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Chapter 8

DRUG USE AMONG THE SPANISH ADOLESCENTS: TESTING FOR A CAUSAL GATEWAY EFFECT

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ABSTRACT

The use of drugs represents a particularly worrying public health problem among adolescents. In this article we present evidence in favour of the causal gateway effect in the use of drugs among the Spanish adolescent population. We use instrumental variables in order to consistently estimate two-stage probit models from the information provided by three waves of the Spanish Surveys on Drug Use in the School Population (1996, 1998 and 2000). After treating the identification problem by means of the natural experimentation methodology, our results indicate that smoking cigarettes does indeed causally induce the use of marijuana with such use, in turn, increasing the probability of the adolescent taking the ultimate step, that is to say, hard drug use. We also provide evidence which confirms the herd effect among this age-group as a reason for starting to consume tobacco and marijuana, although not for the use of hard drugs. As a conclusion, not only does the observed gateway effect in drug use among Spanish adolescents correspond to a spurious correlation, but also to a causal mechanism.

1. INTRODUCTION

The consumption of drugs, whether these are legal or illegal, is related to a number of significant socio-economic aspects. Thus, such consumption may initially offer the consumer a range of pleasant sensations, making him feel happier and, at least for a short period of time,

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helping him to forget his daily problems. However, in spite of this first reaction, a pattern of consumption that leads to continuous addictive use results in multiple physical and psychological problems. For example, prolonged tobacco use causes heart disease and a whole range of carcinomas, such as cancers of the lung, mouth, larynx or oesophagus (Bartecchi, et al., 1994). For its part, the consumption of illegal drugs causes compulsive and obsessive personality dysfunction, as well as diminishing human-capital formation among young people, and having a negative effect on their psychomotor performance (Hawkins, Catalano, and Miller, 1992).

Focusing particularly on this age-group, it has been confirmed that drug consumption represents an integration element inside the peer group, as well as a symbol of liberation and independence from the adult world (Spanish National Committee on the Prevention of Smoking, 1998, *Comité Nacional para la Prevención del Tabaquismo, 1998*). Moreover, drug consumption, above all of illegal substances, imposes specific costs on society in the form, for example, of increased health costs and crime, as well as employment or school difficulties (Duarte, Escario, and Molina, 2006). In short, the use of drugs is a social and economic problem of the first magnitude, with this being especially true in the case of the adolescent population, given the psychologically vulnerable nature of this age group. It should come as no surprise, therefore, that an increasing body of economic literature has focused on analysing drug consumption with the objective, among others, of proposing different policy initiatives aimed at reducing both drug consumption and the external costs generated by this harmful habit (Gill and Michaels, 1991; Saffer and Chaloupka, 1999; Pacula et al., 2000)

In this context, an aspect of the analysis that has acquired increasing importance over the last few years is the gateway effect, given the significant role this plays in evaluating the effectiveness of such initiatives (DeSimone, 1998; Pacula, 1998; Beenstock and Rahav, 2002; Pudney, 2003; Pudney, 2006; Fergusson, Boden, and Horwood, 2006). According to this approach, initially developed by Kandel (1975), young people first begin by consuming tobacco, then move on to marijuana and, finally, end up consuming hard drugs (amphetamines, cocaine, heroine, LSD, or design drugs). At this point, a crucial question arises, namely to determine whether this sequence, or gateway effect, is causal or simply descriptive. If it is causal, then policy initiatives aimed at reducing the consumption of a given addictive substance will have the effect of reducing the consumption of harder drugs that appear later in the sequence. By contrast, if it is only descriptive, then it may be the case that young people who are more likely to consume tobacco have a greater probability of consuming marijuana, independent of whether or not they actually smoke in an earlier stage of the sequence. In this latter case, tobacco consumption, for example, will not induce the consumption of marijuana, in such a way that policy initiatives designed to reduce tobacco consumption will not affect that of marijuana. Therefore, it can be argued that the effectiveness of policies directed towards reducing the consumption of drugs will be more effective in cases where the gateway effect is found to be causal, given that any measures aimed at decreasing the consumption of an initiation drug would not only reduce the consumption of this addictive substance, but also of the continuation drug.

