Assessing the Impact of Antidrug Advertising on Adolescent Drug Consumption: Results From a Behavioral Economic Model

Lauren G. Block, PhD, Vicki G. Morwitz, PhD, William P. Putsis Jr, PhD, and Subrata K. Sen, PhD

Objectives. This study examined whether adolescents' recall of antidrug advertising is associated with a decreased probability of using illicit drugs and, given drug use, a reduced volume of use.

Methods. A behavioral economic model of influences on drug consumption was developed with survey data from a nationally representative sample of adolescents to determine the incremental impact of antidrug advertising.

Results. The findings provided evidence that recall of antidrug advertising was associated with a lower probability of marijuana and cocaine/crack use. Recall of such advertising was not associated with the decision of how much marijuana or cocaine/crack to use. Results suggest that individuals predisposed to try marijuana are also predisposed to try cocaine/crack.

Conclusions. The present results provide support for the effectiveness of antidrug advertising programs. (*Am J Public Health.* 2002;92:1346–1351)

In the present study, we evaluated the effectiveness of the national antidrug advertisements of the Partnership for a Drug-Free America (PDFA). Over the years, PDFA has received more than $3 billion in donated media from a variety of sources, including the major television networks, 11 cable networks, 11 radio networks, more than 1000 newspapers, and more than 100 magazines and medical journals (M. Townsend, chief marketing officer, Partnership for a Drug-Free America; written correspondence; May 1998). PDFA's donated media make it the largest advertiser of a "single product" in the United States after McDonald's.[1]

We analyzed data from the first 4 years of the Partnership Attitude Tracking Survey (PATS), an annual survey conducted by PDFA to independently test whether the commencement of the advertising campaign was associated with a change in adolescents' drug use. The first "wave" of PATS was initiated during February and March 1987, 3 months before the first antidrug messages were aired. Additional waves, which took place during February and March of each year thereafter, measured respondents' recall of PDFA advertisements. These waves formed a "natural experiment" in that respondents during the first wave were not exposed to PDFA advertising, whereas respondents in subsequent waves were subjected to PDFA advertising.

A preliminary examination of the PATS data reveals that the percentages of respondents who reported marijuana or cocaine/crack use in the previous 12 months decreased significantly over the years 1987 to 1990. Other sources of data corroborate this pattern (e.g., survey data from the University of Michigan's Institute of Social Research, National Household Survey on Drug Abuse).[2,3] Although this overall pattern is consistent with the hypothesis that antidrug advertising reduces drug consumption, such a simple analysis does not accommodate other potential explanations for changes in drug consumption over time. To adjust for these other factors, we used a detailed behavioral economic model that investigated the relationship between adolescents' recall of antidrug advertising and their probability of using marijuana, cocaine, or crack, as well as their volume of use given that they were already using these drugs.

METHODS

Data Sources

Data were obtained through multiple-site central location sampling, usually conducted at shopping malls (see Black et al.[4] for a detailed report on the PATS methodology). Respondents at selected sites approximated a national probability sample. Sites were selected along 2 dimensions: (1) regional and (2) urban, suburban, and rural distribution of the population. At each site, sex and race quotas were established. The sample sizes of adolescents aged 13–17 years (numbers of locations from which adolescents were sampled are shown in parentheses) during 1987 through 1990 were 797 (96), 1031 (89), 870 (85), and 1497 (99), respectively.

Self-administered questionnaires were completed by respondents in a private facility and returned anonymously in a blank envelope. This data collection method should have resulted in an increased willingness among participants to reveal illicit or undesirable behaviors compared with other methods.[5] Although evidence indicates that drug use self-reports are highly reliable and valid,[6,7] we also conducted a detailed analysis of the impact of potential reporting biases on the results.

Theory and Key Constructs

We began with an individual-level behavioral economic model of drug use, focusing on the impact of advertising. This well-established economic framework provided the rigorous link between the underlying theory and the statistical model needed to estimate individual behaviors.[8–10] We then relied on health behavior theory to select the specific variables used within this empirical specification. The measures used in the analysis represented the predominant benefits and costs of drug use identified in major health behavior theories.[11–13] Because factor analy-

ses indicated that all of the multi-item measures described below loaded on 1 factor, items were averaged.

