor are there sufficient data on how the industry cost structure has changed to replicate for other years the calculations in Caulkins et al. (1997), from which the base case estimates of the market multiplier are derived.

ADJUSTMENT FOR SPURIOUS CORRELATIONS

This factor is so poorly understood even in the base year that we can do little more than speculate about how it might or might not vary over time. Conditions under which it might vary include the following. Suppose some of the underlying behavioral characteristics that influence cocaine initiation also both influence marijuana initiation and are influenced by the prevention program. Suppose further that other factors that predispose people to use cocaine are neither influenced by prevention nor affect marijuana use. Finally, suppose that over time the relative importance of these two sets of characteristics changes in terms of ability to predict cocaine initiation. Then the adjustment for spurious correlation would change over time.

SCALE-UP ADJUSTMENT

We have no information concerning changes over time in the ability of school systems to expand model programs into large-scale interventions with high fidelity.

SUMMARY AND IMPLICATIONS

Although all eight factors just discussed may have varied to some degree over the course of the cocaine epidemic, the dominant variation is probably with proportion ever using cocaine and the social multiplier. The social multiplier would have been higher near the beginning of the epidemic, and the last of the cohorts with high baseline initiation rates were in junior high school in the early to mid-1970s. Hence, the cost-effectiveness of a model school-based prevention program would probably have been substantially higher if it were implemented between the epidemic's beginning and the mid-1970s than if it were implemented later, e.g., 1992.

To underscore this point, Figure E.1 plots

- the social multiplier for an average initiation function (divided by 10)[2]

- baseline cocaine initiation, shifted eight years earlier to reflect the average time lag between exposure to a school-based prevention program and cocaine initiation

- the product of those two variables, which is labeled "effectiveness multiplier."

The figure is meant only to be illustrative, because the exact nature of the initiation function is not known and the eight year shift in the baseline initiation curve is only a rough adjustment. Nevertheless, it clearly suggests that the contribution of these two factors to the cost-effectiveness of prevention was an order of magnitude higher between 1964 and 1973 than it was in 1983 and 1984. It is unlikely that the product of these two factors has increased much since then. The social multiplier continued to ebb, albeit slowly, and an increase in baseline initiation even to one million people per year would only roughly double the effectiveness multiplier.

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[2]Specifically, it is the average of the 20- and 30-year-consumption measures generated by averaging across all endogenous initiation functions discussed in Appendix D that were estimated with data going back into the 1960s.

RAND MR923-E.1
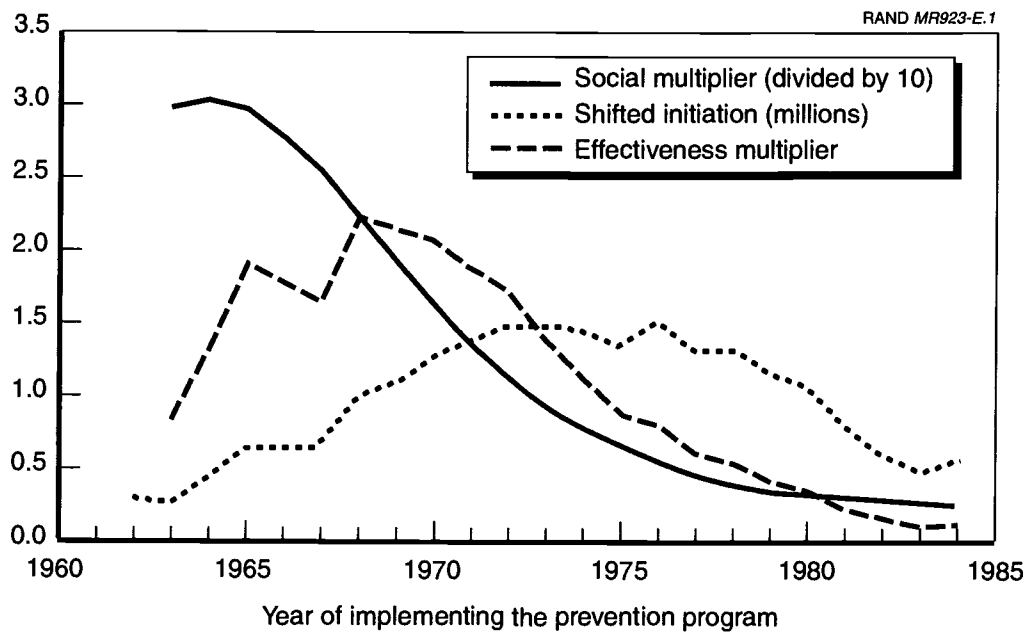
Figure E.1—How Effective a Given Prevention Program Would Have
Been over Time, As the Nature of the Epidemic Varied

ESTIMATING THE MAGNITUDE OF THE MARKET MULTIPLIER

For any commodity whose demand is not infinitely elastic (whether the commodity is licit or not) if the supply curve is not flat, then changing the consumption of one person affects the equilibrium price and, hence, the consumption of others. Depending on which direction the price moves, consumption by others may be increased or decreased. Thus, when an intervention, such as a prevention program, influences the consumption of one person, it might lead to a change in national consumption that is greater or less than the change in the first individual's consumption. We define the market multiplier as the change in national consumption including this market interaction, divided by the change in the individual's consumption.

Note that this interaction is not a personal interaction as in the case of the social multiplier (described in Appendix D). The people whose consumption is indirectly affected might never have met the first person. The interaction is mediated through prices.

The value of the market multiplier is determined by the slope of the supply curve. In this report, we are interested in effects discounted over at least 15 years, so it is the long-run supply curve that is of greatest interest. If the long-run supply curve is flat, which would be the case when few factors of production are in short supply, the market multiplier would be one. Shifting the demand curve by eliminating or stimulating the consumption of one person would have no effect on price and, thus, no effect on consumption by others. If the supply curve slopes up, which would pertain if expanding market

volume required employing less and less efficient factors of production, the market multiplier would be less than one. Eliminating the demand of some people would shift the demand curve back, bringing the price down and expanding the quantity consumed by others. If the supply curve slopes down—e.g., because the unit production costs decrease as market volume expands—then the market multiplier would be greater than one. Eliminating some demand would shrink the market quantity, driving up production costs and prices, thereby reducing the consumption of others.

Caulkins et al. (1997) develop and parameterize a model of the cocaine industry's cost structure. According to the "risks and prices" hypothesis (Reuter and Kleiman, 1986), the long-run industry supply curve for cocaine follows the long-run cost curve because free entry and exit preclude the earning of supernormal profits.

The parameter values used by Caulkins et al. (1997) reflect a belief that there are no significant constraints in the long run on the ability to expand cocaine supply. Land in South America for growing coca bushes, smuggling capacity, labor willing to distribute and sell cocaine within the United States, etc. are not in short supply. The values also reflect a substantial industrywide economy of scale associated with diluting or "swamping" of enforcement (Kleiman, 1993). The notion is that the "tax" that enforcement places on the price of cocaine is determined by the amount of enforcement effort per unit of cocaine distributed. Cocaine distribution can be expanded faster than enforcement can, so expanding the amount of cocaine distributed reduces enforcement effort per unit. That reduces the tax per unit, which in turn reduces the cost of providing cocaine and, ultimately, the price.

In the notation of Caulkins et al. (1997), the market multiplier was the percentage change in consumption associated with a 1 percent decrease in demand, which was derived in Appendix C of that publication to be

$$\frac{1-\gamma}{1-\gamma +\left(\beta-\alpha_1\right)\eta}.$$

The base-case parameter values in that report were as shown in Table F.1.

The second and third lines of the table imply $\alpha_1 = 0$ and $\alpha_0 = 0.55$. Hence, the market multiplier $= (1 - 0.25) \div (1 - 0.25 - 0.2) = 0.75/0.55 = 1.3636$, which is our base-case value of the market multiplier before rounding.

There is uncertainty about these parameters, however, so there is uncertainty about the magnitude of the market multiplier. Caulkins et al. subjectively assessed descriptions of the parameter uncertainty as follows:

- η could be described by a triangle distribution between -0.5 and -1.5.

- α_0 could be described by a triangle distribution between 0.25 and 0.85.

- α_1 was taken as fixed at 0.

- β was taken to be uniformly distributed between $\max[0.05, (1 - \alpha_0) \div 9]$ and $\min[0.35, 7 * (1 - \alpha_0) \div 9]$.

- $\gamma = 1 - \alpha_0 - \beta$.

Table F.1

Parameter Values for Calculating Market Multiplier

Parameter	Value[a]	Definition
η	-1.0	Elasticity of demand for cocaine
$\alpha_0 + \alpha_1$	0.55	Fraction of cocaine sales revenue that compensate dealers for costs that are proportional to the quantity sold
$\alpha_1 \div (\alpha_0 + \alpha_1)$	0	Elasticity of normal business costs with respect to expansion in market size
β	0.20	Fraction of cocaine sales revenue that compensate dealers for the costs of arrest, incarceration, fines, cocaine seizures, and asset seizures
γ	0.25	Fraction of cocaine sales revenue that compensate dealers for costs that are proportional to value of cocaine sales

SOURCE: Caulkins et al., 1997.

[a]In base case, as defined by Caulkins et al., 1997, Table 7.4.

We simulated the probability distribution of the market multiplier by taking 10,000 samples from each of these parameter distributions and calculating the associated multiplier. The average value of the market multiplier over these 10,000 trials was 1.376, and 95 percent of the trials yielded multipliers that were between 1.080 and 2.055, suggesting the use of 1.08, 1.38, and 2.06 as lower, middle, and upper estimates. Descriptive statistics are reported in Table F.2.

