HEDG Working Paper 07/01

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January 2007

ISSN 1751-1976

york.ac.uk/res/herc/hedgwp

Is cannabis a gateway to hard drugs?

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Abstract

The gateway hypothesis proposes that cannabis use increases the risk of starting to consume hard drugs. We test this controversial, but influential, hypothesis on a sample of cannabis users employing a unique set of drug prices. A flexible approach is developed to identify the causal gateway effect using a bivariate survival model with shared frailty estimated using a latent class approach. The model suggests two distinct groups; a smaller group of "troubled youths" for whom there is a statistically significant gateway effect that doubles the hazard of starting to use hard drugs and a larger fraction of "most youths" where previous cannabis use has little impact.

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Cannabis is sometimes said to be a gateway drug that increases the users' probability of taking up hard drugs like amphetamine or heroin. The empirical basis for the hypothesis is the common finding that most hard drug users have started with less dangerous drugs first and that there seems to be a staircase from alcohol and solvents via cannabis and tablets to amphetamine, cocaine and heroin (see e.g., Denise B. Kandel, 1975). Although controversial, the hypothesis has had considerable influence on drug policy and legislation in many countries and has been a powerful argument in debates about legalization or decriminalization of cannabis.

In contrast to most other Western societies, the Netherlands has for long aimed at separating the markets for soft and hard drugs by allowing "coffee shops" to legally sell cannabis while keeping a strict regime against the trade of hard drugs like cocaine and heroin. More recently other countries like Portugal and the UK have experimented with changes in their drug legislation too. Re-classifying cannabis to a class-C drug, as was implemented in the UK in 2004, was a step in the direction of de-criminalizing the drug. Similar developments have taken place in some US states and cities. For instance, after a referendum California accepted medical uses of marijuana in 1996 and in 2005 the city of Denver voted to legalize possession of small quantities of cannabis. In Australia, four of the eight states and territories have de-criminalized use and cultivation of cannabis plants for personal consumption, the most recent change came in Western Australia in 2004. Liberalizing cannabis laws may increase the number of users as it will lower the price of using the drug and probably increase the physical and cultural availability (see e.g. Jenny Williams (2004) for a review of cannabis participation studies). Whether such liberalization will lead to an increase in the number of hard drug users depends, among other things, on whether there is a gateway effect or not. The purpose of this paper is to test the hypothesis to see whether there is evidence of such a causal link between the uptake of cannabis and hard drugs.

There are different pathways that might be the basis for a causal gateway effect in drug use (see e.g., Stephen Pudney, 2003). The first is an addiction effect: use of soft drugs may create a psychological or physiological need for stronger narcotic experiences. The second is an access effect: obtaining and using soft drugs may bring the user into contact with hard-drug users or suppliers whom they would not otherwise have met. The third is a credibility effect: experience of the use of soft drugs with no obvious ill effects may appear to contradict negative publicity against the use of illicit drugs in general, so that advice against hard drugs becomes less persuasive. In addition there may be an adjustment cost effect: for some individuals consuming any illicit drug may cross a psychological threshold that makes it less costly to proceed into another drug stage.

Testing for a gateway effect with retrospective survey data poses a statistical challenge as it involves dealing with selection bias due to unobserved individual heterogeneity. The apparent causal relationship in the observed association may be spurious since unobserved factors may influence both the probability of cannabis use and the use of other drugs. For instance, a traumatic childhood may be causally important for both cannabis and later heroin use. If this factor is unobserved, the effect of childhood trauma will be picked up by the variable for cannabis use. It would be misleading to conclude that cannabis is a gateway drug since it picks up the effects of an omitted third variable.

Two recent tests of the hypothesis report that the gateway effect of cannabis is greatly reduced after taking unobserved heterogeneity into account, even to the point of not being statistically significant (Stephen Pudney, 2003, Jan C. van Ours, 2003). Other recent studies claim, on the other hand, that although the gateway effect is reduced when unobserved heterogeneity is taken account of, there is still a significant association (David M. Fergusson and L. John Horwood, 2000, David M. Fergusson et al., 2006). Hence, the core question of

whether the observed sequential pattern of drug initiation is due to correlation or causality remains unresolved.

This paper builds on this literature and makes new methodological and empirical contributions. The paper develops a new approach for testing the hypothesis using a latent class bivariate hazard model in which both the intercept and all slope coefficients are estimated separately for each latent class. The main difference between this approach and the ones employed by Jan C. van Ours (2003) and Stephen Pudney (2003), is that the individual heterogeneity is allowed to affect the marginal effects of all the variables and not only the random intercept. In this sense, it imposes fewer restrictions on the model that is used to test the gateway hypothesis.

Previous studies have attempted to identify the gateway effect by comparing patterns of initiation of hard drugs between cannabis users and non-users, effectively using non-users as the control group. This approach is problematic when there is unobserved heterogeneity that makes users and non-users systematically different from each other: comparing treatment and control groups fails to compare like-with-like. In contrast, our strategy is to restrict the analysis to those who have used cannabis at some point in their lives and to exploit variation in the timing of cannabis and hard drug initiation to identify the gateway effect. This avoids the problem of having a non-comparable control group. There may still be systematic unobservable heterogeneity within the group of cannabis users, but this can be dealt with by using a bivariate hazard specification with shared frailty.

The empirical results benefit from a unique set of data on drug prices collected through face-to-face interviews with people visiting a needle exchange service in the city of Oslo, Norway. Data on drug prices are rare, especially price information provided directly by users. Changes in the relative prices of drugs may affect the choice of which drug to use first (if at all) and for this reason reliable information about prices are important. Previous

contributions have either adopted approaches in which price variables were ignored or have tried to create proxies for prices.