Against this background, the objective of this paper is to analyse drug consumption among the adolescent population, with emphasis being placed on testing the causal nature of the gateway effect. To that end, we formulate two-stage probit models which will be estimated by using instrumental variables from the statistical information provided by three cross-sections, corresponding to 1996, 1998 and 2000, of the Spanish Surveys on Drug Use in

the School Population (SDUSP), carried out by the Spanish Government's Delegation for the National Plan on Drugs. After specifying the equations which model the consumption of both the initiation drug (tobacco or marijuana) and the continuation drug (marijuana or hard drugs), our two-stage procedure provides consistent estimations of these equations. Thus, whilst in the first stage the initiation drug equation will be estimated by a probit model, in the second we will substitute this initiation drug in the continuation drug equation by the estimated probability of the initiation drug obtained in the first stage. Our empirical results will hopefully allow us to obtain a better understanding of the reality of legal and illegal drug use among the Spanish adolescent population, which must represent the appropriate starting point when seeking to achieve the goal of effective prevention.

The rest of the work is organised as follows. In Section 2 we offer a detailed description of the empirical model. The data used in this study is considered in Section 3. In Section 4 we provide estimations of the proposed model and, finally, Section 5 closes the paper with a review of the main conclusions.

2. THE MODEL

According to the gateway effect, adolescents start by consuming tobacco, then move on to marijuana and, finally, end up using hard drugs, such as cocaine, heroine, amphetamines, LSD or designer drugs. Given that the main objective of this paper is to test whether or not this gateway effect is causal, we are interested in two hypotheses. The first is if tobacco consumption increases the probability of using marijuana, while the second is if marijuana consumption induces the subsequent use of hard drugs. In order to respond to these two questions, we begin by specifying the following notation, where the initiation drug, that is to say, the drug that we will test to determine whether it subsequently causes the consumption of another drug, is denominated as *ID*, whilst this other drug, which we call the continuation drug, is denominated as *CD*. In this way, we can define the dichotomic variables ID_{it} and CD_{it}:

$$ID_{it} \begin{cases} = 1 \text{ if the ith adolescent consumes the initiation drug in period t} \\ = 0 \text{ otherwise} \end{cases} \quad (1)$$

$$CD_{it} \begin{cases} = 1 \text{ if the ith adolescent consumes the continuation drug in period t} \\ = 0 \text{ otherwise} \end{cases} \quad (2)$$

Associated with this last variable, we have a latent variable CD_{it}^{*}, whose sign determines the value of CD_{it}, and which can be modelled in the following way:

$$CD_{it}^{*} = \beta_C XC_{it} + \alpha ID_{i,t-1} + u_{it} \quad (3)$$

where β_C is a vector of parameters associated with the explanatory variables XC_{it}, $ID_{i,t-1}$ indicates if the adolescent has consumed the initiation drug in the previous period and, finally, u_{it} is an error term. In accordance with this formulation, we have:

$$Pr\,(CD_{it}=1) = Pr\,(CD_{it}^{*}>0) = Pr\,(u_{it} > -(\beta_C XC_{it} + \alpha\, ID_{i,t-1})) \tag{4}$$

in such a way that testing whether or not the initiation drug increases the probability of consuming the continuation drug is equivalent to testing the hypothesis $\alpha > 0$. However, the standard estimation of the previous parameter is not consistent, given that the variable $ID_{i,t-1}$ is probably correlated with the error term. To appreciate this point more fully, we can think in terms of an additional equation to model the decision to consume the initiation drug:

$$ID_{it}^{*} = \beta_I XI_{i,t-1} + v_{i,t-1} \tag{5}$$

where β_I is a vector of parameters associated to the explanatory variables $XI_{i,t-1}$, with the XI vector probably containing several elements in common with the vector of variable XC and, finally, where $v_{i,t-1}$ is an error term. Obviously, it is reasonable to think that the two random terms of (3) and (5) will present a positive correlation, that is to say, $E(u_{it},v_{i,t-1})>0$, given that there will be unobservable characteristics of the adolescent, such as the time preference rate, antisocial behaviour, peer pressure, etc., that will either increase or reduce the probability that the individual consumes both the initiation and the continuation drugs.

Given the above, the estimation of α will present a positive bias, in such a way that obtaining a positive coefficient will not always guarantee that the consumption of the initiation drug increases the probability of consuming the continuation drug. To illustrate this point, let us consider for a moment that the true value of α is zero but, due to the bias, the obtained estimation of the coefficient is positive. In this case, there is no effect of one drug on another, with the only point being the presence of non-observable variables that influence the probability of consuming both substances. Therefore, whilst the continuation drug follows the initiation drug, it does not do so by means of a cause-effect mechanism, but rather by way of one that simultaneously causes the consumption of both drugs, first one and then the other. This difference is very important since, in the first case, it will be possible for the legislator to exert an influence over the probability of consuming the continuation drug by means of policy measures aimed at reducing the number of adolescents who consume the initiation drug, whilst in the second case, such measures clearly would not work.

In order to solve this identification problem, we apply an estimation procedure according to which we instrument the explanatory variable that causes this problem, that is to say, *ID* in equation (3). More specifically, we follow Beenstock and Rahav, (2002) and substitute *ID* in equation (3) for an instrumental variable that we obtain from the estimation of equation (5), that is to say, the estimated probability of consuming the initiation drug or $Prob\,(ID^{*} > 0)$. In this case, Beenstock and Rahav, (2002) argued that a two-stage procedure provides consistent estimations of the parameters in both equations (3) and (5). In the first stage, equation (5) is estimated by a probit model whilst, in the second, the estimated probability of *ID* obtained from the first stage is used to replace *ID* in equation (3).

More particularly, when testing for the first hypothesis, that is to say, when we estimate the marijuana consumption equation, as specific instruments we use the price of tobacco and the proportion of consumers present in the particular region in which the adolescent resides. By following this procedure, tobacco consumption is instrumented by constructing a variable in such a way that its effect on the decision to consume marijuana can be identified, given that the problem of endogeneity has been taken into account. As regards the second hypothesis, namely the existence of a gateway effect between marijuana consumption and hard drug use, the price of marijuana is not available. As a result, and in order to instrument the marijuana indicator, as specific instrumental variables we use the predicted tobacco consumption and the proportion of consumers of marijuana that live in the region in question.

3. Data

The data used in this work comes from the three waves of the Spanish SDUSP corresponding to 1996, 1998 and 2000, and carried out by the Spanish Government's Delegation for the National Plan on Drugs. These three waves contain information on 19,191, 18,346 and 20,450 adolescents, respectively, aged between 14 and 18, covering both individual and family socioeconomic characteristics, as well as other factors related, for example, to the effects of available information on the consequences of illegal drug consumption. All this information was obtained directly from the adolescents surveyed, who anonymously answered a complete questionnaire on drug use. Their parents were not present during the interviews and were not informed about the responses of their children, in this way avoiding any underreporting in their responses to illegal drug use or other questions. The information was collected in different public and private centres of secondary education and vocational training. To ensure a representative sample, a random selection procedure was used in order to determine the two classrooms-by-center where the adolescents were to be interviewed.