Measures of drug consumption. We analyzed marijuana use separately from cocaine/crack use because reasons for use differ for specific drugs.[14] We combined cocaine and crack into a single category because 92% of respondents reported using both with equal frequency. Respondents indicated how often in the past 12 months they had used each drug by selecting 1 of 7 alternatives: (1) no use, (2) once, (3) 2 to 3 times, (4) 4 to 9 times, (5) 10 to 19 times, (6) 20 to 39 times, or (7) 40 or more times. We used these responses to determine both the percentages of respondents who reported using each drug in the previous 12 months (0=no use, 1=any use in the past 12 months) and the volumes of use among those reporting use. In the case of users of both drugs, we divided their volume of use at the median and considered those below the median to be light users (coded as 0; representing 1 to 9 times) and those above the median to be heavy users (coded as 1; representing 10 to 40 or more times).

Perceived susceptibility. The more adolescents perceive themselves to be susceptible to the negative consequences of drug abuse, the less likely they are to use drugs.[15] Perceived susceptibility was measured by asking respondents to rate 3 items (on 4-point scales) indicating the degree to which people risk harming themselves by using drugs (physically or in other ways); low scores corresponded to no risk. Scale α values were .86 (marijuana susceptibility) and .94 (cocaine/crack susceptibility).

Perceived severity. The more adolescents perceive the consequences of drug abuse to be severe, the less likely they are to use drugs.[13] Respondents rated 4 items (on 4-point scales) indicating the degree to which they would fear the consequences of being caught with drugs; low scores corresponded to no fear. The perceived severity scale α value was .88.

Attitudes toward drugs. The more favorable teenagers' attitudes toward drugs, the higher their likelihood of using drugs.[15–17] Respondents indicated their level of agreement with 14 items (on 5-point scales) describing benefits of drug use; high scores represented unfavorable attitudes toward drugs. The α value for the attitude scale was .89.

Attitudes toward drug users. As evidenced by national[14] and regional[17] surveys, adolescents with positive attitudes toward drug users are more likely to use drugs. Attitude toward drug users was measured by having respondents indicate whether each of 27 personality characteristics would describe a marijuana, cocaine, or crack user; high scores represented unfavorable attitudes toward drug users. The α values were .80 for the marijuana scale and .82 for the cocaine/crack scale.

Peer pressure. Drug use is influenced by social norms and peer pressures.[18,19] Peer pressure was assessed with 2 items rated on 5-point scales: number of friends who use each drug occasionally at parties or social events and how many close friends get "stoned" or "high" on each drug once a week or more (low scores corresponded to no close friends).

Drug availability. The supply or availability of drugs is also a significant factor in drug use. Respondents rated how difficult it would be for them to obtain each drug on a single-item 5-point scale (low scores corresponded to extreme difficulty).

Addictive properties of drugs. Past drug usage accounts for a significant degree of variability in subsequent drug consumption.[16,20,21] Previous addiction (1=yes, 0=no) was measured via asking respondents whether they had ever thought they were hooked on marijuana, cocaine, or crack.

Antidrug advertising. Recall was measured by asking respondents to read a short description of each advertisement and to indicate how often they had seen the advertisement. Ratings were made on a 3-point scale (low scores corresponded to not at all). All 6 advertisements were aired nationally, and there were no known differences in frequency or reach for the intended teenage audience. The α value for the recall scale was .81.

Demographic covariates. Three covariates in our model controlled for individual heterogeneity. Respondents indicated their sex (0=female, 1=male), their race (1=White, 0=other), and whether they lived in an urban or rural area (1=city or suburb of a city, 2=town/village or rural area).