Our findings demonstrate, first of all, that there is a gateway effect and the hazard of taking up hard drugs doubles after the initiation of cannabis. The partial effects from a univariate hazard model show that the gateway effect typically increases the hazard of starting to use hard drugs by about 0.016 after controlling for the influence of other observable factors. Secondly, the results indicate the importance of taking unobserved heterogeneity into account. Doing so using latent class analysis suggests that there is one group of "troubled youths" for whom cannabis use has a statistically significant effect. In this group the recent use of cannabis increases the hazard of starting to use hard drugs by 0.026. The relative gateway effect is substantial and again the hazard of taking up hard drugs almost doubles after the person has initiated cannabis use. For the second group – the "most youths" - use of cannabis makes little difference to the hazard of using hard drugs. The marginal effect is not as large and not significant at the 5% level. Thus, the analysis indicates that the overall gateway effect is created by a large effect in a small group and a small or non-existent effect for most people.

I. Identifying the gateway effect

To identify a possible causal effect of cannabis on subsequent use of hard drugs one needs to account for self-selection into the group of cannabis users. Ideally, to single out the effect of cannabis use, one would like to know the counterfactual outcome for cannabis users: what the probability of hard drug use would have been if they had not started to use cannabis in the first place. In practice this counterfactual cannot be observed and, in the absence of

randomized experiments, attention must focus on alternative estimation strategies for handling the potential endogeneity of the gateway variable.

One response to the problem of spurious correlation between the use of cannabis and hard drugs is to adopt a selection on observables strategy and include as many as possible of the potential confounders in the analysis as control variables. Kazuo Yamaguchi & Denise B. Kandel (1984) and David M. Ferguson & L. John Horwood (2000) are two studies that have included a wide range of variables that are assumed to influence drug use and deviant behavior. A problem with this approach is that many variables affecting drug use are simply unavailable or are very difficult to measure. For instance, in addition to childhood traumas, time preferences are sometimes argued to be an important causal factor in the decision to use illegal substances (George Ainslie, 1992). Although it is possible to measure (indicators of) time preference, this is seldom done in large surveys of drug use in the population. Similarly, studies of twins suggest that genetic factors are important in determining an individual's use of illegal drugs (Michael J. Lyons et al., 1997), but this is, for all practical purposes, an unobserved variable for researchers who use general questionnaire-based surveys. In short, there are good reasons to expect that unobservable factors like emotional experiences, time preferences and genetics are important in the decision to use drugs and therefore it is necessary to take these unobservables into account when testing the gateway hypothesis that cannabis is a stepping stone to harder drugs.

One well-known approach is the instrumental variable (IV) technique. For instance, Rosalie L. Pacula (1998) uses past prices of alcohol as instruments for previous consumption of the drug and estimates a gateway effect of alcohol on current use of marijuana. Other examples include Jeffrey DeSimone (1998), who uses information on individual characteristics and local alcohol prices as instruments, and Michael Beenstock and Giora Rahav (2002), who apply variants of the IV approach to sequences of events using prices by

birth cohorts as instruments. The main problem with the IV approach is finding valid instruments. From an economic perspective, prices provide relevant instruments and alcohol and cigarette prices have been used frequently. These prices vary over time and between countries and states, but they cannot reflect contemporaneous individual differences in behavior within the same area. Prices of illicit drugs are, in addition, hard to obtain. Credible instruments for previous consumption that are not based on prices are rare. Altogether this does not imply that the instrumental variable approach should be rejected, but that is seems worthwhile to explore alternative approaches. Our access to unique data on drug prices is used as part of our identification strategy but we also adopt a method that does not necessarily rely on exclusion restrictions and hence the need for instruments.

Depending on the types of data at hand and assumptions one is willing to impose, there are various ways to take account of unobserved factors. It is frequently assumed that unobserved variables are stable over time, specific to the individual and influence a range of behaviors.[1] When analyzing retrospective survey data using duration models, one needs to create time-to-event data by taking advantage of questions that relate to events that have occurred at some earlier point in time. The structuring of the data as a panel is based on an underlying approach in which one uses bivariate or multivariate hazard models. Analysing two or more behaviors simultaneously allows the unobserved heterogeneity to be captured. Employing models of this kind usually requires exclusion restrictions (James Heckman and Bo Honorè, 1989) but an important paper by Jaap H. Abbring and Gerard J. van den Berg (2003) shows that sample variation in the timing of treatments and outcomes provides identifying information in hazard models without the need for such exclusion restrictions.

[1] One alternative is to employ fixed effects models as did David M. Ferguson and colleagues when they examined the gateway hypothesis (David M. Ferguson, Joseph M. Boden and L. John Horwood, 2006). This fixed effects approach requires panel data and cannot be used to model 'one-off' decisions such as the initiation of drug use.

Both Jan C. van Ours (2003) and Stephen Pudney (2003) employ retrospective survey data and refer to Abbring and van den Berg's (2003) result as a source of identification in their hazard model specifications. Jan C. van Ours (2003) analyses the use of cannabis and cocaine by employing a finite mixture approach to model the unobserved heterogeneity. Stephen Pudney (2003) takes a broader approach and analyses six categories of deviant behavior simultaneously including minor offending and serious crime in addition to solvent abuse, soft drug use, social drug use and hard drug use. He employs a discrete time hazard model and a parametric specification of the unobserved heterogeneity which is estimated using maximum simulated likelihood estimation.

Given the nature of the information on drug use available in the Oslo survey data and the unique set of price data for illicit drugs at hand we too have chosen a hazard model approach. This means that identification of the causal gateway effect could be based solely on the timing of events, as demonstrated by Abbring and van den Berg. Compared to Pudney (2003) and van Ours (2003), we impose fewer restrictions on the model by allowing the unobserved variables to influence all the parameters of the model not just the intercepts.

Our focus is on testing whether cannabis is a gateway drug and we have chosen to merge use of amphetamine, cocaine and heroin into one cluster for hard drugs; we test whether recent cannabis use increases the probability of starting to consume a hard drug irrespective of which type of hard drug that might be.