These values, however, assume that reduced cocaine demand does not lead to reduced law enforcement effort against the drug. This is probably true for marginal changes in demand over the short run (months), but false over the long run (years). Redirection of law enforcement effort would reduce or eliminate this prevention benefit (as far as cocaine control is concerned), driving the market multiplier closer to 1.[1] We have no idea how much closer to 1, so we simply round the low, mid-range, and high values down slightly to 1, 1.3, and 2. This at least allows for the possibility that there is no multiplier effect through the market.

Table F.2

Monte Carlo Simulation's Description of Uncertainty About Market Multiplier

Mean Multiplier Value	1.376
Percentiles of distribution	
2.5	1.080
5	1.096
10	1.120
90	1.709
95	1.891
97.5	2.055

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[1]Redirection to control of other drugs, however, could result in "bonus" reductions in consumption of those drugs that could be considered a benefit of cocaine prevention. Of course, this does not affect the calculation of cost-effectiveness at reducing cocaine consumption.

ESTIMATING THE MAGNITUDE OF THE CAUSATION/CORRELATION QUALIFIER

People who initiate marijuana at older ages have lower expected life-time cocaine consumption than those who initiate earlier. In Appendix B, we calculate the proportional reduction in cocaine consumption associated with a given delay in marijuana initiation. In doing so, we assume that the program that caused the delay of marijuana initiation[1] also reduces lifetime cocaine consumption to the level typical of persons at the new initiation age. Suppose, for example, that an individual's marijuana initiation is delayed from age 12 to 15. We then assume that the individual's lifetime cocaine consumption is reduced to the average lifetime consumption of people who initiate at age 15. However, it is possible that some of the correlation between age of initiation and lifetime cocaine consumption is due to factors unaffected by the prevention program. If so, these calculations would overstate how effective prevention is at reducing cocaine consumption. That is, it is possible that the prevention program merely treats a symptom (marijuana initiation), not the underlying disease (e.g., the individual's attitudes and values). If that is the case, the program would have less effect on lifetime cocaine consumption than the calculations suggest.

The goal of this appendix is to estimate the magnitude of this effect and, thus, the magnitude of a correction factor or qualifier. This cor-

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[1]To avoid constantly using the cumbersome phrase "delay or complete prevention of" initiation, we refer only to delay, but the calculations also apply to the effects of a complete prevention of initiation.

rection factor may be described as an adjustment for the extent to which the correlations underlying the calculations of proportional reduction in cocaine consumption are spurious. We refer to it informally as the "stripe-changing parameter" for brevity and for reasons that will become apparent shortly.

The need for and nature of this adjustment are subtle, so it is worth repeating the description of the underlying problem with a specific, albeit hypothetical, numerical example. Suppose a prevention intervention caused 100 people who would have initiated marijuana at age 12 to initiate marijuana at age 15. On average, people who initiate marijuana use at age 15 will consume 200 grams of cocaine over their lifetime, whereas those who initiate marijuana at age 12 can be expected to consume 375 grams. The method in Appendix B would infer that the prevention program reduced these 100 individuals' lifetime cocaine consumption by $100 \times (375 - 200) = 17,500$ grams.

Suppose, however, that in the absence of a prevention program, all males initiate marijuana at age 12 and all females initiate at age 15. Hence, the average lifetime consumption of males is 375 grams and for females it is 200 grams.

When the prevention program delays 100 people's initiation from age 12 to age 15, it is delaying the initiation of 100 *boys* from age 12 to age 15. What will that do to their lifetime cocaine consumption? If age of marijuana initiation is the one and absolute determinant of lifetime cocaine consumption, it will reduce the boys' lifetime cocaine use from 375 grams to 200 grams, on average. But if gender is the one and absolute determinant of lifetime cocaine consumption, the program will have no effect on the boys' cocaine consumption. In that case, the "tiger will not have changed its stripes"; hence, the shorthand name of this parameter.

In reality, neither age of marijuana initiation, nor gender, nor any other single factor absolutely determines lifetime cocaine consumption. The question is, to what extent does a prevention-induced change in marijuana initiation portend changes in the underlying, generally unobservable, traits that predict lifetime cocaine use? If the answer is "not at all," then the stripe-changing parameter's value is zero. If the answer is proportionately, then the stripe-changing parameter's value is one.

We cannot determine that parameter's true value, but we can shed some light on it by examining how the calculations in Appendix B change if they are replicated for various subpopulations. In particular, we explore how lifetime cocaine use depends on age of initiation while controlling one at a time for various socioeconomic and demographic variables for NHSDA respondents.

METHOD

Measures of Lifetime Use

We used two NHSDA variables to measure lifetime cocaine use. The variable COCTOT measures the number of times someone has used cocaine in his or her life on a seven-point interval scale. We coded it as shown in Table C.2.

The variable CCLFDALY is a yes-or-no question that codes the response to the question: "Thinking back over your whole life, has there ever been a period when you used cocaine, in any form, daily or almost daily for *two or more weeks?*"

Which Observations to Include

We want to know how much cocaine a respondent will use in his or her lifetime, but respondents can only describe how much they have already used. There is no guarantee that they will not use more in the future.

As explained in Appendix C, in evaluating how the age of cocaine initiation affects lifetime cocaine use, we did the calculations using three sets of respondents: All those who had ever used cocaine, people who had ever used cocaine but not in the last 12 months, and people who had used cocaine and initiated at least 12 years before the survey. The second and third groups are approximations to people who will never use cocaine again, but they have smaller sample sizes. The results with all three samples were quite similar when looking at the relationship between lifetime use and age of initiation. Hence, the results reported below are those based on the first group because it had the largest sample size. In evaluating how the age of marijuana and tobacco initiation affected lifetime use, we analyzed the responses of all people who were 25 or older.

Control Variables Analyzed

The control variables we analyzed are listed in Table G.1. These variables are measured at the time of the survey, not at the time of initiation. For some variables, this obviously does not matter. For others, like economic status and residential area, there may be a correlation between the value at the time of initiation and the value at the time of the survey, but they need not be the same.

Note, we have very little ability to control for individual personality traits. For instance, we have no measure of an individual's attitude

Table G.1

Control Variables Analyzed

Binary Variables
Gender
Whether respondent received income from welfare
Whether a family member received food stamps
Trinary Variables
Race (coded as non-Hispanic white, non-Hispanic black, or Hispanic; those categorized as "Other" were dropped because of insufficient number)
Percentage of owner-occupied households in census segment (<10%, 10–50%, or >50%)
Percentage of non-Hispanic black households in census segment (<10%, 10–50%, or >50%)
Percentage of non-Hispanic black population in census segment (<10%, 10–50%, or >50%)
Quintuple Variables
Median value of owned homes in census tract
Percentage of families below poverty line in census tract
Percentage of housing rented in census tract
Median rent for rental units in census tract
Created Variables[a]
Age when first tried cigarettes
Age when started smoking daily
Age when started drinking once a month or more often

[a]These were based on the variables CIGTRY ("About how old were you when you first tried a cigarette?"), CIGAGE ("About how old were you when you first smoked daily?"), and ALCAGE ("About how old were you when you first began to drink beer, wine, or liquor once a month or more often?"). They were redefined to take on one of five values depending on the age of initiation. The value 1 indicated having never done the activity. Values 2 through 5 were associated with approximate quartiles of the distribution of ages of first use for those who ever used cocaine.

toward risk. The three constructed variables pertaining to age of alcohol and cigarette initiation warrant particular attention because they may be the best proxies for individual personality traits.[2]

Definition and Calculation of Stripe-Changing Attenuation Factor

We define the stripe-change parameter as the ratio of the weighted average efficacy computed when considering subpopulations to the average efficacy computed from the aggregate data. Ideally, in estimating the numerator, we would construct completely homogeneous subpopulations. Practically, we cannot do that because of lack of information (e.g., no variables measuring personality traits) and small sample size (the number of cocaine users is too small to divide into more than a few subpopulations). We take a step in that direction, however, by calculating this attenuation parameter considering each of the control variables in turn.

To understand exactly what weighted average was calculated, consider the following notation:

- X_i = quantity of cocaine consumed by group i without prevention.

- d_i = percentage of users in group i whose marijuana initiation is delayed by a prevention program.

- e_i = percentage reduction in consumption for people in group i when their initiation is delayed.

The true effectiveness of a program is the sum over i of $(d_i * e_i)$, weighted by X_i. Here we empirically estimate the e_i values: for any given group, we have NHSDA data on eventual cocaine consumption by those who initiate marijuana at different ages. We cannot, however, estimate the d_i values: Data on the d_i values come from the program evaluations, which do not break results down into the groups we are interested in. So, we imagine a hypothetical program that delays all initiation by one year. Hence, we take all the d_i values to be one, and so simply compute the average of the e_i values,

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[2]We are grateful to Mark Kleiman for pointing this out.

weighting by the X_i values, and compare that weighted average to the e_i computed from aggregate data.[3]

To calculate the e_i values, we want to look at general trends in cocaine consumption in response to delaying cocaine initiation over the 12 through 22 age range and in response to delaying marijuana and tobacco initiation over the 12 through 17 age range.[4]   So, we ran a regression line through those points for all people and for each subpopulation to find the estimated cocaine consumption for those who initiate the use of the drugs of interest at age 12, 17, and 22. We denote these cocaine consumption values by $y(12)$, $y(17)$, and $y(22)$, respectively.   For each regression associated with delaying cocaine initiation, we report the tenth root of $[y(12) - y(22)] \div y(12)$ as an estimate of the average annual percentage reduction achieved by delaying initiation for one year.   Similarly, for delaying tobacco or marijuana initiation, we report the fifth root of $(y(12) - y(17)) \div y(12)$.

