Climbing the Drug Staircase: An Analysis of Accessibility, Proneness and Previous Cannabis Use on the Initiation of Hard Drug Use

Anne Line Bretteville-Jensen
Norwegian Institute for Alcohol and Drug Research (SIRUS)
email: alb@sirus.no

Liana Jacobi
Department of Economics
University of Melbourne
email: ljacobi@unimelb.edu.au

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Abstract

This paper explores the role of previous cannabis use, accessibility and proneness on a person's hard drug uptake. Existing empirical drug research and policy discussions have focused on a constant gateway effect of previous cannabis use, that is hypothesized to contribute to the initiation pattern commonly found in empirical studies (cannabis use precedes hard drug use) due to a causal linkage between cannabis and subsequent hard drug use. Despite mixed evidence and a very limited understanding of possible transmission mechanisms, the gateway hypothesis has been influential in formulating a strict drug policy in many western countries. Proneness and accessibility provide possible alternative/additional explanations for the observed staircase pattern of drug use to the gateway effect of previous cannabis use and offer potentially different policy implications. We use unique survey data from a representative sample of young adults in Norway (collected in 2006) with individual level information on both accessibility and proneness, to explore the role of previous cannabis use, accessibility and proneness on the initiation of hard drugs like cocaine, amphetamine, ecstasy and heroin. Motivated by possible gateway transmission channels proposed in the literature, we propose a novel modeling approach in the spirit of an endogenous switching regression model, which allows for a more complex effect of previous cannabis use on hard drug uptake. Our model is formulated within the Bayesian paradigm. We formulate suitable prior distributions based on data from a related 2002 survey. We employ the Bayesian predictive approach to investigate the relative importance of the three effects and to explore potential policy implications of our findings. We find evidence for the effects of accessibility, proneness and previous cannabis us, as well as for a heterogeneous gateway effect.

Key words: Accessibility, Bayesian Analysis, Bayesian Predictive Analysis, Cannabis Gateway, Cannabis Use, Hard Drug use, Markov Chain Monte Carlo, Policy, Proneness.

1 Introduction

Cannabis is the most widely-used illicit drug in Europe, Australia and North America and an increasing number of people are seeking treatment for cannabis use (EMCDDA 2007; UN 2007). In many EU countries prevalence figures are higher now than ten years ago. An interesting and important question is whether the widespread cannabis use will also lead to an increased number of hard drug users. The question emerges because one of the most robust findings in the epidemiology of illicit drug use is the initiation pattern, where most illicit drug users seem to have started with legal drugs like cigarettes and alcohol before proceeding to cannabis and subsequently, if further involvement, to hard drugs (Kandel 2002). Very few hard drug users go straight to heroin without previously having used several other drugs, and cannabis is often the first of the illegal drugs being used. Kandel (1975), employing a US sample, was the first to point out this "staircase" but a similar pattern has been retrieved in many subsequent studies of drug initiation (Kandel 2002).

The observed staircase pattern of illegal drug initiation has led to the so called gateway hypothesis which states that the use of a cannabis increases the risk of using a hard drug later on, i.e. it claims that there is a causal relationship between the various drugs. The existing empirical work has focused on examining whether there is such an effect. Many studies report to have found a positive "gateway" coefficient on previous cannabis use (e.g. DeSimone 1998, van Ours 2003, Fergusson et al 2006, Melberg et al 2007 and Bretteville-Jensen et al 2008) whereas Pudney (2003) and Beenstock and Rahav (2002) found weak or no support for the hypothesis. Despite the mixed and limited evidence for a gateway effect, the hypothesis has been influential in formulating a strict drug policy in many western countries and opponents of decriminalizing cannabis often refer to the assumed causal relationship in their argumentation.

There are, however, possible alternative or additional explanations for the observed higher probability of hard drug use among cannabis users that do no claim a causal relationship between the two and may lead to different policy recommendations. First, a common proneness against deviant behavior may lead some subjects to consume illicit drugs, and cannabis is just used prior to others because it is cheaper and more readily available. Excessive drug use is then viewed as

one out of many potential responses to unfavorable genetic endowments or traumatic childhood experiences. If people with a certain vulnerability have an increased risk for drug use, there may be no causal link between cannabis and hard drugs that explain the familiar initiation pattern (Morral et al. 2002). Second, accessibility (economic, cultural and/or physical) to the different illicit drugs will vary and may explain the observed initiation pattern. Accessibility may to a large extent also vary between youngsters, as some people have better knowledge of how and where to obtain drugs, what prices to pay etc. and drug use is more accepted in some youth cultures than in others.