A. *The model*

Our starting point is a bivariate mixed proportional hazard model with shared frailty of the type discussed by Jaap H. Abbring and Gerard J. van den Berg (2003) in their approach to the identification of treatment effects with duration data. In our application the treatment is use of the gateway drug and the outcome of interest is initiation of hard drug use. The first equation

defines the hazard for initiation of cannabis use at a certain age, while the second equation estimates the hazard for initiation of hard drug use:

$$(1) \qquad \begin{aligned} h_{it}^{1} &= \lambda_1(t)\phi_1(x)u_1 \\ h_{it}^{2} &= \lambda_2(t)\phi_2(x)\delta(t \mid s,x)u_2 \end{aligned}$$

h_{it}^{j} is the hazard of individual (i) starting to use drug j (1=cannabis, 2=hard drugs) at age t, given the values of the covariates (x) and the shared frailty associated with an individual that affects both cannabis and hard drug use (u_j). $\lambda_1(.)$ and $\lambda_2(.)$ are the baseline hazards for cannabis and hard drug use. The function $\delta()$ captures the treatment effect, which depends on elapsed time (t), treatment time (s) and potentially on (t-s) the time between treatment and outcome. This formulation also allows for interactions with the covariates (x). This emphasis on the use of the timing of events to identify the treatment effects is exemplified by Jan van Our's (2003) use of the concept of an incubation period to identify the gateway effect. He defines an indicator variable for whether use of cannabis had been initiated within a given number of years (the incubation period). We adopt a similar approach, using four years as the incubation period in our baseline model.

The observed covariates (x) used in this paper can be categorized in three groups. First, to isolate the effect of cannabis, we control for time-dependent covariates (the price of cannabis, amphetamine, cocaine and heroin). Second, given the correlation between gender, childhood problems and drug use, we have included gender and self-reported measures of serious childhood problems with parents, friends, police and school. Third, duration dependence is modelled using a cubic function of time (t), as in Stephen Pudney (2003).

The likelihood function for the model defined above will depend on the sampling scheme. In our case there is stock-sampling; the sample consists of individuals all of whom

are initially at risk of starting both cannabis and hard drugs and these individuals are then followed over time. To deal with this we adapt the approach proposed by Stephen Jenkins (1995) for discrete time hazard models. He has shown how the complex sequence-likelihood function that results from stock sampling schemes can be simplified and estimated as a standard binary choice model. The procedure depends on reorganizing the data so that each individual (1,…, n) is associated with multiple observations – one at each point in time from the initial period until either the individual starts to use the drug or the time of the survey interview if they are a right censored observation (t_i=1…t_{ij}). A new binary variable y_{it} is created that equals 1 for the period at which drug use begins and 0 otherwise. For those individuals who do not start within the survey period y_{it} always equals 0. For those who start, y_{it} only equals 1 in the final period and subsequently the individual is dropped from the sample for all remaining periods. Using this reorganization of the data, Jenkins shows that the likelihood for a univariate discrete-time hazard function can be written as:

$$\log L = \sum_{i=1}^{n}\sum_{t=1}^{t_{ij}} y_{it} \log\left[\frac{h_{it}}{1-h_{it}}\right] + \sum_{i=1}^{n}\sum_{t=1}^{t_{ij}} \log[1-h_{it}]$$
$$= \sum_{i=1}^{n}\sum_{t=1}^{t_{ij}} y_{it} \log[h_{it}] + \sum_{i=1}^{n}\sum_{t=1}^{t_{ij}} (1-y_{it}) \log[1-h_{it}] \quad (2)$$

This log-likelihood takes the form of a standard binary choice model applied to the expanded dataset. Common choices of functional form for h_{it} are the complementary log-log model, which is the discrete-time equivalent of a continuous-time proportional hazard specification, and the logit model, which gives a non-proportional hazard specification.

This discrete time specification can now be extended to the bivariate model for both cannabis and hard drug use. The sample likelihood function for the bivariate model is:

(3) $$L = \prod_{i=1}^{n} E_u \left\{ \prod_{j=1}^{2} \left\{ \prod_{t=1}^{\tau_j - c_j} \left[1 - h_{it}^{j} \right] \right\} h_{it}^{j\,c_j} \right\}$$

where c_j is a dummy indicating a non-censored case. For each hazard function, the product runs from t=1 to t=τ-1 for uncensored observations and t=1 to t=τ for censored. An observation is censored if substance j hasn't been initiated by the end of the survey. The presence of unobservable heterogeneity (u) means that the two hazard functions must be estimated jointly and $E_u\{\}$ denotes the expectation over the distribution of unobserved heterogeneity. Stephen Pudney (2003) adopts a parametric approach by assuming that the u's are jointly normally distributed and using maximum simulated likelihood estimation to deal with the numerical integration. Jan van Ours (2003) adopts a semiparametric approach using a bivariate finite density estimator for u.

B. *The mixture model*

We adopt a latent class approach to deal with the unobserved heterogeneity in the hazard functions. This approach has several advantages over previously applied methods for dealing with unobserved heterogeneity in models of the gateway effect. One advantage is that we can relax the parametric assumptions that are necessary in other approaches that rely on Gauss-hermite quadrature or maximum simulated likelihood estimation. We do not make any assumptions about the parametric distribution of the unobserved heterogeneity. Another advantage is that the approach imposes fewer restrictions on the parameters of the model. For instance, in Pudney and van Ours, only the constant term in the regression is assumed to differ depending on whether or nor the person has a high or low value on the unobserved

characteristic. This is restrictive because there is no a priori reason to assume that the unobserved heterogeneity does not affect the marginal effects of the other variables. A person with a poor childhood, for instance, may react differently to changes in prices than a person with a normal childhood. Hence, this more general approach allows all coefficients to vary.

The latent class model assumes that each individual is drawn from one of K possible sub-groups or latent classes that exist in the population where π^k indicates the share of the population that belongs to the group *k*.[2] The parameters of the hazard function for each drug may be different depending on which group the individual is assumed to belong to.