Categorization of Possible Effects of Control Variables

Here we present four of the possible outcomes when comparing the effect of delay for subpopulations relative to the aggregate.   We describe them in the abstract and illustrate each with a stylized, hypothetical example, in Tables G.2 through G.5.   Each of the examples below assumes a representative NHSDA-based sample of 100 initiators in each of two age ranges:

- No effect.   The absolute levels of use and their reductions are the same for all subgroups.   In that case, the control variable has no relationship to either drug use or the benefits of delaying initiation.

- The same percentage reductions in all groups, but different levels.   In that case, delay has the same proportionate effect on con-

[3]In ignoring differences among d_i values in this calculation, we do not mean to obscure the point that d_i values may indeed vary among groups, meaning that the efficacy of a program may vary with the characteristics of its participants.

[4]We also looked at the effect of moving people from one broad range of initiation age to another—e.g., from initiating cocaine before 19 to initiating between the ages of 19 and 22. The results were similar.

Table G.2

Example of No Effect

| | Males | | Females | | Total | |
	N	Average Use	N	Average Use	N	Average Use
Age of initiation						
< 19 years	50	100	50	100	100	100
19 to 22 years	50	50	50	50	100	50
Percentage change		–50%		–50%		–50%

N is number of initiators.

Table G.3

Example of Proportional Effects

| | Males | | Females | | Total | |
	N	Average Use	N	Average Use	N	Average Use
Age of initiation						
< 19 years	50	150	50	50	100	100
19 to 22 years	50	75	50	25	100	50
Percentage change		–50%		–50%		–50%

N is number of initiators.

Table G.4

Example of Different Effect With Same Average

| | Males | | Females | | Total | |
	N	Average Use	N	Average Use	N	Average Use
Age of initiation						
< 19 years	50	100	50	100	100	100
19 to 22 years	50	75	50	25	100	50
Percentage change		–25%		–75%		–50%

N is number of initiators.

sumption for each group, but the control variable is related to the extent of use.

- Different percentage reductions for different subpopulations, but the average of those percentage reductions equals the percentage reduction for everyone taken together. In that case, delay has a greater proportionate effect for some groups than for

Table G.5

Example in Which Omitting Controls Is Deceiving

	Males		Females		Total	
	N	Average Use	N	Average Use	N	Average Use
Age of initiation						
< 19 years	75	125	25	25	100	100
19 to 22 years	25	125	75	25	100	50
Percentage change		–0%		–0%		–50%

N is number of initiators.

other groups, but on average the estimate of efficacy derived from aggregated data is accurate.

- The average percentage reduction across the subpopulations is different from the apparent aggregate reduction observed without controls because age of initiation was correlated with the control variable and the control is correlated with lifetime use (i.e., a Simpson's paradox is occurring).

In the first case, the control variable is irrelevant and effectiveness estimates made from aggregate data are sound for all purposes. In the second and third instances (Tables G.3 and G.4), estimates made from aggregate data would reflect the overall effectiveness of programs applied to representative populations, but they may over- or underestimate the effects of programs targeted to particular subpopulations.

For the example in Table G.3, a program that delayed initiation for 100 girls would reduce the quantity of cocaine consumed by only one-third as much as a comparable program administered to 100 boys. However, the percentage reduction in consumption by those in the program would be the same whether the program was administered to boys or girls.

In the third instance (Table G.4), the choice of target population affects not only the total reduction in cocaine consumption but also the percentage reduction in consumption by those in the program. Administering the program to girls instead of boys would not only have three times as great an impact on total U.S. consumption, it

would also have three times as great an effect (in percentage terms) on consumption by program participants.

The second and third instances are problematic for estimating the efficacy of targeted programs from aggregate data. However, they do not affect such estimates for the efficacy of programs directed at *representative* subpopulations or at the population as a whole, which is what we are interested in. In each of those cases, the stripe-changing parameter would be 1. Correlation of efficacy trends with a potentially confounding variable thus does not necessarily imply a stripe-changing parameter less than 1.

Only the fourth instance would indicate that the simple estimate, obtained without controls, might be naively optimistic. In the situation depicted in Table G.5, early initiators are dominated by males while later initiators are dominated by females.. In the case shown, a program that delayed initiation would have *no* effect on cocaine consumption, regardless of whether it was administered to boys or girls. The stripe-changing parameter would be zero.

RESULTS

Again, the stripe-changing parameter is the ratio of the weighted average efficacy computed when considering subpopulations to the average efficacy computed from the aggregate data. By "efficacy" we mean efficacy at reducing cocaine consumption, and the efficacies are calculated on the basis of assumed relations to delays in the initiation of using some substance (marijuana in Chapter Two). Table G.6 reports the calculated values of the stripe-changing parameter for 15 ways of dividing the NHSDA sample into subpopulations (i.e., 15 control variables). For each, we estimate two measures of lifetime cocaine consumption (reported lifetime use and probability of ever using cocaine daily), each based on delays in initiating use of three substances (cigarettes, marijuana, and cocaine).[5] Taking the first

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[5]We include delays in cigarette and cocaine initiation to determine whether the findings for these factors are roughly consistent with those for marijuana. As stated above, this analysis offers only indirect evidence for the value of the stripe-changing parameter, so we cannot regard the results with a high degree of confidence. The use of other factors offers a means of possibly finding cases with low parameter values. Finding

Table G.6

Stripe-Changing Parameter Values
(in percentage)

Control Variable[a]	Delay Cigarette Initiation 1 yr.		Delay Marijuana Initiation 1 yr.		Delay Cocaine Initiation 1 yr.	
	Lifetime Use	P(Daily Use)[b]	Lifetime Use	P(Daily Use)[b]	Lifetime Use	P(Daily Use)[b]
Gender	94.3	92.0	97.5	99.3	94.0	91.3
Welfare	98.8	98.0	99.8	99.8	101.9	99.9
Food stamps	99.2	99.0	99.0	99.4	95.4	95.3
Race	99.5	98.2	99.8	99.6	97.4	98.5
Owner occupied	99.9	98.7	99.3	98.7	96.2	94.0
Black households	97.3	98.6	100.3	98.0	96.2	98.5
Black pop.	98.6	100.1	99.8	97.9	96.7	98.7
Central city	100.2	100.0	99.0	99.7	98.3	99.0
Home values	98.6	97.1	99.6	100.3	79.9	80.5
Rent in area	100.0	100.0	100.3	99.3	89.9	89.6
Poverty	96.2	95.6	99.5	98.4	97.2	95.9
Percentage renting	94.9	95.0	92.4	94.8	95.0	96.7
Age of cig. initiation	NA	NA	93.7	86.8	82.4	82.7
Age of daily cig. use	NA	NA	94.9	88.8	78.1	78.7
Age of monthly alcohol use	54.5	61.5	86.0	75.7	78.1	75.5

[a]See Table G.1 for full definitions of variables.

[b]Probability of ever using cocaine daily.

row, third data column, as an example, then, the interpretation of the value in the cell is as follows: Suppose we were to break the population down by gender and calculate on that basis the effect of a year's delay in marijuana initiation on lifetime cocaine consumption. The effect we would calculate would be 97.5 percent of the effect we calculated in Chapter Two for the population as a whole. Chapter Two's failing to break the population down by gender thus led to a slightly optimistic estimate of the benefit of delaying initiation. This bias could be eliminated by multiplying the naive estimate by 0.975.

For all control variables except the three constructed variables concerning age of cigarette or alcohol initiation, the stripe-changing parameter is close to one, usually above 95 percent. For those three variables, the stripe-changing variables' values were 80–90 percent

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such values would further decrease our confidence in the marijuana numbers; failure to find such values bolsters our confidence to some degree.

for efficacy measures based on delaying marijuana or cocaine initiation.

For a variety of reasons, the ability to infer the value of the overall stripe-changing parameter from this analysis is limited: The analysis relied on self-report data; it controlled for only one variable at a time; the values of the variables were those at the time of the survey, not at the time of initiation; the variables do not measure personality traits; etc. Hence, the fact that these estimates are roughly in the 90 percent range does not imply that the true value of the stripe-changing parameter must be that high. Nevertheless, if delaying initiation had no effect on lifetime cocaine use, we would have expected even these simple analyses to have uncovered greater evidence of spurious correlation. Hence, this analysis gives some circumstantial evidence suggesting that the value of the stripe-changing parameter is probably not extremely low.

We take 0.5, 0.9, and 1.0 to be the low, medium, and high estimates of the overall stripe-changing parameter.[6] However, because the evidence is so thin, some may think our choices are as much a matter of judgment as of objective analysis. Our eight-factor model was constructed to accommodate such differences of opinion. Readers whose judgment differs from ours may substitute some other value for this parameter into the calculations and arrive at their own conclusions about the overall effectiveness of prevention.

As a closing note, delaying marijuana and cocaine initiation had about the same proportionate effect on lifetime cocaine use for all subpopulations. Delaying cigarette initiation had the greatest effect on populations that might traditionally have been perceived as low-risk for heavy cocaine use (wealthy, suburban, white, female, etc.). That is, in terms of the four possible outcomes described above, with respect to cigarette initiation, the data seemed most like the example

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[6]Note also that the parameter values given in Table G.6 do not take into account the proportions of prevention-delayed marijuana initiators falling into the categories shown (male versus female, etc.). That is, they are not based on efficacy in the population sense but on efficacy for the average user. Error could thus result from substitution of the values given here, based on these unweighted, "d_i-free" parameters, into the eight-factor model. However, we assume the error is negligible in the context of the other imprecisions involved in deriving the three estimates given.

in Table G.4; with respect to marijuana and cocaine initiation, the data seemed most like the example in Table G.3.