Mean and standard deviations of the variables appear in Table 1. The dependent variables are *Tobacco, Marijuana* and *HardDrugs,* with these indicating if the adolescent has used tobacco, marijuana or hard drugs (cocaine, heroine, amphetamines, LSD or design drugs) during the last 30 days. With respect to the independent variables, we first include physical characteristics (*Gender* and *Age*), the education level of the parents (*StudiesFather*, *StudiesMother*) and also the *MonoParental* variable, which takes the value 1 if the adolescent lives in a household where either the father or the mother are absent and 0 otherwise. Other variables try to measure significant aspects in the adolescent's environment, namely the weekly disposable income (*Income*) or the effect of information provided at school or vocational training centre on the dangers of drug consumption (*Information*). Additionally, in each of the three equations we add the regional proportion of consumers of this substance (*Tobacco%, Marijuana%* and *HardDrugs%)* and, in the case of the marijuana and hard drug equations, we also include the estimated probability of consuming the initiation drugs, that is to say, tobacco and marijuana, respectively. Furthermore, in the case of tobacco consumption we incorporate a price index that reflects the cost of tobacco during the period when the adolescent was at the mean age at which these young people begin to smoke (13 years old). This index has been obtained from the Spanish National Institute of Statistics after having divided the tobacco price index by the general price index. Similarly, in order to control for

regional unobservable differences, dummy variables corresponding to the 17 autonomous regions in which Spain is divided are introduced into all specifications, given that their omission could attribute the effects of regional characteristics to the socio-economic variables. Finally, we have included two time dummy variables, corresponding to the first two cross-sections.

Table 1.Variable definitions.

Variable	Definition	Mean (Std. Dev.)
Tobacco	This takes the value 1 if the adolescent has smoked cigarettes during the last 30 days and 0 otherwise	0.293 (0.455)
Marijuana	This takes the value 1 if the adolescent has used marijuana during the last 30 days and 0 otherwise	0.174 (0.379)
HardDrugs	This takes the value 1 if the adolescent has used hard drugs (cocaine, heroine, amphetamines, LSD or designed drugs) during the last 30 days and 0 otherwise	0.047 (0.211)
TobaccoPrice	The price of tobacco when the adolescent was at the mean age at which he/she begins to smoke (13 years old) (in constant 2000 euros)	0.497 (0.071)
Tobacco%	This variable measures the proportion of tobacco users in the region where the adolescent lives.	0.313 (0.084)
Marijuana%	This variable measures the proportion of marijuana users in the region where the adolescent lives.	0.181 (0.054)
HardDrugs%	This variable measures the proportion of hard drugs user in the region where the adolescent lives.	0.048 (0.017)
Gender	This takes the value 1 if the adolescent is male and 0 if female	0.483 (0.500)
Age	Age of adolescent	15.602 (1.229)
MonoParental	This takes the value 1 if the adolescent live without his/her father or mother and 0 otherwise	0.099 (0.299)
StudiesFather	This takes values according to the father's studies level (1: no studies, 2: basic school certificate, 3: secondary school certificate; 4: first level of vocational training, 5: second level of vocational training; 6: superior secondary school certificate, 7: University diploma, 8: University degree)	3.954 (2.424)
StudiesMother	This takes values according to the mother's studies level (1: no studies, 2: basic school certificate, 3: secondary school certificate; 4: first level of vocational training, 5: second level of vocational training; 6: superior secondary school certificate, 7: University diploma, 8: University degree)	3.571 (2.244)
Income	Adolescent's available income per week (in constant 2000 euros)	12.919 (14.310)
Information	This takes the value 1 if the adolescent studies at a school which has information campaigns on the risks associated with drug consumption and 0 otherwise	0.543 (0.498)

From a reading of this Table, we can first appreciate that 29.3%, 17.4% and 4.7% of the sample adolescents have consumed tobacco, marijuana or hard drugs, respectively, during the last 30 days. As regards the independent variables, we can note that 48.3% of the sample

corresponds to male adolescents and that the average age of the entire sample is 15.6 years. Furthermore, it emerges that 9.9% of sample adolescents live in a mono-parental family. The average income is 12.92 euros/week (in constant 2000 values) and some 54.3% of the sample adolescents have received information on the risk factors associated with drug use by means of informative campaigns carried out at their school or vocational training centre.