Given that the individual belongs to one of the two (or more) groups, and since we do not know which, we may use π_k to denote the probability that individual *i* is a member of group k. The probability of an observed value for drug use (y_{it}^j) is then the sum of the probability of observing the value conditional on group membership, weighted by the respective probabilities of being in each group. In this case the log-likelihood function for the whole sample can be written as:

$$LogL = \sum_{i=1}^{n} \left\{ \sum_{k=1}^{K} \pi_k \left\{ \sum_{j=1}^{2} \left\{ \sum_{t=1}^{\tau_j - c_j} \log\left[1 - h_{it}^{jk}\right] + c_j \log\left[h_{it}^{jk}\right] \right\} \right\} \right\} \tag{4}$$

This approach to capturing unobserved heterogeneity helps us solve the problem of maximizing the likelihood function because it reframes the maximization problem in a way that allows us to use the EM algorithm (see for example, Shue Kay Ng et al., 2002).[3] This is

[2] Previous results indicate that relatively few classes are needed (see for example Partha Deb and Pravin K. Trivedi, 1997)

[3] The expectation (E) step uses Bayes' rule for posterior probabilities to give an updated and improved estimate of the probability that an individual is a member of each latent class and these estimates can be substituted for the unknowns in the likelihood function. When we have estimates for the unknown individual heterogeneity (interpreted as probabilities of being a

possible since the unobserved heterogeneity is integrated out of the likelihood, in this case by factoring the sum over latent classes. The probabilities of class membership are treated as parameters to be estimated.

II. The Oslo study

We have combined two different data sources. The first dataset was collected through postal questionnaires sent to a representative sample of 21-26 year olds living in Oslo in 2002. It provides information on the development of drug use initiation in a general population of youths. The data do not, however, contain any price information on illicit drugs so a second data source is required. The Norwegian Institute for Alcohol and Drug Research (SIRUS) has on a regular basis since 1993 conducted face-to-face interviews with drug addicts visiting a needle exchange service in Oslo and prices for various types and quantities of drugs have been recorded.

A. *Data for drug use*

The response rate for the postal questionnaires was roughly 50 per cent, with more women than men answering the questions (see Table 1). A reminder was sent and a total of 1984 questionnaires were registered. The respondents reported their experience with licit and illicit drugs in addition to information on age, gender and possible childhood problems with parents,

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member of a group) it is possible to use weighted estimation of the binary choice models to estimate the coefficients of the hazard functions using that particular set of probabilities of group membership (this is the maximization (M) step). These coefficients give rise to a new set of estimates of the contributions to the likelihood which, in turn, can be used to improve the estimates of class membership, and so on, until the likelihood converges. The algorithm is implemented in a Stata v.9 program that is available from the authors on request.

friends, school and police. More than 40 per cent of the sample reported to have tried cannabis at least once in their life time. As mentioned, in order to reduce the problem of unobserved heterogeneity and compare like-with-like we have confined our analyses to these cannabis users only. For comparison, however, Table 1 displays descriptive statistics for the cannabis users as well as for the sample as a whole.

(Table 1 about here)

There was no age difference between the full sample and the smaller group of cannabis users, the average age was 24 years in both. The proportion of males, however, was higher among the cannabis users (44 per cent versus 38 per cent) and a larger fraction of the cannabis users reported childhood problems with parents, school, friends or police (22 per cent versus 16 per cent stated at least on of these). Problems with parents were reported most frequently in both samples (13 and 9 per cent, respectively) whereas problems with the police were stated by 3 per cent of the cannabis users and 2 per cent of the whole sample.

As expected, the cannabis users reported a higher lifetime prevalence of alcohol and illicit drugs. Practically every cannabis user had used alcohol (99 per cent) compared to 92 per cent in the full sample, and the corresponding numbers for amphetamine and cocaine use were 30 versus 13 per cent and 26 versus 11 per cent, respectively. The heroin prevalence was relatively low in both groups (3 and 1 per cent, respectively). For all drugs, substantially smaller fractions reported frequent use, defined as use on 25 or more occasions. The debut ages did not differ between the groups, and in line with the pattern for initiation found in other studies, the average debut ages suggest that drug consumers in Oslo start to use alcohol prior to cannabis, then proceed to amphetamine, heroin and cocaine.

Figure 1 illustrate the "staircase" in drug use initiation where the highest hazard rate for starting with alcohol peaks at an earlier age than the highest hazard rate for cannabis use and use of amphetamine and cocaine. The hazard rate figure for heroin is left out due to the small sample size (n=24) but compared to the other substances it indicated a less uniform pattern. The hazard rates give the probabilities for various age groups of starting with a drug given that the person has not started up to that age.

(Figure 1 about here)

The "staircase" pattern is also confirmed when we examine the individual ages of initiation for the various drugs. Less than 5 per cent reported a lower debut age for amphetamine than for cannabis, 1 per cent a lower debut age for cocaine and no one claimed to have started with heroin before they tried cannabis for the first time. Further, nearly every hard drug users seem to have used cannabis at some point and only 4 of the 254 amphetamine users, 5 of the 218 cocaine users and 1 of the 24 heroin users claimed no cannabis experience.

It is well known that in general surveys like the one used here, homeless and institutionalized people are under-represented, as are people with various sorts of deviant behavior. Reported income and educational achievements suggest that the sample is better off than the average of young people in Oslo. The relatively high prevalence of illicit drug use in the present sample indicates that many drug users do respond to postal questionnaires.

Recall bias may be a problem, as people are asked to recall the debut age of behaviors that occurred, in some cases, more than a decade ago. One may argue, however, that using an illicit drug for the first time is a unique event and that users will tend to remember it. In line with this, one recent study of response reliability in adolescent substance use progression

suggests that the initiation sequences were reported consistently when checked again three years after the first interview (Andrew Golub et al., 2000).