ESTIMATING THE MAGNITUDE OF THE SCALE-UP DEGRADATION QUALIFIER

To estimate prevention's effectiveness in reducing drug consumption, we utilize estimates of the effects on marijuana use of two important primary prevention programs, Project ALERT and Life Skills Training. However, both of these programs have been implemented only in carefully run studies. We now consider the extent to which the results would be degraded if the programs were implemented in a wider public setting. In our eight-factor analysis, this degradation adjustment is termed the *scale-up factor.*

The distinction between efficacy and effectiveness has long been recognized within the health care research community (Flay, 1986). Efficacy is a measurement of how well a program performs under optimal conditions. Effectiveness is a measure of how well a program performs under real-world conditions. A precise definition of scale-up degradation that seems promising is the difference between efficacy and effectiveness. Ideally, the magnitude of scale-up degradation could be estimated by comparing program efficacy and effectiveness for a large number of different programs. However, we are unaware of any published analysis that does so. Nor have we found any programs for which both efficacy and effectiveness studies have been conducted and published in the literature and from which we could derive our own estimates of scale-up degradation.[1]

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[1]No doubt, the difference between efficacy and effectiveness could vary greatly depending on the nature of the program in question. Thus any generalizations about scale-up degradation could be made only with caution, anyway.

178

What follows is a discussion of the literature we did find that is relevant to the issue of scale-up degradation. We first address the drug prevention literature, and then explore literature from the fields of health care, education, and early intervention for high-risk children. We conclude with the low, middle, and high estimates of the scale-up factor incorporated into the rest of our analysis.

Only two primary drug prevention programs have been widely implemented and adopted in numerous schools across the country, DARE (Drug Abuse Resistance Education) and the "Here's Looking at You" program. Unfortunately, neither case provides much evidence about the expected magnitude of scale-up degradation. DARE was developed jointly by the Los Angeles Police Department and the Los Angeles Unified School District. Because it is taught by police officers instead of teachers and because of the intensive, structured training the DARE officers receive, the probability of strong fidelity to the DARE model is high. It is based to a large extent on the SMART (Self-Management and Resistance Training) curriculum (Gerstein and Green, 1993). However, it (like the other few research-generated programs that have been put into practice widely) is a modified version of the original SMART model (Pentz and Trebow, 1991). Similarly, the "Here's Looking at You" program has evolved significantly over the years, and research results are inconsistent (Gerstein and Green, 1993).

More generally, many health promotion programs have been implemented widely without the benefit of efficacy or effectiveness testing, or have been tested under poor conditions (Flay, 1986). Many programs are evaluated without regard to implementation issues, leading to inconclusive results or an underestimate of program efficacy (Pentz and Trebow, 1991). In addition, many reports of efficacy trials fail to emphasize factors that are critical to understanding the effectiveness of the treatment, such as a complex learning curve or the limitations to the applicability of the treatment (Diamond and Denton, 1993).

There are different theoretical approaches to describing and measuring the implementation of drug use prevention programs. Pentz and Trebow (1991) address the concepts of fidelity, adherence, exposure, and reinvention. *Fidelity* means whether the program was implemented as originally designed by the researchers. It is closely related

to *adherence*, which means whether the treatment and control groups adhered to their respective experimental conditions, and to *exposure*, which measures how much of the program the subjects received. According to Pentz and Trebow, "The question of fidelity is especially important for determining whether a program shown to be effective in an efficacy trial can show effects under effectiveness trial conditions."

Presumably, the fidelity of implementation in a carefully run research study will be greater than in a wider public setting. Why might we expect reduced fidelity when a program is implemented more widely? Most important, widely replicated programs are unlikely to attract staff that are as well-trained, dedicated, or generally capable as the staff involved in the research program. Public agencies may be unable to garner sufficient resources to adequately duplicate the model program. Programs administered on a large scale would have to deal with issues irrelevant to the model program (Karoly et al., 1998). Such bureaucratic issues further reduce the resources that go directly to the intervention.

Reinvention refers to planned changes to the program with the intention of enhancing program effectiveness. Reinvention is also important in determining effectiveness, especially when efficacy measured on one subpopulation may not apply to other treated subpopulations (Pentz and Trebow, 1991). The drug prevention literature in general supports the notion that programs must be adapted to local needs (OSAP, 1991). Reinvention and local adaptation are, obviously, in tension with fidelity.

Flay (1986) takes a different approach to describing and measuring the implementation of social programs. Adapting concepts from the health care research field, he considers three factors: program implementation (standardized, efficacious, or unproven), acceptance and participation by the program recipients, and availability of the program to the target audience. He argues that there should be four phases of testing a health promotion program (that is, after the program has been completely defined and pilot tested):

- Efficacy trials, in which program implementation is standardized and both acceptance and availability are optimized.

- Treatment effectiveness trials, in which program implementation is known to be efficacious and availability is optimized but acceptance is variable.

- Implementation effectiveness trials, in which program implementation is known to be efficacious but both availability and acceptance are variable.

- Demonstration evaluations, in which the effects of an efficacious program when implemented on a whole system are determined.

Although the research framework Flay proposes and his terminology are instructive, it is difficult to place existing research into the framework. More simply, efficacy and effectiveness can be seen as opposite ends of a continuum in which the degree of "real-worldness" varies. Neither Pentz and Trebow (1991) nor Flay (1986) addresses the magnitude of the difference between efficacy and effectiveness. Both emphasize that to understand the significance of outcome measurements, the setting in which a program is tested must be considered.

The Project ALERT data were gathered in a randomized experiment that included 30 junior high and middle schools in Oregon and California. The curriculum was delivered by health educators (in one treatment group) or by health educators assisted by teen leaders (in the other treatment group), all of whom had been trained. Direct observation of lessons by monitors and classroom logs were used to assess the fidelity of the curriculum delivery; they indicate that the curriculum was delivered largely as intended (Ellickson and Bell, 1990a).

The Life Skills Training Program was tested in a randomized trial involving 56 schools in suburban and rural New York state. Providers were trained either by annual workshops and ongoing consulting (in one treatment group) or videotaped training without consultation (in the other treatment group). Results are reported both for the entire sample and a high-fidelity subsample consisting of individuals who received a relatively complete version of the program—i.e., individuals who, based on classroom observation data, received at least 60 percent of the intervention over its three years (Botvin et al., 1995). In contrast to the Project ALERT experiment, in which the school districts spanned urban, suburban, and rural locations, and varied

ethnically and socioeconomically, the Life Skills Training evaluation was run in suburban and rural locations in which the population was predominantly white (Gerstein and Green, 1993).

These descriptions of setting highlight two phenomena that contribute to scale-up degradation: variation in the fidelity of the implementation and variation in the population treated. Both fidelity to a model proven efficacious and appropriate reinvention responsive to variation among participant groups contribute to the overall effectiveness of a program. The Project ALERT and Life Skills Training Program experiments incorporated in this analysis are clearly much closer to the efficacy end of the continuum than to the effectiveness end.[2] What we really need to establish for this cost-effectiveness analysis is their effectiveness.

The issue of scale-up plays a prominent role in other fields besides health care and health promotion. One that is particularly relevant to an analysis of school-based drug prevention is the field of education. Klein et al. (1995) explain that educational reform usually begins at a few demonstration, or "alpha," sites that have revealed apparently promising results. Scale-up means moving from those few demonstration sites to a larger number of more representative sites, and eventually to many, most, or all schools in the country. Because most sites do not enjoy the resources and publicity of the alpha sites, scale-up can present a substantial challenge. Although researchers and practitioners in the field of education are very interested in the issue of scale-up, we have not found any research that suggests the magnitude of degradation.

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[2]When incorporating the results of the Life Skills Training experiment in this analysis, we utilize the results for the full sample, not the high-fidelity subsample. While the high-fidelity subsample results appear to be closer to the efficacy end of the continuum than the full sample results, they may simply represent a self-selected subsample that is no longer comparable to the control group (Gorman, 1998). For example, perhaps the high-fidelity subsample comes primarily from a subset that has lower values for drug-use risk factors, more dedicated faculty, greater overall academic success, etc., than the average school in the experiment. Botvin et al. (1995) report that the demographic characteristics of the subsample are virtually identical to the full sample. However, that does not negate the possibility of heterogeneity in less obvious but equally relevant factors, such as how well the school is run. In general, post-hoc analyses, like this analysis of the high-fidelity subsample, have important limitations and must be interpreted with great care (Diamond and Denton, 1993).

The most prominent example of a widely implemented program in the area of early childhood intervention is Head Start. Since its beginning over 30 years ago, Head Start has served over 15 million children at a total cost of $31 billion (GAO, 1997). The design of this program is similar to that of the High/Scope Perry Preschool Project in Ypsilanti, Michigan, in the mid 1960s, but the programs differ in many respects, such as staff ratio and instructor qualification and training. The earliest evaluations of Project Head Start, first implemented as a pilot program in 1965 serving 500,000 children, showed promising results. But these assessments were of limited value because few outcomes were measured and the evaluations were not randomized controlled trials. Later evaluations of the Head Start program have shown mixed results, some suggesting persistent cognitive and health effects and others not (Greenwood et al., 1998). The 1997 GAO report on Head Start concludes that although an extensive body of literature on Head Start exists, it includes insufficient information to draw reliable conclusions about the effect of the national program. So, neither good information about the efficacy nor conclusive evidence about effectiveness of Head Start exists. Thus, it is not possible to estimate scale-up degradation on the basis of the Head Start literature.