In this paper we investigate the role accessibility and proneness factors and previous cannabis use (gateway) in explaining a subject's probability of hard drug uptake and explore potential policy implications of our findings. We employ a novel modeling approach to explore the potentially complex nature of the effect of previous cannabis use on hard drug uptake. As pointed out in recent literature (e.g. Fergusson et al. 2006, Kandel et al 2006), the mechanisms which could drive the effect of previous cannabis use on hard drug use are not well understood and require empirical investigations. A number of possible transmission mechanisms channels have been discussed in the recent literature (see e.g. Pudney 2003, Melberg et al. 2007): 1) the use of cannabis may create a psychological or physiological need for stronger narcotic experiences; 2) personal experiences with cannabis may reduce the credibility in warnings against hard drugs; 3) having used one illegal drug may lower the threshold for trying also another drug; 4) obtaining and using cannabis may induce contact with hard drugs users and dealers whom they would not otherwise have met. However, existing work has focused on testing for a constant gateway effect that has been modeled in terms of a control variable for previous cannabis use. In some cases a control for intensity of previous use or the effect of early initiation have been tested. As the effectiveness of a policy will depend not only on the role of accessibility and proneness factors versus the gateway effect, but also on which mechanism(s) drives the latter, a better understanding of the mechanisms would be very valuable for policy purposes.

Our analysis exploits a unique survey of young Norwegians that in 2006 collected detailed data on drug use and initiation patterns, and which also provides a number of subject-based

variables on drug accessibility and proneness. Despite this relatively rich data set, there may be unobserved factors, such as genetic factors, that can influence both the initiation of cannabis and hard drugs. We therefore base our empirical analysis of the three effects on a joint model for cannabis use and hard drug uptake. To account for the likely complex nature of the gateway effect, hard drug uptake is modeled as partial (endogenous) switching regression model where we estimate a hard drug model under each previous cannabis use state. Motivated by the proposed transmission mechanisms, the model allows for a direct constant effect of previous cannabis use and also for an effect via accessibility to hard drugs (hereafter termed "the extended gateway effect").

Our model is formulated within the Bayesian paradigm. We exploit information from a similar (but more restrictive) 2002 survey of young Norwegians to formulate our prior assumptions about the model parameters. The estimation of the model is based on Markov Chain Monte Carlo simulation methods, thus avoiding a solution via the optimization of the likelihood which can be challenging in such type of models. Further, we employ the Bayesian predictive approach to analyze the relative importance of the three effects on the probability hard drug uptake. We estimate the marginal predictive probabilities of hard drug uptake under no and previous cannabis intake to quantify the effect of previous cannabis use and compute, for example, average marginal effects of accessibility, proneness and previous cannabis. We also use the predictive approach to investigate various policy relevant scenarios. One advantage of the Bayesian predictive approach is that the predicted probabilities are based on the posterior distribution of the parameters rather than point estimates. Given the limited availability of strictly exogenous control variables in this type of problems a number of relevant coefficients are often estimated with a low precision. In the predictive analysis under the Bayesian paradigm this imprecision is accounted for directly trough the posterior distribution. Suitable Markov Chain Monte Carlo methods for the model fitting and predictive analysis are developed in the paper, building on work by e.g. Chib (2007) and Chib and Jacobi (2007).

Overall, a robust finding that emerges from our analysis is that all three effects contribute to the observed higher hard drug use pattern among cannabis users, of which previous cannabis

use being the most influential one. We have also found an interesting link between previous cannabis use and the effect of accessibility factors, which helps us to improve our understanding of the gateway mechanisms.

2 Data

In 2002 and 2006 the Norwegian institute for alcohol and drug research (SIRUS) sent postal questionnaires to a representative sample of young adults to collect data about the respondents' legal and illegal drug use. In this paper we employ the most recent of these surveys which includes data on ethnicity and for which we can construct price variables. The rich data set provides information on the frequency of current and life time drug use, as well as on the starting ages for the various drugs. In addition to personal characteristics, several variables related to proneness and accessibility are available for each respondent. The survey was aimed at the population of the 21 to 30 year old. This age group is particularly well suited for the analysis of illicit drug use initiation, as these and other data show that most recreational drug users have started drug use by the age of 21 and that frequency and extent of drug use is highest among young adults.