Statistical Analyses

We present an abbreviated description of the statistical methodology here. A comprehensive explanation of the models and analyses is available separately.[22]

Stage 1: The decision to use marijuana or cocaine/crack. The probabilities of a respondent's reporting use of marijuana and cocaine/crack over the previous 12 months were expressed in a standard "probit" formulation[23] as a function of both the attributes of the individual (e.g., demographic characteristics) and his or her perceptions of drug use itself (e.g., perceived severity). We considered 3 versions of this formulation, each involving a slightly different assumption about the relationship between the cocaine/crack and marijuana use decisions.

First, we estimated the marijuana and cocaine/crack equations independently, assuming that the decision to try the 2 drugs is independent. However, empirical research suggests that the process may be sequential; that is, one first tries marijuana and then cocaine/crack.[17,24] Second, the common-syndrome theory[25] suggests that individuals have a "predisposition" to use drugs that manifests itself first in marijuana use. Third, certain factors associated with the experience of using marijuana could lead people to use harder drugs, such as cocaine/crack; this has been referred to as a "gateway" or "stepping stone" theory.[26,27] These 3 alternatives resulted in different statistical specifications, allowing us to test the hypotheses with the available data.

Stage 2: The volume decision. In addition to the "use" choice, we investigated the decision regarding how much to use (the "volume" decision), given that an individual has reported using marijuana or cocaine/crack. Although the decision regarding how much to use is a continuous one, data limitations (data were reported categorically, and there were too few observations in key cells)[22] forced us to categorize individuals as "light" or "heavy" users.

The result is a classic sequential-choice decision: an individual uses the drug and then, on the basis of his or her experience and additional information (e.g., antidrug advertising), decides whether or not to use the drug again.[22,23] Accordingly, for each drug,

we initially estimated stage 1 probability equations and then estimated the probability of a given individual's being a light or heavy user conditional on previous use. Thus, including only those who had previously used drugs, we estimated each second stage equation using a dichotomous dependent variable that took on a value of 1 if the respondent was a "heavy" user and 0 if he or she was a "light" user.

Stage 3: Evaluation of advertising effectiveness. The first "wave" of PATS (conducted before the initiation of antidrug advertising) provided us with the data necessary to assess the determinants of drug use in the absence of PDFA advertising (the "control" in our natural experiment). We were then able to assess the significance of recall of PDFA advertising in terms of use and volume decisions via a series of "treatment" groups consisting of each of the subsequent waves exposed to advertising.

We began by estimating the 3 sets of probability-of-use equations ("independent," "gateway," and "predisposition") using the wave 1 data for marijuana and cocaine/crack. Then, on the basis of the best fitting of these equations, we estimated the second stage regressions for the probability of being a light vs heavy user, also using the wave 1 data. This provided us with a detailed analysis of the factors influencing the decision to use and the volume of use for each drug before the commencement of PDFA advertising.

One way of assessing the impact of PDFA advertising would be to repeat the stage 1 and stage 2 probability equations for waves 2, 3, and 4, including the additional variable capturing respondent recall of advertising. The problem with this approach is that advertising recall may be related to an individual's previous drug use behavior. For example, a heavy drug user may tune out antidrug advertising. Accordingly, in measuring the impact of PDFA advertising in waves 2, 3, and 4, we had to control for the endogeneity of advertising recall by adjusting for probability of use at the individual level. Fortunately, we had a ready-made estimate of an individual's probability of use from the wave 1 control group probability equations.

Specifically, using the estimated coefficients from the wave 1 control group, we predicted the probability of use for each individual in wave 2 (the first wave exposed to PDFA advertising). This provided us with estimates of the probability of use of marijuana (Ψ_{MJ}^2) and cocaine/crack (Ψ_{CC}^2) in wave 2 in the absence of PDFA advertising (because the parameter estimates were generated by control-group relationships).

The probability of using drugs in wave 2 was expressed as a function of 2 variables: probability of use in the absence of PDFA advertising and recall of PDFA advertising. The coefficient for the advertising-recall variable provided a test of the impact of PDFA advertising on the 2 probability-of-use equations for respondents in wave 2.

This process was repeated for marijuana and cocaine/crack use in waves 3 and 4. The same methodology was then employed for the set of users based on the stage 2 analysis of volume of marijuana and cocaine/crack use among existing users.