B. *The price data*

In order to examine the economic aspects of drug use more than 3,500 interviews with drug injectors visiting a needle exchange service in Oslo have been conducted since 1993. Interview sessions were first held on a monthly basis, then quarterly from June 1994 and bi-annual since September 1997. The interviews were anonymous, and it was not possible, therefore, to register the interviewees to help recognize them from one interview session to the next. Some individuals will have been interviewed more than once, but precautions were taken to prevent it from happening within the same interview session. In addition to some background variables, interviewees were asked detailed questions about their level and source of monthly income, levels of drug consumption and the prices they had paid for the different types and quantities of drugs. More details about the sampling procedure and representativeness can be found in Anne Line Bretteville-Jensen and Erik Biørn (2003).

We use annual median prices for the different drugs and the nominal prices are deflated by the consumer price index (CPI 1998=100). The prices are reported for small quantity buys: we use the price of 1 gram of cannabis, ¼ gram of amphetamine and cocaine and 1/24 gram of heroin. The survey does not provide price data for the period 1988-1992. Such data are needed since some of the youngsters in the sample set turned 12 during those years (the starting age for inclusion in the pseudo-panel data set). According to the Oslo police, however, drug prices were very stable, in nominal terms, in that period, so we have used deflated 1993-prices for those years. For cocaine, police records of prices are used throughout the period.

With the exception of cocaine, drug prices were substantially reduced over the study period (Figure 2). Heroin in particular, has become cheaper. In 1988 users were charged more than 400 Norwegian kroner (NOK) for the smallest unit sold at the market whereas the equivalent price in 2002 was less than 100 NKR. The price of amphetamine has also fallen substantially whereas cocaine users paid around the same in 2002 as in 1988. Nominally, the price of one gram of cannabis has been stable throughout the period although the CPI deflated price has fallen.

(Figure 2 about here)

III. Results

In the following tables we first present the results from the univariate and the bivariate hazard models, then a more detailed examination of the gateway variable. Thereafter, we examine more closely the two groups that the model identifies and test the robustness of the model. As our main interest is the possible gateway effect of cannabis, only the results for the hazards of hard drug use are presented (the full set of results are available upon request). Our preferred specification uses the logistic hazard model and relies on exclusion restrictions, for the price variables, as well as the timing of events to identify the gateway effect.

A. *Hazard models*

The univariate hazard model for initiation of hard drugs provides a benchmark for the subsequent analyses. The results from running a separate logistic hazard equation, that does

not take unobserved heterogeneity into account, suggest that cannabis is a statistically significant but small stepping stone to the use of harder drugs (see Table 2). The dummy variable for cannabis initiation in the past four years is statistically significant at the 1-percent level. Childhood problems and the price of heroin also seem to have a significant impact on the uptake of hard drug use and the hazard increases with time (age).

(Table 2 about here)

The results from the bivariate hazard model with shared frailty reveal that the picture changes substantially when unobserved heterogeneity is taken into account. First of all, the latent class model separates the individuals into two quite distinct groups. Within the larger group (Group 1, n=617), cannabis is not an important predictor of later hard drug use and the marginal effect (Table 3) is so small as to make it unimportant. In contrast, cannabis is a statistically significant predictor of later hard drug use within the smaller group (Group 2, n=194). The marginal effects suggest that the influence of cannabis is relatively large (doubling the probability of later hard drug use), but the hazard is still small in absolute terms. Among the other explanatory variables, Table 2 shows that the coefficients for childhood problems, heroin price and time are statistically significant for Group 1 whereas gender, childhood problems, amphetamine and cocaine prices and time are statistically significant for Group 2.

(Table 3 about here)

Table 3 provides an overview of the magnitude of the estimated gateway effects. The first row presents estimates of the hazard rate for the 'untreated' (h^0): evaluated with recent initiation of cannabis (the gateway effect) set to zero and at the means of the other regressors. This

gives a sense of the rate of initiation of hard drug use in the absence of a gateway effect. As expected the estimated probability of initiating use of hard drug without previous cannabis use is very low (0.011) and practically zero for Group 1. All estimates for the hazard rate increase when evaluated with previous use of cannabis set to one, as shown in the second row. The third row presents estimates of the partial effect ($h^1 - h^0$): the difference in hazard rates with and without previous use of cannabis evaluated as the mean of the regressors. This shows how the hazard of hard drug initiation is increased by the gateway effect. In absolute terms previous cannabis use is estimated to have a small impact on the initiation of hard drugs. However, as the last row shows, the gateway effect as a relative impact on the hazard rate (h^1 / h^0) is larger. For Group 2 this is statistically significant. Recent use of cannabis almost doubles the hazard of later hard drug use, but it is still a very rare occurrence. For this reason one should be careful not to equate a statistically significant gateway coefficient with a strong gateway effect. Moreover, the results do not imply that cannabis users are twice as likely to become *habitual* hard drug users since only a small minority of those who try hard drugs end up as addicts.

The difference with respect to the estimated gateway effect makes it interesting to gain more knowledge about the two groups, and the descriptive statistics of individuals with a high probability of being in the first – and largest – group show no extreme values on any of the background variables (see Table 4). The second group is interesting in the sense that it consists of a small subset of what one might call "troubled youths". They do worse on the background variables (childhood problems with police, school, friends and parents) as well as reporting to have started their illicit drug use at an earlier age (e.g. mean debut age for cannabis is 15.6 versus 18.9 in Group 1). Group 2 not only states a much higher life time prevalence of amphetamine, cocaine and heroin use but also a more frequent use of illicit

drugs. More than 80 per cent among the "troubled youths" reported that they had used cannabis on more than 25 occasions and 57 per cent had as frequent use of amphetamine. The corresponding numbers for the larger group of sample are 35 and 29 per cent, respectively.