In conclusion, the literature is thick with conceptual work that clearly lays out the issues. It contains hints of serious concerns that scale-up degradation can be a major problem, but it is all but silent when it comes to offering specific numeric estimates. So we base our point estimate on the one comparable specific estimate that is in the literature. Scale-up degradation is addressed by Greenwood et al. (1998) in their study of the cost-effectiveness of early interventions to prevent violence. They assume that the broadly targeted interventions would suffer a scale-up degradation in effectiveness of 40 percent, which corresponds to a scale-up factor of 0.6. In the absence of further evidence of what the scale-up degradation factor should be, it is necessary that we treat scale-up as a sensitivity excursion in our analysis. Namely, we posit what we believe are reasonable lower and upper bounds. We incorporate a low estimate of 0.5 based on the assumption that any greater degradation would likely reflect differences between the model and the program beyond what should be considered scale-up. We incorporate a high estimate of 0.7 based on the assumption that some scale-up degradation is inevitable. The

choice of 0.7 is arbitrary; it allows for symmetry of low and high estimates around the mid-range.

PREVENTION'S EFFECT ON HEAVY ALCOHOL USE
AND CIGARETTE SMOKING

This appendix gives rough estimates of the magnitude of a model school-based prevention program's effect on heavy alcohol use and cigarette smoking. The estimates produced here parallel our analysis of prevention's effects on cocaine use, with emphasis on the third factor (percentage reduction due to the prevention program). That is, the estimates translate empirical evidence concerning prevention programs' effects on a risk factor into estimates of effects on lifetime use. The translation is done using the correlation observed in the NHSDA between the risk factor and some measure of subsequent use. For cocaine, we use age of marijuana initiation as a predictive risk factor for lifetime cocaine use. For alcohol, we use the age when someone begins drinking monthly, or more often, as a risk factor for predicting the number of days in which five or more drinks are consumed and the number of times the individual gets drunk. For cigarettes, we use the age when someone first tried a cigarette as a risk factor predicting two variables of interest: first, the likelihood of ever smoking five or more packs of cigarettes in a lifetime and second, current rates of smoking. These estimates are used in Chapter Four to roughly estimate the magnitude of the social benefits generated by model school-based prevention programs through reduction in smoking and heavy alcohol use.

The risk factor relations are based on the combined 1992 and 1993 NHSDA surveys, so some of the same limitations associated with the NHSDA data discussed above apply. For example, we really have no independent data on how often people get drunk, just their self-report of how often they got drunk in the last 12 months.

ESTIMATING REDUCTION IN HEAVY ALCOHOL USE

Alcohol use per se is not generally a problem, and even frequent alcohol use is not necessarily indicative of a pattern of use that creates harm to the drinker or third parties. So we focus on measures of heavy drinking because that is probably more closely correlated with alcohol-related problems. The NHSDA asks two relevant questions. The variable DR5DAY codes the response to the question: "On about how many days did you have five or more drinks of beer, wine, or liquor on the same occasion *during the past 30 days?* By 'occasion' we mean at the same time or within a couple of hours of each other." The variable DRUNKYR codes the response to the question "How many times *in the past 12 months* have you gotten very high or drunk on alcohol, that is, beer, wine, or liquor?" (Emphasis in original in both cases.) Responses for variable DR5DAY outside the range 0 through 31 were coded as 0. Responses to the second question were coded as indicated in Table I.1.

We divided the NHSDA sample according to the age at which respondents reported first drinking monthly or more often (variable ALCAGE). We then computed the average frequencies of heavy drinking and of getting drunk for each of those initiation-age-specific subsamples. We assume that the subsample for initiation age x is representative of the current-age distribution of individuals initiating at x. Thus, the results tell us the average rate of consumption by individuals initiating at x over the years between initiation and death.

Table I.1

Coding of the Variable DRUNKYR

DRUNKYR Value in NHSDA	Meaning in NHSDA	Recoded to Be
1	Daily in the past 12 months	365
2	Almost daily or 3 to 6 days per week	234
3	About 1 or 2 days a week	78
4	Several times a month (about 25 to 51 days a year)	38
5	1 to 2 times a month (12 to 24 days a year)	18
6	Every other month or so (6 to 11 days a year)	8.5
7	3 to 5 days in the past 12 months	4
8	1 or 2 days in the past 12 months	1.5
All other		0

We are interested in the average rate of use over all ages beyond age 12. For example, if someone used at an average rate of 1 from when he or she initiated at age 20 until he or she died at age 72, his or her average rate of use for all ages beyond 12 would be $((72 - 20) \times 1 + (20 - 12) \times 0) \div (72 - 12)$, or just $(72 - 20) \div (72 - 12)$. We are interested in producing only rough estimates of prevention's effects on alcohol and cigarette use, so we approximated the average rate of use for all ages by multiplying the rate observed after initiation by $(72 - \text{age of initiation}) \div (72 - 12)$, without adjusting for the relationship between age of initiation and life expectancy.

We estimated the effect of the prevention program on the risk factor (age when someone began drinking at least once a month) during junior high and high school years from the data in Table B.1. Because the risk factor is age of first monthly use, not age of initiation, both Project ALERT and Life Skills have outcome variables that are directly relevant. We took the smaller of the two effects (Life Skills' observed effect of 1.7 percent) as our low estimate, the larger of the two effects (ALERT's observed effect of 5.4 percent) as our high estimate, and the average of the two (3.5 percent) as our base estimate.

As with the cocaine estimates above, we considered the three cases of 0 percent, 50 percent, and 100 percent of this reduction being permanent.[1] Where the reduction was not permanent, we again assumed the delayed initiation (in this case into monthly use) was spread over ages 18 through 21 in proportion to baseline initiation rates at those ages. This allowed us to construct Table I.2 parallel to Table C.1 for cocaine. The difference between Table C.1 and I.2 is that Table C.1 gives net present value of quantity of cocaine consumed, whereas Table I.2 gives only the undiscounted current average level of use. A discount factor will be computed and factored in shortly. Implicitly, we are assuming that trends in trajectories of alcohol consumption are negligible. Thus, we can predict lifetime consumption of someone who initiates at age x by looking at the current consumption rates of people of different current ages who initiated at age x.

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[1]A base case of 50 percent permanence may be a little optimistic for alcohol since using alcohol at least monthly at some point in a person's life is very common.

Table I.2

Alcohol Consumption, by Age of First Monthly Use

Age of First Monthly Alcohol Use	Proportion of Cohort Starting Monthly Alcohol Use (Without Prevention)	Proportion of Cohort Starting Monthly Alcohol Use (With Prevention)	Average Number of Occasions Consuming at Least 5 Drinks in Past Month[a]	Average Number of Times Got Drunk in Past Year[a]
11 or younger	0.0064	0.0064	2.2201	56.7890
12	0.0052	0.0050	3.4267	32.5725
13	0.0066	0.0064	2.6872	30.3045
14	0.0154	0.0149	2.1637	26.9923
15	0.0276	0.0267	2.7625	24.9325
16	0.0575	0.0555	2.1518	17.3640
17	0.0463	0.0447	1.6749	13.1800
18	0.1204	0.1216	1.2636	9.3440
19	0.0396	0.0400	1.1141	7.0615
20	0.0484	0.0489	0.8508	12.1614
21	0.0663	0.0670	0.9595	8.5181
22	0.0169	0.0169	0.6029	10.2362
23	0.0093	0.0093	0.6288	8.7477
24	0.0071	0.0071	0.6476	6.4000
25	0.0241	0.0241	0.5895	6.0129
26 or older	0.0452	0.0452	0.4132	3.9233
Never used monthly	0.4575	0.4603	0.0071	0.8996
Aggregate				
Without prevention			0.719	7.040
With prevention			0.710	6.960
Difference			−1.262%	−1.130%

[a]Averages are lifetime beyond age 12.

Table I.2 is based on the following assumptions:

- Prevention reduces initiation into monthly or more frequent alcohol use by 3.5 percent during junior and senior high school.

- Fifty percent of that reduction is permanent.

- The correlation observed in the NHSDA between this risk factor and the outcome variables in columns 4 and 5 predicts how the intervention would affect those variables.

On the basis of Table I.2, we would predict that a model prevention program could reduce the number of times participants got drunk

over their lifetimes by 1.13 percent. The result is very similar (1.26 percent) if we focus on the number of instances in which five or more drinks are consumed at once.

The calculations in Table I.2 are for our base-case reduction in initiation into monthly alcohol use during junior and senior high school (3.5 percent) and permanence (50 percent of the reduction being permanent). We replicated the calculations for all nine combinations of effect and permanence yielding the results in Tables I.3 and I.4. Again the results are very similar regardless of which outcome variable is considered.

Table I.3

Percentage Reduction in Number of Occasions Per Month in Which at Least Five Drinks Were Consumed

Percentage Permanence	Percentage Reduction in Initiation into Monthly Alcohol Use		
	1.7	3.5	5.4
100	0.8	1.7	2.6
50	0.6	1.3	1.9
0	0.4	0.8	1.3

NOTE: The table is read as follows. Assume, for example, the prevention program results in a 1.7 percent reduction in initiation into monthly alcohol use during junior and senior high school and that all of that reduction (100 percent) is permanent. The program then results in an 0.8 percent drop in the average number of occasions per month in which participants consume at least five drinks.