The response rate of the 2006 mail survey was somewhat less than 50 per cent, which is roughly in line with other studies of this type (see e.g. Reinarman et al. 2004). Our net sample consists of 4164 observations. It is well known that various sub-groups (institutionalized people, intravenous/heavy drug users etc.) are likely to be under-represented in net samples from national surveys as these groups are harder to reach and may be less willing to fill out a questionnaire. Our findings may thus be more relevant for the group of recreational users of illicit and licit drugs than for the group of problematic drug users. Figure 1 shows the initiation pattern of drug use in terms of mean starting ages for alcohol and the main illegal drugs (cannabis, amphetamine, heroin, ecstasy and cocaine, respectively) in our sample.

The graph reflects the commonly reported initiation pattern for drug use with cannabis being the illegal drug with the lowest starting age. The mean age of starting with alcohol is 15.4 which is much lower than the mean starting ages for the illegal drugs: 18.1 for cannabis; 19.2

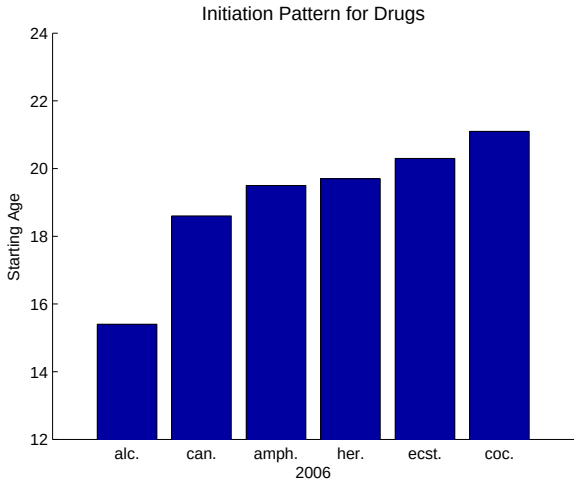


Figure 1: *Starting ages for various legal and illegal drugs in the 2006 SIRUS survey.*

for amphetamine; 19.3 for heroin; 19.4 for ecstasy and 20.5 for cocaine. Since we primarily are interested in the effect of cannabis on subsequent use of any hard drug, and in line with Melberg et al (2007), we have chosen to merge the heroin, amphetamine, ecstasy and cocaine variables to one hard drug variable.

Good and reliable data on illicit drug use is difficult to obtain. General population surveys, like the present, based on self-reported information may e.g. suffer from false negatives (people claiming not to have used illegal substances or reporting a lower consumption level or frequency than the real one) and/or false positives (people exaggerating their actual drug use). For phenomena with low frequency, like heroin use, the false positives is considered a bigger problem than the false negatives (Skog 1992). We have no means to test for false negatives. However, the reported prevalence of the non-existing drug "relevin" gives an indication of false positives in the present sample. "Relevin" was listed as just another drug the respondents were asked about and with only 9 people (0.2 per cent) reporting to have ever used this particular drug, false positives does not seem to be a pervasive problem in this sample. Since our data is based on a younger cohort it is reasonable to assume that recall bias is less of a problem compared to studies including people in their 40-60s. For most subjects in these older cohorts drug initiation would have occurred two or more decades before the time of the survey.

The sampling procedure employed by SIRUS implies an over-sampling of people from the

capital city, Oslo. This increases the overall prevalence of drug use as young people in Oslo use more drugs than do people from comparable age groups outside the capital but should not otherwise affect the sample. The prevalence of cannabis use in the 2006 data is 38.2%. However, prevalence rates adjusted for the over-sampling are in line with corresponding rates found in other Norwegian surveys for the same age group. In general, illegal drug use in Norway seems to follow the pattern found elsewhere in Europe. According to national surveys of 15-34 year olds published by the European Monitoring Centre for Drug use and Dependence (EMCDDA) the life time cannabis use in Norway (25.5%) is lower than in e.g. Denmark (49.5%), U.K (42.3%), France (43.6%)and the Netherlands (32.3%), but higher than e.g. in Sweden (19.1%) and Finland (22.0%) (EMCDDA 2007). According to a recent report from the United Nations, cannabis is even more prevalent in Oceania and North America than in Europe (UN 2007).