4. Empirical Results

Given that our objective is to test for the existence of a causal gateway effect between tobacco and marijuana, on the one hand, and between marijuana and hard drugs, on the other, we first estimate a tobacco consumption equation, which appears in the first results column of Table 2. This column confirms that the tobacco price, although helping us to instrument the tobacco indicator in the marijuana estimation, does not appear to be significant, probably because there are only four years of time variation in our sample, from 1996 to 2000, in such a way that the price change is not big enough to have some perceptible effects. By contrast, one significant result that emerges is the confirmation of the peer group effect, as shown by the positive and significant coefficient of the *Tobacco%* variable. This is indicating that young people who live in regions where there is a higher proportion of smokers and, as a consequence, who have more peers at school who smoke, have a higher probability of themselves being a smoker. With respect to the physical characteristics, we can see that the proportion of adolescent smokers is higher among females than among males and, moreover, that the probability of being a smoker increases with age. Similarly, the fact that the adolescent lives in a household where one of the parents is absent (maybe because one spouse has died or because the marriage has broken down) and the level of disposable income would, according to our results, appear to increase the probability of smoking. By contrast, the education level of the mother and the fact that the school organizes information campaigns about the negative consequences of drug consumption seems to reduce the number of adolescents who decide to smoke.

The results corresponding to the consumption of marijuana are set out in the second results column of Table 2. Here, the large and highly significant positive coefficient of the *EstimatedProbabilityTobaccoConsumption* variable reflects the existence of a causal gateway effect from tobacco to marijuana, that is to say, there would appear to be a causal mechanism which indicates that smoking cigarettes induces marijuana consumption (the same result was found by Beenstock and Rahav, 2002), in such a way that policy measures aimed at reducing the number of tobacco smokers will also reduce the number of marijuana users. Our results also confirm the peer group effect, according to which some adolescents start to use marijuana because their peers do so and they feel the pressure to do likewise. As regards the remaining variables, we can note that the marijuana use is higher among adolescent males and that it increases with age. By contrast to the tobacco equation, the education level of the parents does seem to have a positive effect on the probability of using marijuana. Although this is an apparently contradictory result, it can be justified given that marijuana consumption was very popular among students who were at university in the 1970s and 1980s, and who are now parents, with these individuals perhaps exhibiting a more tolerant attitude than parents who did not go to university during this era. Other differences between the tobacco and marijuana equations are that the income and information variables do not now appear to be

significant and, secondly, that the time dummy variables are now significant, indicating that marijuana consumption increased markedly over the length of the three sample years.

Table 2. Two-stage estimates

Variable	Tobacco	Marijuana	HardDrugs
Intercept	-11.377***	-13.738***	-4.028
	(-7.956)	(-8.141)	(-1.460)
TobaccoPrice	-0.055	-	-
	(-0.082)	-	-
Tobacco%	3.370***	-	-
	(8.449)	-	-
EstimatedProbabilityTobaco-	-	2.782***	-
Consumption	-	(11.295)	-
Marijuana%	-	1.624**	-
	-	(1.995)	-
EstimatedProbabilityMarijuana-	-	-	2.026***
Consumption	-	-	(6.584)
HardDrugs%	-	-	3.968
	-	-	(1.177)
Gender	-0.377***	0.469***	0.121***
	(-23.959)	(13.164)	(4.038)
Age	1.079***	1.399***	0.177
	(6.504)	(6.724)	(0.527)
AgeSquared	-0.029***	-0.043***	-0.005
	(-5.540)	(-6.653)	(-0.499)
MonoParental	0.177***	0.060	0.063
	(5.922)	(1.611)	(1.145)
StudiesFather	-0.001	0.017***	0.001
	(-0.159)	(3.510)	(0.094)
StudiesMother	-0.015***	0.015***	-0.019**
	(-3.110)	(2.651)	(-2.276)
Income	3.688***	-0.030	1.183***
	(30.233)	(-0.127)	(4.350)
IncomeSquared	-2.778***	0.007	-0.344**
	(-21.155)	(-0.060)	(-2.314)
Information	-0.109***	-0.007	-0.036
	(-6.848)	(-0.310)	(-1.175)
T96	-0.016	-0.125***	0.078**
	(-0.186)	(-5.561)	(2.138)
T98	0.001	-0.055***	0.029
	0.020	(-2.415)	(0.813)