Table I.4

Percentage Reduction in Number of Times Drunk Per Year

Percentage Permanence	Percentage Reduction in Initiation into Monthly Alcohol Use		
	1.7	3.5	5.4
100	0.7	1.5	2.3
50	0.5	1.1	1.7
0	0.4	0.8	1.2

NOTE: The table is read analogously to Table I.3, with the outcome variable indicated by this title replacing that in Table I.3's title.

ESTIMATING REDUCTION IN CIGARETTE SMOKING

For cigarettes, the NHSDA provides three types of variables relevant to heavy use and harms: respondents' descriptions of how many years they smoked daily and at what intensity, respondents' recent smoking behavior, and whether or not respondents have ever smoked five or more packs of cigarettes (cumulative). The first is clearly important, but its analysis is complicated by the selection effects associated with differential rates of mortality among smokers and nonsmokers and the difficulty of estimating how much more respondents will smoke in the remainder of their lives. Recall that the parallel problem with cocaine was side-stepped by recognizing that most people do not use cocaine when they are older, something that is not true of smokers.

Analysis with the second type of variable is completely parallel to that for the alcohol variables just discussed. We focus on the variable AVCIG, which records the response to the question: "How many cigarettes have you smoked per day, on the average, during the past 30 days? Give me the average number per day." We recoded it to measure the average number of packs consumed per day as indicated in Table I.5. As with the alcohol variables, we compute the average by age of cigarette initiation (the risk factor: variable CIGTRY) and multiply by (72 − age of initiation) ÷ (72 − 12) to get the average rate of consumption for all ages over 12.

Table I.5

Coding of the Variable AVCIG[a]

AVCIG Value in NHSDA	Meaning in NHSDA	Recoded to Be
1	Less than one cigarette a day	0.025
2	One to 5 cigarettes a day	0.15
3	About 1/2 pack a day (6 to 15 cigarettes)	0.5
4	About a pack a day (16 to 25 cigarettes)	1
5	About 1-1/2 packs a day (26 to 35 cigarettes)	1.5
6	About 2 packs or more a day (over 35 cigarettes)	2
All other		0

[a]Response to the question: "How many cigarettes have you smoked per day, on the average, during the past 30 days? Give me the average number per day."

Analysis of the third variable is straightforward. The vast majority of people who will smoke five or more packs of cigarettes in their lives will have done so by age 25. Thus, in contrast to the first variable, we do not have to worry, for any given individual over that age, whether the value of the variable will change in future years. We can simply examine how the likelihood of ever smoking at least five packs (to date) varies with age of cigarette initiation for people between the ages of 25 and 60. (We truncate at age 60 to minimize any bias associated with smokers not living as long as nonsmokers, on average.)

We estimate a school-based program's effect on initiation into cigarette use from Table B.1 by the same reasoning we used to estimate the effect on marijuana initiation. Specifically, we base one estimate on Project ALERT's effect on ever using through eighth grade; base another on the average of the Life Skills and ALERT estimates of the effect on weekly, monthly, and past-month use; and average those two values to get our middle estimate. For cigarettes, these three numbers are 4.3 percent, 7.0 percent, and 9.8 percent. As before, we round these off yielding low, middle, and high estimates of the reduction in initiation during junior and senior high school of 4 percent, 7 percent, and 10 percent, respectively.

In parallel with Table I.2, Table I.6 translates the base estimate (7 percent reduction in cigarette initiation) and an assumption of 50 percent permanence into an estimate of the change in current and lifetime use. Likewise, Tables I.7 and I.8 summarize the results of these calculations for all nine combinations of low, base, and high estimates of the effect and permanence of that effect, for each of the two outcomes of interest.

One striking characteristic of Tables I.7 and I.8 is that the estimated effect depends very heavily on how permanent the reductions in initiation observed through the end of high school are, particularly for ever smoking five or more packs of cigarettes. Recall that when age of marijuana initiation was used as a risk factor predicting lifetime cocaine use, it made relatively little difference how permanent the reductions in initiation were. Delaying marijuana initiation from, say, age 14 to 19 was correlated with almost as much of a decline in lifetime cocaine consumption as was completely eliminating that marijuana initiation (see Table C.1). That was less true for age of ini-

Table I.6

Cigarette Consumption, by Age of First Monthly Use

Age of Cigarette Initiation	Proportion of Cohort Starting Cigarette Use (Without Prevention)	Proportion of Cohort Starting Cigarette Use (With Prevention)	Average Number of Packs Smoked Per Day[a]	Proportion of Cohort Ever Smoking at Least 5 Packs
11 or younger	0.1289	0.1289	0.2960	0.6350
12	0.0850	0.0791	0.2939	0.6851
13	0.0656	0.0610	0.3326	0.7426
14	0.0815	0.0758	0.3073	0.7152
15	0.0857	0.0797	0.3331	0.7604
16	0.1077	0.1002	0.2564	0.7019
17	0.0544	0.0506	0.2251	0.6918
18	0.0585	0.0661	0.2363	0.6812
19	0.0226	0.0255	0.1892	0.7120
20	0.0310	0.0350	0.1748	0.6505
21	0.0174	0.0196	0.1644	0.6153
22	0.0087	0.0087	0.1512	0.4794
23	0.0043	0.0043	0.2791	0.7110
24	0.0049	0.0049	0.1690	0.5958
25	0.0056	0.0056	0.2222	0.8013
26 or older	0.0124	0.0124	0.1227	0.5142
Never initiated	0.2258	0.2426	0.0000	0.0000
Aggregate				
Without prevention			0.2109	53.33%
With prevention			0.2045	52.05%
Difference			−3.033%	−2.399%

[a]Averages are lifetime beyond age 12.

tiation into monthly alcohol use as a risk factor for heavy drinking. It is much less true for age of initiation into cigarette use as a risk factor for smoking. Regardless of age of cigarette initiation, about 70 percent of those who try a cigarette will smoke five or more packs in their lifetime. Those who initiate cigarettes at later ages tend to have somewhat lower current rates of cigarette use, but not dramatically lower.

The fact that the permanence of the reductions in cigarette initiation observed through twelfth grade plays such a dominant role is problematic. It means there is relatively greater uncertainty in predictions of prevention's effect on lifetime cigarette use than for other

Table I.7

**Percentage Reduction in Average Number of Packs
Smoked Per Day**

Percentage	Percentage Reduction in Cigarette Initiation		
Permanence	4	7	10
100	2.7	4.7	6.7
50	1.7	3.0	4.3
0	0.8	1.4	2.0

NOTE: The table is read as follows. Assume, for example, the prevention program results in a 4 percent reduction in cigarette initiation and that all of that reduction (100 percent) is permanent. The program then results in a 2.7 percent drop in the average number of packs participants smoked per day.

Table I.8

**Percentage Reduction in Proportion Ever Smoking at
Least Five Packs**

Percentage	Percentage Reduction in Cigarette Initiation		
Permanence	4	7	10
100	2.6	4.5	6.4
50	1.4	2.4	3.4
0	0.2	0.3	0.4

NOTE: The table is read analogously to Table I.7, with the outcome variable indicated by this title replacing that in Table I.7's title.

outcome variables. Conversely, a prevention evaluation follow-up around age 25 would do relatively more to reduce the uncertainty for the estimates of effects on cigarette use than it would for effects on use of the other substances considered.

CHOOSING DISCOUNT FACTORS

The estimates considered above are analogous to the product of the first three factors in our eight-factor model for cocaine, but they do not discount for the time lag between program implementation and its effects. We are only interested in developing rough estimates in this appendix that will allow us to make some adjustment for differences in the lifetime profile of use for different drugs. Thus, we take

a simple, approximate approach. We look at the average amount of current cigarette use and heavy drinking reported in the NHSDA, broken down by age of respondent. (Specifically, we look at the average levels of the AVCIG, DR5DAY, and DRUNKYR variables, coded as described above.) We weight these age-specific averages by the number of residents of the United States by age (data on size of age cohorts from the U.S. Bureau of the Census, *Statistical Abstract of the United States: 1995*, Table 16, p. 16). We then compute the ratio of the discounted to the undiscounted sums of these vectors, discounting at 2 percent, 4 percent, and 6 percent per year to age 12, as we did with cocaine. The results are summarized in Table I.9.

The discount factor is a little smaller for alcohol than for cocaine (0.507 at 4 percent) because alcohol use, even heavy alcohol use, extends into older ages than does cocaine use. The cigarette discount factor is even smaller because cigarette use continues into older ages to an even greater extent.

SETTING THE OTHER FACTORS

To complete the comparison of the effects on cocaine, cigarette, and heavy alcohol use, we need to briefly consider the other four factors in the eight-factor model. There is little reason to think that either of the multipliers is substantially greater than 1 for either cigarette or alcohol outcomes. Recall that the social multiplier captures the fact that initiation of one person into cocaine use can have a "contagious" effect, inducing initiation by others. Similar contagion effects may exist for marijuana, alcohol, and cigarettes. However, we need to explicitly account for contagion only in the case of cocaine. It is the only one of the four substances for which the bulk of the ini-

Table I.9

Discount Factors for Alcohol and Cigarette Use

Discount Rate	Occasions Per Month Consuming at Least 5 Drinks	Times Drunk Per Year	Average Number of Cigarettes Smoked Per Day
2%	0.662	0.674	0.597
4%	0.468	0.489	0.385
6%	0.347	0.374	0.264

tiation takes place after twelfth grade, the end of the time period covered by the follow-up evaluations upon which we base our estimates of program performance. For the others, the social-multiplier effect will be captured in the basic program effectiveness estimate. For example, if one youth's cigarette initiation prompts others to start smoking, most of those others are probably in the participant's school and would initiate in time for it to be observed in the twelfth grade follow-up. (And the same applies to program participants who are prevented from smoking and those they would have influenced to start had they not participated.)