Because the 3-stage methodology involved the use of the results from the wave 1 "control" group data as the basis for the subsequent analysis, many of the statistical problems associated with self-reported survey data were alleviated. For example, consider "social desirability bias," the tendency of individuals to provide responses that they think are socially desirable. A detailed analysis of this potential reporting bias suggested[22] that it served only to strengthen the results by (1) lowering the estimated marginal impact of antidrug advertising on drug use and (2) inflating coefficient standard errors,[28] thereby increasing the likelihood of concluding that advertising had no effect. Thus, our results represent a conservative estimate of the impact of antidrug advertising.

TABLE 1—Wave 1 Probits on Probabilities of Marijuana and Cocaine/Crack Use

	Coefficient	SE	*z* (β/SE)	*P* (*Z*>*z*)
		Marijuana Use[a]		
Constant	0.4343	. . .	. . .	. . .
Attitude toward drugs	–0.5719	0.1093	–5.233	0.000
Perceived severity	–0.2854	0.0870	–3.280	0.001
Attitude toward users	–0.2043	0.5808	–0.352	0.363
Peer pressure	0.6479	0.0801	8.086	0.000
Perceived susceptibility	–0.3085	0.0954	–3.232	0.001
Previous addiction	0.8349	0.3423	2.439	0.007
Availability	0.0901	0.0542	1.664	0.048
Urban residence	0.0860	0.1587	0.542	0.588
Male	0.1675	0.1438	1.165	0.244
Race	0.4100	0.2116	1.938	0.053
		Cocaine/Crack Use[b]		
Constant	5.0748	. . .	. . .	. . .
Attitude toward drugs	–0.5279	0.1480	–3.567	0.000
Perceived severity	–0.4407	0.1309	–3.366	0.001
Attitude toward users	0.0731	0.6707	0.109	0.457
Peer pressure	0.6592	0.1134	5.814	0.000
Perceived susceptibility	–0.3891	0.1437	–2.709	0.004
Previous addiction	2.2655	0.5876	3.856	0.000
Availability	0.0991	0.0869	1.151	0.125
Urban residence	0.2870	0.2535	1.132	0.258
Male	0.3205	0.2371	1.352	0.177
Race	0.0198	0.3397	0.058	0.954
Decision independence	0.8549	0.1854	4.612	0.000

[a]No. of observations = 642, log-likelihood = –402.87, χ^2 = 383.54 (*P* = .0000).
[b]No. of observations = 630, log-likelihood = –202.60, χ^2 = 232.85 (*P* = .0000).

RESULTS

Stage 1 ("Use") and Stage 2 ("Volume") Probits: Wave 1 Data

Using nested tests, we concluded that the "predisposition" formulation fit significantly better than the "independent" process. Consequently, we used this formulation throughout. In addition, in the "gateway" formulation, the binary variable representing previous marijuana usage in the cocaine-use probit was statistically nonsignificant, leading us to reject the hypothesis that marijuana use increases the probability of cocaine/crack use. Although individuals who have used marijuana in the past are indeed more likely to use cocaine/crack, the reason is that, statistically, individuals who are predisposed to try marijuana are also predisposed to try cocaine/crack.

Table 1 presents the wave 1 results of the stage 1 equations for marijuana and cocaine/crack use. All of the variables were of the predicted sign, and the overall fit was excellent.

We next estimated the stage 2 volume-of-use equations (Table 2). The fit and parameter estimates for volume of marijuana use were quite good, but the overall equation for volume of cocaine/crack use was barely significant, at $\alpha=0.10$. In regard to the marijuana volume equation, White and male respondents were more likely to be heavy (vs light) users. (Note that a positive and significant "decision independence" value implies that the decision to use marijuana and the decision to use cocaine/crack are not independent; that is, the use probability influences the volume decision.[22])

TABLE 2—Wave 1 Second-Stage Probits for Light vs Heavy Marijuana and Cocaine/Crack Use