(Table 4 about here)

An interpretation of the main findings is that for most people, cannabis does not seem to be a statistically significant stepping stone to hard drugs. The use of cannabis does not significantly increase their hazard of taking up drugs like cocaine, amphetamine or heroin. On the other hand, the sample consists of a minority of individuals whose hazards for hard drugs do increase significantly after having used cannabis. The unobserved characteristic – genetic composition, time preferences or upbringing - seem to make the individuals in this group more vulnerable to the influence cannabis on later hard drug use.

IV. Sensitivity analysis

Computation of latent class models is prone to problems due to local optima. To examine the robustness of the results, the analysis was repeated with different starting values (for individual group membership) and the process of convergence was traced. The algorithm produced a likelihood that increases gradually and monotonically towards a maximum. Repeated tests using different starting points also showed that the routine converged to the same solution regardless of the starting point, indicating that the maximum really is a global maximum.

The model also gave robust results when we shortened the incubation period: the length of time in which cannabis could be viewed as causally important (Table 5). Decreasing the original 4 year time window to 2 years and re-running the analysis produced comparable results: two distinct groups, one small group with a statistically significant gateway coefficient and another group in which the effect was smaller and/or statistically insignificant (at the 5-percent level).

(Table 5 about here)

In the empirical analysis presented it is assumed that the unobserved heterogeneity divides the sample into two groups. To test whether this is a reasonable assumption, a model with three groups was also estimated. However, in this model the solutions became unstable as the algorithm gave different answers for different starting values. The same problem was also encountered in some other variations of the model. For instance, eliminating the exclusion restrictions led to non-converge of the likelihood. This reflects the problems of local optima, identification and convergence for over-parameterized models. It could also be interpreted as an argument for using a relatively simple and parsimonious model as the one used in this paper.

V. Conclusion

The commonly observed sequential pattern of drug use initiation may well be explained by an increased risk of starting to consume a more harmful drug after first having used a soft drug but there could also be other factors influencing the uptake of various drugs. As no survey, no

matter how detailed, will include all potentially important variables, testing the influential gateway hypothesis imposes a statistical challenge. This paper has argued that the issue of unobserved individual heterogeneity is central, that the empirical methods should take into account time-varying covariates like prices and that it should make as few restrictions on the model as possible. After developing such a model, based on a bivariate discrete time hazard model with shared frailty, the conclusion from the empirical findings is that the gateway effect of cannabis is statistically significant for a small sub-group of "troubled youths". Within this group the risk of taking up hard drugs almost doubles after the initiation of cannabis. For most youths, however, there does not seem to be a significant or large gateway effect.

The results underline the importance of adopting a general approach in which all the coefficients are allowed to vary. The large differences between some of the coefficients in the two groups indicate this and statistical tests of the hypothesis that the coefficients in the two latent classes are similar are clearly rejected. This may explain some of the discrepancy between our results and Pudney (2003) and van Ours (2003). Pudney, and to some extent van Ours, argue that the gateway effect is greatly reduced after taking unobserved heterogeneity into account. A more general test in which all variables are allowed to vary reaches a more nuanced conclusion in which the gateway effect actually increases for almost a quarter of the sample when comparing their results to that of the full sample.

As previously listed, there are at least four possible mechanisms underpinning an observed gateway effect. A better understanding of these mechanisms is needed but beyond the scope of this paper. It is important to notice, however, that the finding of a gateway effect in itself not necessarily suggest a ban to any liberalization of cannabis laws. If the driving force behind cannabis users' initiation of hard drugs is that they come in contact with hard drug users whom they not otherwise would have met, a separation of the markets for soft and

hard drug, for instance in line with the Dutch model, could be an option. Further, if people, after experiencing no obvious ill effects of soft drug use, have reduced confidence also in the strong negative publicity directed against hard drug use, the solution may be to make more distinct the differences between the various drugs, perhaps including more distinct differences in information material and campaigns directed against illicit drug use. If, on the other hand, it is the addiction or the adjustment cost effects that operate a liberalization of penal sanctions may not be wanted, even though the number of additional hard drug users resulting from the policy change may be quite modest.

Whatever mechanism or combinations of mechanisms that operates it seems clear that one group of cannabis users are more vulnerable to the effect cannabis use has on subsequent hard drug use. We saw that not only did they start to consume illicit drugs at a younger age, a substantially larger proportion also used drugs like amphetamine and heroin more intensively, i.e. more people in this group reported to have used these drugs on more than 25 occasions. They also reported more childhood problems with police, parents, friends and school. Whether the underlying reasons are childhood traumas, negative peer influence, high time preferences, less beneficial genetic endowments or other factors, the "troubled youths" seem to have less resistance with respect to further drug involvement. Thus, some policy implications of the findings may still be suggested: Early identification of the "troubled youths" and adequate help to minimize the effect of their risk factors can be effective in reducing the number of hard drug users.

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TABLE 1 – VARIABLE DEFINITIONS AND DESCRIPTIVE STATISTICS

(Full sample, n=1,984; Cannabis users only, n= 811)

Variable Label	*Variable Definition*	*Full sample*		*Cannabis users*	
		Mean	Std.dev.	Mean	Std.dev.
Age	Age in years	24.1	1.64	24.0	1.66
Gender	Dummy; 1 if male	0.38	0.49	0.44	0.50
Parents	Dummy; 1 if problems with parents	0.09	0.29	0.13	0.34
School	Dummy; 1 if problems at school	0.07	0.26	0.10	0.30
Friends	Dummy; 1 if problems with friends	0.04	0.20	0.05	0.21
Police	Dummy; 1 if problems with police	0.02	0.13	0.03	0.16
Childhood Problems	Dummy; 1 if any problems	0.16	0.37	0.22	0.41
Alcohol	Dummy; 1 if ever used alcohol	0.92	0.27	0.99	0.09
Cannabis	Dummy; 1 if ever used cannabis	0.41	0.49	1	1
Amphetamine	Dummy; 1 if ever used amphetamine	0.13	0.33	0.30	0.46
Cocaine	Dummy; 1 if ever used cocaine	0.11	0.31	0.26	0.44
Heroin	Dummy; 1 if ever used heroin	0.01	0.11	0.03	0.16
Hard drugs	Dummy; 1 if ever used hard drugs	0.16	0.36	0.36	0.48
Alco-age	Debut age for first use of alcohol	15.3	2.25	14.5	1.96
Cann-age	Debut age for first use of cannabis	18.0	2.76	18.1	2.73
Amph-age	Debut age for first use of amphetamine	18.8	2.39	18.8	2.30
Hero-age	Debut age for first use of heroin	19.5	2.57	19.6	2.67
Coca-age	Debut age for first use of cocaine	20.0	2.32	19.9	2.32