Likewise, there is no reason to believe there is any significant market multiplier for alcohol or cigarettes. They are legal industries, so there is no "enforcement swamping" effect. Indeed, if they have upward sloping industry supply curves, they should have market multipliers that are less than one. However, in the long run, the industry supply curve is probably basically flat, so any multiplier effect would be negligible. As for marijuana, the market multiplier is probably lower than for cocaine because marginal enforcement risk may play a smaller role in driving up marijuana prices than in driving up cocaine prices, but it may well be greater than one. Nevertheless, we set that multiplier equal to one for sake of comparability with the alcohol and cigarettes results. Hence, the estimates for the effect on marijuana may be viewed as being conservative in that respect.

We have no special reason for thinking that the scale-up qualifier should operate much differently for alcohol and cigarettes than for cocaine. We might suppose larger causation-to-correlation ratios than for cocaine and its relationship with marijuana. It is a smaller inferential leap to go from initiation of a drug to later use of that drug than to later use of a different drug. However, the ratio cannot be much higher than what we use for cocaine (0.9) and, since we have no basis for estimating different values, we keep the same values for both qualifiers that we used for cocaine.

RESULTS IN COMPARISON WITH MARIJUANA AND COCAINE FINDINGS

If we multiply the base estimate of the undiscounted, unqualified effect on program participants' use by the discount factor and two

qualifiers, we obtain the base-case estimate of a model school-based prevention program's effect on the net present value of drug use. These factors and their product (the latter in bold) are given in Table I.10. This table is similar to Table 2.3 but varies in two respects. First, the outcome is in terms of the discounted percentage reduction in the activity, not in grams. That is, the proportion of persons using the drug and lifetime consumption are not taken into account. Second, because we set the multipliers for all drugs other than cocaine at 1, we give estimates for cocaine with the multipliers set to 1 (outside the parentheses) and set to the values derived in Appendixes D and F (within parentheses).

Table I.10 suggests that, in percentage terms, school-based drug prevention is most effective against drugs whose use is most deviant. It has the smallest effect on alcohol use, somewhat larger on cigarette use, much greater on marijuana use, and, particularly if the multipliers are included, the greatest on cocaine use. However, even though prevention's effects on smoking and heavy drinking are estimated to be small in percentage terms, they are not unimportant. The social

Table I.10

Summary of Estimates of Effects on Alcohol and Cigarette Use and Comparison With Cocaine and Marijuana Estimates

	Rate of Consuming 5+ Drinks at a Time	Rate of Getting Drunk	Likelihood of Ever Smoking 5 Packs	Rate of Smoking	Mari-juana Use	Cocaine Use
Undiscounted effect						
Low estimate	0.4%	0.4%	0.2%	0.8%	1.8%	2.9%
Base estimate	1.3%	1.1%	2.4%	3.0%	6.1%	7.6%
High estimate	2.6%	2.3%	6.4%	6.7%	12.8%	13.6%
Discount factor	0.468	0.489	0.385	0.385	0.552	0.507
Causation/correlation ratio	0.9	0.9	0.9	0.9	0.9	0.9
Scale-up factor	0.6	0.6	0.6	0.6	0.6	0.6
Overall effect on NPV of use						
Base estimate	0.3%	0.3%	0.5%	0.6%	1.8%	2.1% (5.4%)[a]

[a]Parenthetical quantities assume multipliers with values given in Chapter Two; all other quantities in this row assume that the multiplier is 1.

costs of smoking and heavy drinking are so great that even small percentage reductions can be very valuable in absolute terms.

There is about a factor of six ratio between the high and low estimates of effectiveness for each drug. This ratio is largest for the likelihood of ever smoking five packs because, as discussed above, delaying but not completely preventing initiation into smoking is associated with almost no reduction in that outcome. The result is a broader range between the high and low estimates for program effectiveness (see Table I.8). Delaying marijuana initiation beyond twelfth grade is associated with substantial reductions in both marijuana and cocaine use, even if marijuana initiation is not completely prevented; that is not true for delaying cigarette initiation.

In Table I.11, we supplement the discounted base overall-effect estimates from Table I.10 with several other representations of our bottom-line results. We give both point estimates and ranges, and

Table I.11

Base Estimates and Ranges of Effect, Discounted and Undiscounted, for All Drugs Considered

	Rate of Consuming 5+ Drinks at a Time	Rate of Getting Drunk	Likelihood of Ever Smoking 5 Packs	Rate of Smoking	Marijuana Use	Cocaine Use
Overall effect (undiscounted)						
Base estimate	0.6%	0.7%	1.3%	1.6%	3.3%	4.1% (10.6%)[a]
Low estimate[b]	0.2%	0.2%	0.2%	0.5%	1.0%	1.4% (3.3%)[a]
High estimate[b]	1.3%	1.1%	3.1%	3.2%	6.2%	6.7% (22.6%)[a]
Discount factor	0.468	0.489	0.385	0.385	0.552	0.507
Overall effect (discounted)						
Base estimate	0.3%	0.3%	0.5%	0.6%	1.8%	2.1% (5.4%)[a]
Low estimate[b]	0.1%	0.1%	0.1%	0.2%	0.6%	0.7% (1.7%)[a]
High estimate[b]	0.6%	0.5%	1.2%	1.2%	3.4%	3.4% (11.5%[c])[a]

[a]Parenthetical quantities assume multipliers with values given in Chapter Two; all other quantities in this row assume that the multiplier is 1.

[b]From Monte Carlo simulation analysis.

[c]Rounded from 11.48; thus, 11 when rounded to nearest whole number.

we express them both with and without discounting. The indications of ranges are in the form of low and high estimates produced by a Monte Carlo analysis similar to that described in Chapter Two. For this analysis, we fix the discount factor at that corresponding to a discount rate of 4 percent. We vary the size of the effect, the scale-up qualifier, and the causation/correlation qualifier uniformly between their lower and upper bounds. The two multipliers' values are set equal to 1, except for the parenthesized cocaine numbers, for which they are varied uniformly between their lower and upper bounds.

ACCUMULATION OF PREVENTION'S EFFECT
NATIONWIDE

The analysis elsewhere in this book estimates prevention's effect on consumption. This effect is not felt immediately upon implementation of the program. The delay's consequence for prevention's cost-effectiveness is reflected in the discount factor (factor 4). But when thinking about the effect of a nationwide prevention program on aggregate consumption, it is useful to see the rate at which total use declines—not just use by the cohort that received the program. That distinction is important. Even if a prevention program eliminated all consumption for those who received it, it would not, in the short run, eliminate all consumption in the country. People who had already left junior high school before the prevention program was implemented would not be affected directly by the program. Similarly, a prevention program that reduces use by those in the program by 10 percent will not reduce national consumption by 10 percent until the population is made up entirely of people who received the program.

The goal of this appendix is to plot projected aggregate cocaine use with and without nationwide implementation of a model school-based prevention program. We cannot do this exactly, but the approximation we do create is accurate in its qualitative features. Three difficulties prevent an exact projection.

The first problem is that we do not know how many people in future birth cohorts would use cocaine in the absence of a nationwide prevention program. That is, we do not know the future values of factor 1. We assume a hypothetical scenario in which 17.5 percent of each succeeding cohort initiates cocaine in the absence of the pre-

vention program. (We also apply this 17.5 percent assumption to cohorts that have already been born but that are still too young for us to determine empirically what proportion will eventually use cocaine before they die.)

The second problem is that we do not have good data on quantities of cocaine consumed by age. The sources we used in Appendix A to estimate lifetime consumption in grams have limited ability to describe how prevention affects the quantity consumed as a function of the individual's age, and NHSDA responses to questions concerning amounts consumed are unreliable. So, as with our calculations of the discount factor, we will focus on whether an individual used cocaine in the last twelve months. In particular, we use only the responses to the NHSDA questions about annual and lifetime prevalence of cocaine use. Thus, we will not project prevention's effect on the quantity consumed but rather on the number of past-year users that would be recorded by an instrument like the NHSDA. However, the rate of decline in the number of users that we calculate is not necessarily very different from the corresponding rate of decline in quantity consumed, for the following reason. In any given year, the population of cocaine users contains a mixture of lighter and heavier users. If that mixture stayed the same, number of users and quantity consumed should follow the same trend. Indeed, at this late point in the epidemic, we would not expect to see large changes in the mixture of light and heavy users—barring an intervention dramatically reducing user numbers (Behrens et al., 1998). Prevention would not appear to be capable of such an effect.[1]

Third, we can estimate explicitly the time trajectory of only that portion of prevention's effect that results from actions by people in the program. The timing of the effects through the two multipliers may be different. Initiations averted through indirect social- and market-multiplier effects may, for example, lag direct program effects. We assume, however, that the timing of direct and indirect effects is the

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[1]Number of users and quantity consumed would not necessarily follow similar trends earlier in a drug epidemic, when the mixture of lighter and heavier users is changing.

same and thus simply multiply the effect without the multipliers by the value of the multipliers.[2]

The calculations involve combining enough different pieces of information that they are best explained with mathematical equations, not words. We are interested in estimating the number of users in year t, or $U(t)$, both with and without a nationwide prevention program. Let $t = 0$ be the year when the prevention program would be implemented. We will use y to index the birth cohort, j to index the year of initiation into cocaine use, and i to index the year of initiation into marijuana use. The indices for year of initiation run from "never" through "less than 12," "13," "14," ..., "25," and "over 25." We ignore cocaine use by people over the age of 100.

$$U(t) = \sum_{y=t-100}^{t} S_y P\{u(t-y)\,|\,b(y)\}$$

where S_y = the size of the birth cohort y[3]
$u(t-y)'$ = use of cocaine at age $(t-y)$
$b(y)$ = birth in the year y.

$$P\{u(t-y)\,|\,b(y)\} = \sum_{j="never"}^{">25"} \left[P\{u(t-y)\,|\,I_c(j)\} \right]\left[P\{I_c(j)\,|\,b(y)\} \right]$$

where $I_c(j)$ = initiation of cocaine at age j.