	Coefficient	SE	z (β/SE)	$P(Z>z)$
	Marijuana Volume[a] (0 = light user, 1 = heavy user)			
Constant	–2.0510	. . .	. . .	. . .
Attitude toward drugs	–0.6927	0.3065	–2.260	0.012
Perceived severity	–0.4294	0.1729	–2.483	0.007
Attitude toward users	–1.0230	0.7443	–1.374	0.085
Peer pressure	0.8800	0.2744	3.206	0.001
Perceived susceptibility	–0.4023	0.2005	–2.007	0.023
Previous addiction	1.5386	0.5398	2.850	0.002
Availability	0.0763	0.1012	0.753	0.226
Urban residence	0.2008	0.2505	0.801	0.423
Male	0.5896	0.2430	2.426	0.015
Race	0.9078	0.4038	2.248	0.025
Decision independence	1.3481	0.7885	1.710	0.087
	Cocaine/Crack Volume[b] (0 = light user, 1 = heavy user)			
Constant	0.7405	. . .	. . .	. . .
Attitude toward drugs	–0.0259	0.2373	–0.109	0.457
Perceived severity	–0.1925	0.2532	–0.760	0.224
Attitude toward users	0.3341	0.8981	0.372	0.355
Peer pressure	0.3167	0.1856	1.706	0.044
Perceived susceptibility	–0.4509	0.2567	–1.757	0.040
Previous addiction	–0.9231	0.5627	–1.641	0.051
Availability	0.0641	0.1556	0.412	0.341
Urban residence	0.4104	0.4477	0.917	0.359
Male	0.4210	0.4206	–1.001	0.317
Race	0.0561	0.5535	–0.101	0.920

[a]Stage 2 dependent. No. of observations = 206, log-likelihood = –137.61, $\chi^2 = 234.34$ ($P = .0000$).
[b]Stage 2 independent. No. of observations = 64, log-likelihood = –43.86, $\chi^2 = 234.34$ ($P = .0861$).

Stage 3 Probits and the Impact of Antidrug Advertising: Analysis of Waves 2, 3, and 4

This analysis, conducted with the wave 1 "control" group, provided the basis for analyzing the significance of recall of PDFA advertising in waves 2, 3, and 4. The results are presented in Table 3.

The findings demonstrate that recall of antidrug advertising was associated with a decreased probability of marijuana use. The advertising coefficients in the marijuana use equation (see top section of Table 3) were all statistically significant and of the "correct" sign. In the case of cocaine/crack use, the advertising variables were also significant in waves 2 through 4. The estimated advertising coefficients in the bottom section of Table 3, however, were all statistically nonsignificant with the exception of the wave 4 marijuana volume-of-use equation. This suggests that recall of PDFA's antidrug advertising had little or no impact on the volume of use among existing users.

Finally, to ensure that the negative advertising coefficients imply that recall of advertising leads to lower marijuana and cocaine/crack use and are not due to omitted-variable bias (e.g., omission of variables such as exposure to other antidrug programs), we examined the correlation between the advertising-recall variable and the estimated equation error. This correlation was found to be statistically nonsignificant (according to a significance level of $P<.0001$) for each equation, suggesting that omitted-variable bias was not a significant problem.

Marginal Impact of Advertising

We also estimated the marginal impact of the advertising-recall variable[23] to determine the change in the probability of use associated with a 1-unit change in advertising recall. We estimated the cumulative impact on use probability given a particular wave's level of advertising awareness by subtracting the average predicted probability of use in the absence of PDFA advertising from the average predicted probability given the level of recall generated by PDFA advertising in that wave.