TABLE 2 - COEFFICIENT ESTIMATES FOR HAZARD MODELS FOR HARD DRUG USE

	Single equation model	Latent class models (taking unobserved heterogeneity into account)	
		Group 1 (n=617))	*Group 2 (n=194)*
Male	0.109	0.115	0.569***
	(0.122)	(0.161)	(0.161)
Childhood problems	0.448***	0.490***	0.627**
	(0.080)	(0.112)	(0.115)
Price of amphetamine	-0.002	0.000	-0.022***
	(0.003)	(0.003)	(0.004)
Price of cocaine	0.000	0.000	0.001**
	(0.000)	(0.000)	(0.000)
Price of heroin	-0.003***	-0.014***	0.002
	(0.001)	(0.003)	(0.002)
T (time)	1.876***	3.435***	2.182***
	(0.463)	(1.126)	(0.494)
t2	-0.181***	-0.285**	-0.201***
	(0.057)	(0.12)	(0.061)
t3	0.005**	0.007*	0.004*
	(0.002)	(0.004)	(0.002)
Previous use of cannabis	0.742***	0.294*	0.710***
	(0.128)	(0.163)	(0.185)
Constant	-8.310***	0.115***	-5.029***
	(1.303)	(0.161)	(1.449)

* Significant at 10-percent level

** Significant at 5-percent level

*** Significant at 1-percent level

TABLE 3 - ESTIMATES OF GATEWAY EFFECTS FOR HAZARD MODELS FOR HARD DRUG USE

	Single equation model	Latent class models (taking unobserved heterogeneity into account)	
		Group 1 *(n=617)*	*Group 2* *(n=194)*
Hazard without previous cannabis use: h^0	0.011	0.00067	0.026
Hazard with previous cannabis use: h^1	0.027	0.00091	0.051
Gateway effect on hazard rate: $h^1 - h^0$	0.016	0.00023	0.025**
Relative gateway effect: h^1 / h^0	2.542	1.342	1.981

* Significant at 10-percent level

** Significant at 5-percent level

*** Significant at 1-percent level

TABLE 4 – DIFFERENCES BETWEEN THE CLUSTERS IN THE LATENT CLASS ANALYSIS

	Group 1, n = 617 (Most youths),		Group 2, n = 194 ("Troubled youths"),	
Variable	*Mean*	*St. dev.*	*Mean*	*St. dev*
Gender (percentage male)	44.2	0.497	42.3	0.495
Percentage with reported use of …				
… amphetamine (0/1)	13.0	0.336	85.1	0.357
… heroin (0/1)	0.6	0.080	8.8	0.283
… cocaine (0/1)	11.0	0.313	72.7	0.447
Mean age of starting to use …				
…cannabis	18.9	2.518	15.6	1.485
…amphetamine	20.6	1.887	18.1	2.044
…heroin	21.0	1.547	19.3	2.845
Frequency of use (percentage reported to have used the drug more than 25 times) …				
…cannabis	35.2	0.478	82.5	0.381
…amphetamine	28.8	0.455	57.0	0.497
…heroin	0	0	41.2	0.507
Percentage with serious childhood problems …				
… with the police	1.3	0.113	7.2	0.259
… in school	8.9	0.285	13.9	0.347
… with friends	4.5	0.208	5.2	0.222
… with parents	13.0	0.336	14.9	0.357

TABLE 5 – ROBUSTNESS OF GATEWAY COEFFICIENT TO ASSUMPTIONS ABOUT THE INCUBATION PERIOD

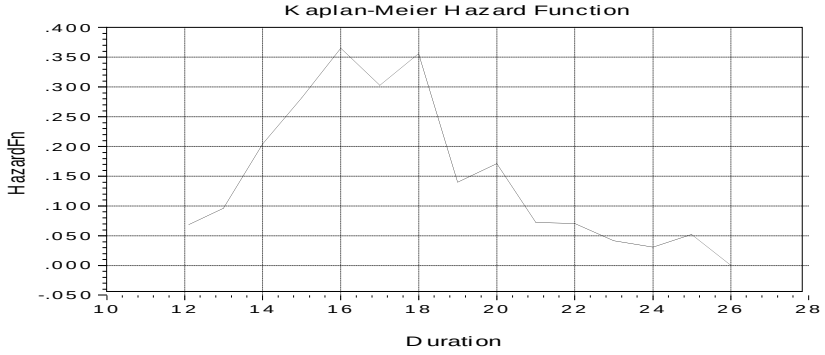
Cannabis is assumed to have an effect on the probability of starting to use hard drugs within …	*Overall gateway coefficient*	*Gateway coefficient, (Most youths)*	*Gateway coefficient ("Troubled youths")*	*Probability of belonging to the "Troubled youths" group*
2 years	0.742***	0.351*	0.684***	0.20
4 years	0.949***	0.295*	0.710***	0.24