t-statistics are in parentheses. *** significant at the 1% level. ** significant at the 5% level.

With the aim of verifying whether the consumption of marijuana induces the use of other hard drugs, the third results column of Table 2 presents the hard drugs estimation. As can be appreciated from the significant and positive effect of the *EstimatedProbabilityMarijauanaConsumption* variable, our estimations again appear to offer support for the causal gateway effect, showing that the use of marijuana, other things being

equal, increases the probability that the adolescent will go on to consume harder drugs, for example, amphetamines, cocaine, heroine, LSD or designer drugs. Similar results appear in DeSimone (1998), who find a causal peer effect from cannabis to cocaine and in Fergusson, Boden, and Horwood (2006) who find a causal peer effect from marijuana to several illicit drugs other than cannabis (including among others methamphetamine, barbiturates, heroin, morphine, cocaine LSD).

If we now focus on the remaining exogenous variables, the probability of using hard drugs is greater among adolescent males, decreases with the studies level of the mother and increases with disposable income. With respect to the *Information* variable, we can note that the coefficient is negative, but not significant, as was the case in the tobacco equation. Thus, we can conclude that the mounting of information campaigns at school or vocational training centres on the consequences of drug consumption only has the effect of significantly reducing tobacco consumption. However, given the gateway effect between tobacco and marijuana, on one hand, and between marijuana and hard drugs, on the other, it could be argued that these campaigns would not only reduce the proportion of smokers, but also of those who use marijuana and hard drugs. It is precisely this latter result which highlights the importance of distinguishing between causal and descriptive effects at policy-making level.

5. Summary and Conclusions

In this paper we have set out to test the causal nature of the gateway effect between tobacco consumption and the subsequent use of marijuana and, thereafter, hard drugs. To that end, we have employed two-stage probit models which have been estimated by using data drawn from three waves of the Spanish Surveys on Drug Uses in the School Population corresponding to 1996, 1998 and 2000.

The use of instrumental variables has allowed us to conclude that the observed gateway effect in drug consumption among Spanish adolescents responds to a causal mechanism, rather than only resulting from a spurious correlation induced by unobserved heterogeneity. Thus, we have found that tobacco smoking induces marijuana use and this, in turn, leads to the consumption of hard drugs, such as cocaine, heroine, LSD, amphetamines and design drugs. These results, which are in accordance with those published earlier in the literature (see Beenstock and Rahav (2002), for the case of our first gateway hypothesis, that is to say, from tobacco to marijuana and, secondly, DeSimone (1998) and Fergusson, Boden, and Horwood (2006), for the case of our second gateway hypothesis, more concretely from marijuana to illicit drugs), have important consequences for policy makers. For example, the effects of tobacco regulation (taxes, restrictions on sales, etc.) will not only modify smoking, but will also have an impact on marijuana consumption and, subsequently, on the use of hard drugs. At the same time, if, for example, marijuana is decriminalized and, as a consequence, changes in the consumption of marijuana emerge, this measure will also have effects on the consumption of hard drugs. Additionally, we have clearly confirmed the effect of peer group pressure, in such a way that adolescents who live in one of the regions of Spain where there is a higher level of drug use among the student population are more likely to themselves consume such a substance.

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