We estimate

$$P\{u(t-y)\,|\,I_c(j)\},$$

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[2]Over a number of years, as a greater percentage of the population has participated in model prevention programs, the proportion of cocaine consumers who are light users would change, thereby changing the social multiplier slightly. We have ignored this effect.

[3]Birth cohort sizes were taken from the U.S. Bureau of the Census (1995, Tables 4 and 87). Sizes for cohorts in nondecennial years were linearly interpolated. For years before 1950, birth cohorts were assumed to decrease by 100,000 per year for earlier years, down to a level of 2.5 million. Note: Sizes of these birth cohorts have very little effect on the results because only a small fraction of those cohorts use cocaine.

the probability of use at a certain age, given initiation in a designated year, from the NHSDA. This is taken as the ratio of the number of people reporting cocaine use in the last year divided by the number of people reporting having ever used cocaine, among those who are age $(t - y)$ and who initiated at age j.

We calculate

$$P\{I_c(j)|b(y)\} = \alpha P\{I_c|b(y)\} \sum_{i="never"}^{">25"} \left[P\{I_c(j)|I_m(i)\} \right]\left[P\{I_m(i)\} \right]$$

where α is a proportionality factor that is essentially the reciprocal of the weighted average of the probabilities of cocaine initiation by birth cohort and where $I_m(i)$ = initiation of marijuana use at age i.

The summed product of probabilities gives $P\{I_c(j)\}$, the probability of initiating cocaine at age j, taken as a weighted average over all birth cohorts. To turn this general probability into the cohort-specific probability on the left side of the equation, it must be multiplied by a factor indicating how much more or less likely to initiate cocaine cohort y is than the average. That factor is

$$\alpha P\{I_c|b(y)\},$$

which is essentially the ratio between cohort y's probability of initiating cocaine and the weighted average probability of initiating cocaine across all cohorts.

For birth cohorts born between 1927 and 1969,

$$P\{I_c|b(y)\},$$

the proportion ever using cocaine, is taken from Figure 2.2. (Consumption by cohorts born before 1927 is ignored.) For cohorts born after 1969, it is assumed that 17.5 percent would initiate cocaine at some time in their lives in the absence of a nationwide model prevention program.

$$P\Big\{I_c(j)\Big|I_m(i)\Big\},$$

the probability of initiating cocaine at age j given marijuana initiation at age i, is estimated as the following quotient: the number of NHSDA respondents who report initiating marijuana at age i and initiating cocaine at age j, divided by the number who report initiating marijuana at age i. $P\{I_m(i)\}$ is where the effects of prevention enter. For birth cohorts not exposed to a prevention program, the historical distribution of ages of marijuana initiation is used. Thus, in the expression for

$$P\Big\{I_c(j)\Big|b(y)\Big\},$$

the sum of probabilities is taken over the historical distribution of the ages of cocaine initiation. (Therefore, it is irrelevant that different historical cohorts may have had a different relationship between the marijuana initiation risk factor and cocaine initiation.) For cohorts that receive the prevention program, the age distribution of marijuana initiation is modified accordingly. In either case, the distribution is given in Table C.1. (Table C.1 with prevention is for the middle case of 11 percent reduction and 50 percent permanence. Calculations for the low and high estimates of prevention's effectiveness are parallel.)

We perform these calculations with the probability of initiating marijuana at age i reflecting no prevention and then again with prevention. The multipliers and qualifiers are incorporated by multiplying the gap between results with and without prevention by the product of those four factors. In particular, the product of the middle values of those four factors is $2.0 \times 1.3 \times 0.9 \times 0.6 = 1.404$. So if $U(t)$ with and without prevention in a particular year, t, were calculated to be 5.0 and 4.9, respectively, then an adjusted $U(t)$ with prevention that reflected the multipliers and qualifiers' base values would be $5.0 - 1.404 \times (5.0 - 4.9) = 4.86$.

Figure J.1 shows the result of these calculations for nationwide implementation of the model prevention program in 1992—i.e., assuming the 1980 birth cohort is the first one to receive the new
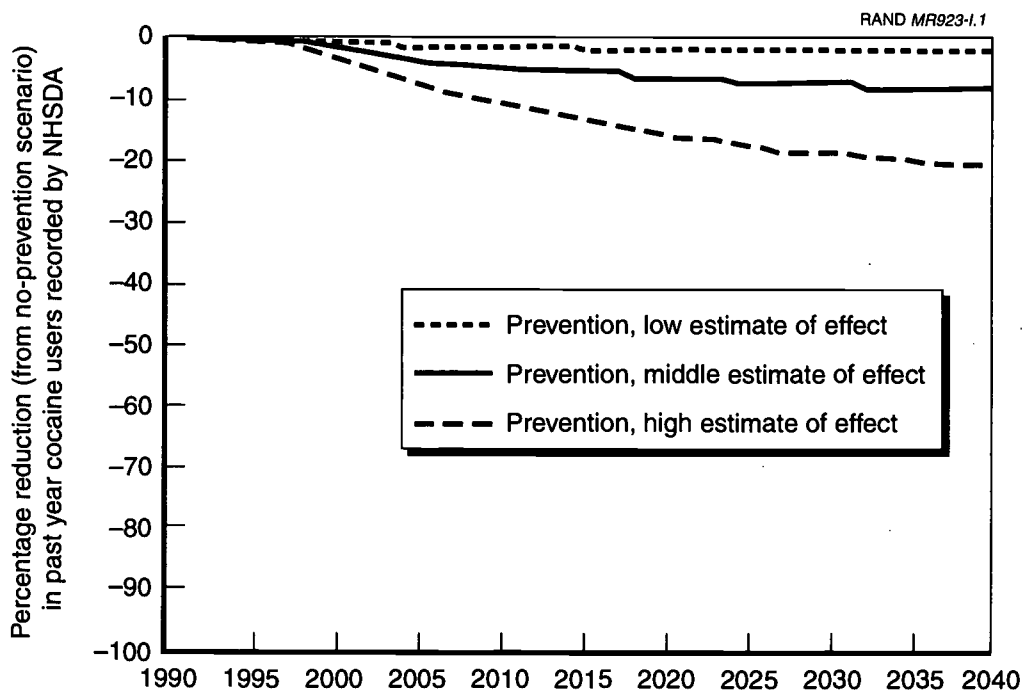
Figure J.1—Prevention Will Only Modestly Reduce the Number of Cocaine Users over a Period of Decades

prevention program. The multipliers and qualifiers are set at their base values. Curves show the cumulative percentage reduction in number of users as of each year up to 2040 for the low, middle, and high estimates of prevention's effectiveness (factor 3), relative to a no-prevention scenario. As mentioned above, that scenario assumes that 17.5 percent of each cohort born after 1969 will initiate cocaine.

By "number of users," we mean the number of past-year cocaine users as measured by NHSDA self-report, not the true number of cocaine users. Our assumed no-prevention value is 4.94 million in 1992 (the base year for our calculations), which is similar to the 4.98 million past-year cocaine users actually recorded in the 1992 NHSDA. Both are lower, however, than the 6.7 million Rhodes et al. (1995) estimate by combining information from the NHSDA and other sources.

Note that, because the no-prevention scenario is hypothetical, none of our projections is a prediction of what will actually happen. We

merely intend to give some sense of the potential overall effect of a prevention program. Perhaps the most striking aspect of Figure J.1 is that even with the most optimistic estimates of prevention's effectiveness, prevention will not dramatically alter the course of the current cocaine epidemic. Although prevention can eventually reduce the number of users by a nontrivial amount, that effect is far from immediate. With the middle estimate of prevention's effectiveness, it would take a nationwide model prevention program six years to achieve a 1 percent reduction in the number of past-year cocaine users relative to a no-prevention baseline. It would take 10 years to see a 2.5 percent reduction, 20 years to achieve a 5 percent reduction, and 40 years to see a 7.5 percent reduction.

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Prescription for Drug Prevention

"There are signs that, after many years of policy emphasis on enforcement, prevention may be about to have its day."

—Jonathan P. Caulkins et al.
An Ounce of Prevention, a Pound of Uncertainty

Many programs are being pursued in an effort to ameliorate America's drug problem. These programs range from interventions in source countries to border control, enforcement within U.S. borders, treatment of users, and efforts intended to prevent use. They currently cost about $40 billion a year across all levels of government in the United States, yet drug consumption remains at levels widely acknowledged to be unacceptable.

Belief in the adage that "an ounce of prevention is worth a pound of cure" predisposes many to believe that money invested in drug use prevention programs will yield considerable returns. But is this really true? How effective is prevention? How much does it cut drug use? How does it compare, dollar for dollar, with other approaches to drug control, such as enforcement or treatment? What would be the costs and consequences of nationwide implementation of a cutting-edge prevention program?

The authors, who have already published milestone works on the economics of criminal-justice issues, use the results from the highly regarded school-based drug prevention programs "Project ALERT" and "Life Skills" to answer these questions. They discuss the implications of their findings for school-based drug prevention as a matter of national drug control policy, and show how different analytical assumptions may result in different answers—rendering the ounce of prevention an uncertain cure.

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