* Significant at 10-percent level

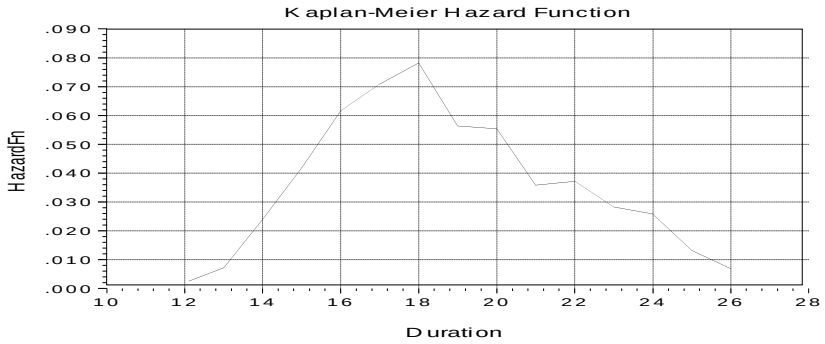
*** Significant at 1-percent level

FIGURE 1 - HAZARD RATES FOR THE ONSET OF ALCOHOL, CANNABIS AMPHETAMINE AND COCAINE USE

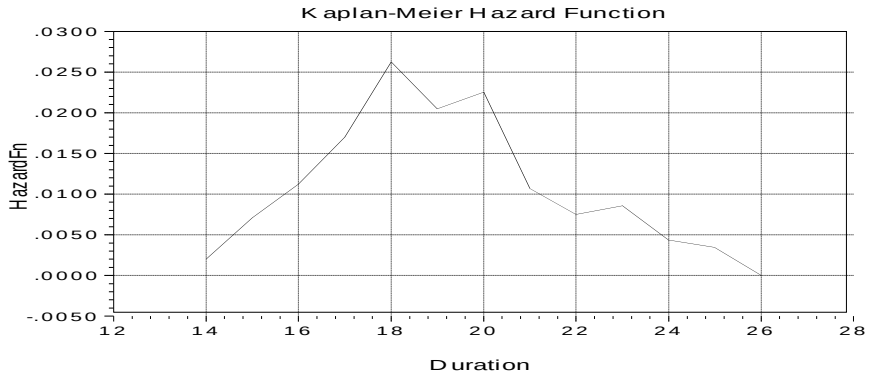
Alcohol:



Cannabis:



Amphetamine:



Cocaine:

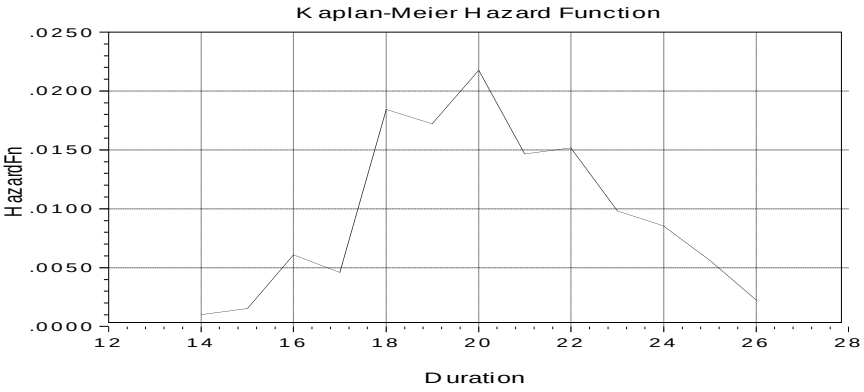
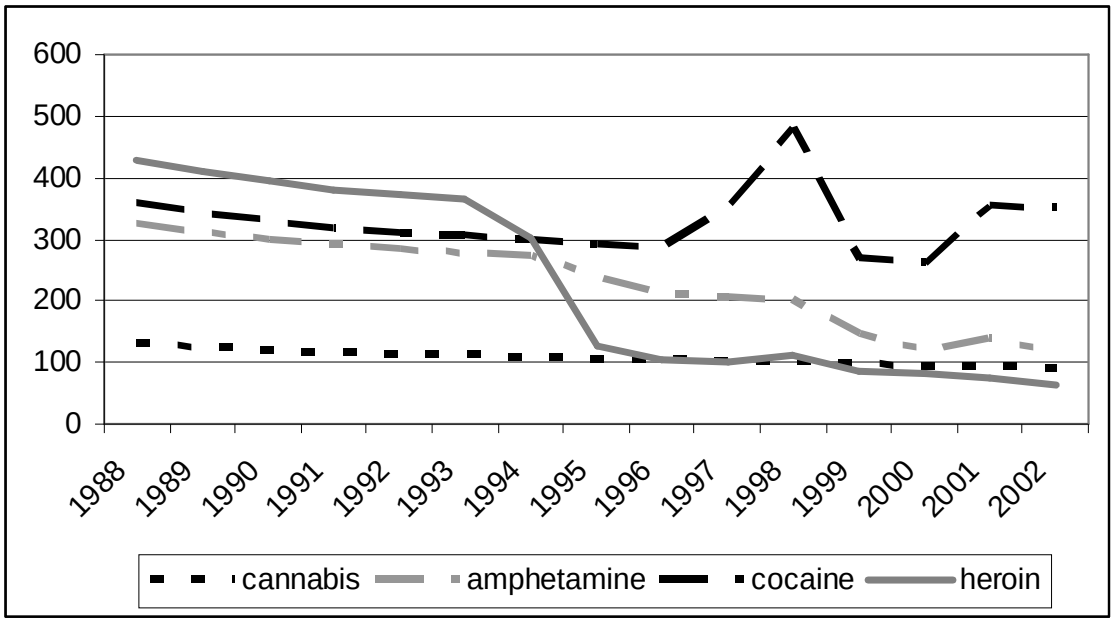

Kaplan-Meier Hazard Function
HazardFn
.0250
.0200
.0150
.0100
.0050
.0000
12
14
16
18
20
22
24
26
28
Duration

FIGURE 2 – PRICES OF CANNABIS, AMPHETAMINE, COCAINE AND HEROIN, 1998-2002*



*Note: The prices relates to the following quantities of the drugs: 1 gram of cannabis, ¼ gram of amphetamine, ¼ gram of cocaine and 1/24 gram of heroin. The prices have been deflated by the CPI (1998=100).