TABLE 3—"Stage 3" Advertising Impact Coefficients, Use Decision, and Light/Heavy Use Decision: Waves 2, 3, and 4

Equation and Variable	Wave 2	Wave 3	Wave 4
	Use Decision		
Marijuana			
Recall of advertising	−0.183	−0.305	−0.303
	(*P* = .043)	(*P* = .005)	(*P* = .001)
Ψ_{MJ}	3.429	2.973	3.581
	(*z* = 19.03)	(*z* = 15.731)	(*z* = 20.94)
Log-likelihood	−574.02	−410.19	−659.97
df	864	672	1184
χ^2 (*P*)	538.62 (.0000)	344.31 (.0000)	707.00 (.0000)
Cocaine/crack			
Recall of advertising	−0.205	−0.162	−0.421
	(*P* = .05)	(*P* = .05)	(*P* = .000)
Ψ_{CC}	3.490	2.665	3.600
	(*z* = 12.59)	(*z* = 10.13)	(*z* = 14.60)
Log-likelihood	−333.90	−216.27	−320.75
df	880	700	1211
χ^2 (*P*)	210.71 (.0000)	123.51 (.0000)	308.48 (.0000)
	Light/Heavy Use Decision		
Marijuana			
Recall of advertising	−0.087	−0.086	−0.406
	(*P* = .273)	(*P* = .330)	(*P* = .003)
Ψ_{MJ}	2.166	1.847	2.263
	(*z* = 5.73)	(*z* = 6.81)	(*z* = 8.00)
Log-likelihood	−214.40	−129.89	−193.90
df	315	191	276
χ^2 (*P*)	48.65 (.0000)	52.19 (.0000)	91.95 (.0000)
Cocaine/crack			
Recall of advertising	−0.048	−0.076	−0.289
	(*P* = .389)	(*P* = .400)	(*P* = .127)
Ψ_{CC}	2.946	2.531	3.004
	(*z* = 2.45)	(*z* = 2.00)	(*z* = 2.97)
Log-likelihood	−60.58	−37.36	−57.71
df	101	73	83
χ^2 (*P*)	9.46 (.0088)	4.41 (.1101)	14.05 (.0009)

Marginal effects of PDFA advertising on the probability of drug use were estimated to be 6.8%, 9.2%, and 6.5% for marijuana and 3.3%, 2.8%, and 2.5% for cocaine/crack across waves 2, 3, and 4, respectively. In wave 2, for example, a 1-unit change in advertising recall (measured on a 3-point scale) would have resulted in 6.8% and 3.3% reductions in the probability of using marijuana and cocaine/crack, respectively. Cumulative effects of PDFA advertisements were estimated to be 9.6%, 11.98%, and 9.25% for marijuana and 4.7%, 3.6%, and 3.6% for cocaine/crack, respectively, across the 3 waves. These measures suggest that, after 3 years of PDFA advertising, approximately 9.25% fewer adolescents were using marijuana.

DISCUSSION

Our results are consistent with the hypothesis that antidrug advertising reduces the probability of marijuana and cocaine/crack use among adolescents. However, our results also suggest that recall of antidrug advertising is not associated with adolescents' decisions regarding how much marijuana or cocaine/crack to use among those already using each drug.

This study was not without limitations. Although the sample was constructed to be representative of American adolescents, central-location sampling was used. Sudman[29] has shown that when central-location sampling is used carefully, it will provide close estimates of the total population. It is also possible that respondents were exposed to other antidrug intervention programs in addition to their exposure to antidrug advertising. However, past research has demonstrated that these alternative programs have been largely ineffective.[30]

Despite these potential limitations, our findings have important public policy implications. Our model, based on survey data from 1987 to 1990, indicates that increases in amounts of antidrug advertising are associated with decreases in teenage drug use. During this time period, media financial support for antidrug advertising increased, from a low of $115 million in 1987 to a high of $365 million in 1991.[31,32] Given our results, this increase appears to have been a worthwhile investment. ■

About the Authors

Lauren G. Block is with the Department of Marketing, Baruch College, New York City. Vicki G. Morwitz is with the Department of Marketing, New York University, New York City. William P. Putsis Jr is with the Department of Marketing, London Business School, London, England. Subrata K. Sen is with the Department of Marketing, Yale University, New Haven, Conn.

Requests for reprints should be sent to William P. Putsis Jr, London Business School, Regent's Park, London NW1 4SA, United Kingdom (e-mail: bputsis@london.edu).

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Contributors

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Human Participant Protection

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