Drug users were asked to report their frequency of illicit drug use (the categories were 0, 1-4, 5-10, 11-25. 26-50, 50+). Life time prevalence of cannabis use in the current sample is 38.2 per cent. Many subjects report to have tried cannabis less than five times. It is questionable, however, whether using cannabis once or a few times only is sufficient to cause an effect on hard drug use. Here we assume that for cannabis to have an effect on further drug involvement a life time frequency of 5 or more times is required, i.e. "accidental" cannabis users will in the following analyzes be categorized together with non-users of the drug. We will however relax this assumption in our sensitivity analysis. In the case of hard drug use, we set the dependent binary hard drug variable equal to one if any of the hard drugs has been use at least once (including subjects from 1-4 category) since using a hard drug even once, still increases the risk of adverse health effects (heroin overdosing etc.). Given these definitions, the cannabis prevalence in the sample is 23.4% and the hard drug prevalence is 13.7%. Among the 973 cannabis users, 48% (n=469) have also used hard drugs, while 102 out of 571 hard drug users (18%) do not report any previous cannabis use (see Table 1).

Table 1 also presents a summary of the sample means for relevant demographic, proneness and accessibility related variables by three different drug use states: no drug use; cannabis use only; hard drug use. The table shows that the proportion of males increases with drug

		No Drug Use (n=3,089)	Cannabis (only) (n=504)	Hard Drugs (n=571)
Drug Use	Cannabis	0	1	0.82
	Hard Drug	0	0	1
Demographics	Male	0.36	0.43	0.52
	Age (21-24)	0.29	0.29	0.27
Proneness	Police	0.007	0.004	0.06
	Parents	0.06	0.10	0.20
	School	0.04	0.07	0.13
	Friends	0.03	0.04	0.06
	Leave School Early	0.04	0.04	0.12
	Alcohol young	0.04	0.10	0.16
	Smoker	0.45	0.86	0.84
Accessibility	Country	0.42	0.24	0.27
	Non-western	0.06	0.02	0.04
	Obtain cannabis	0.54	0.55	0.55
	Obtain hard drug	0.42	0.43	0.43
	Seizure	294	366	374
	Cannabis prev	0.23	0.25	0.25
	Hard Drug prev	0.08	0.09	0.09
	Oslo	0.50	0.66	0.67
	Cannabis price	101	100	101
	Amphetamine price	542	539	545

Table 1: *Summary of key variables of 2006 sample of the SIRUS data by drug intake.*

involvement whereas the percentage of respondents in the age group 21-24 ("age") stays roughly the same across the categories. The accessibility and proneness variables are discussed in more detail in the next section.

3 Proneness and Accessibility in the Data

The data set provides several variables that can be taken as proxies for proneness and accessibility of drugs. The respondents were asked whether they had serious problems with police, parents, school or friends during their childhood, respectively. In line with the proneness hypothesis, Table 1 shows that these indicators have higher means among hard drug users than among the other two groups. Very few in the non-user and cannabis-only categories reported any problem with police (less than 1 per cent), compared to 6 per cent among the hard drug users. Problems with parents and school show an increasing trend when moving from non-users to hard drug users, with the latter group reporting three times the prevalence of non-users. Also, more hard drug users report problem with friends during childhood (6%) than did cannabis-only and non-

users of drugs. The large majority of young adults (86%), however, did not claim any of the listed childhood problems.

Table 1 further indicates that the group of illicit drug users has a much higher proportion of respondents that started with alcohol at a very young age and who dropped out of school after only 10 years. Starting with alcohol before turning 13 or leaving school at an early age (15/16 years) could be indications of deviant personality or reflect adverse personal experiences or it could suggest a relatively high time preference rate, which according to "the theory of rational addiction" increases the probability of problematic drug use (Becker and Murphy 1988). Finally, given the well-known health risks associated with cigarette smoking we will include a dummy for smoking (set equal to 1 if ever smoked with some regularity) and use this as an additional proneness proxy.

According to microeconomic theory we also expect the subject's access (cultural, economic and/or physical) to drugs to be of importance for drug uptake. The survey provides information on various aspects of accessibility to cannabis and hard drugs. For example, we assume that the drug use culture among non-western immigrants differ from that of native Norwegians, and Table 1 shows that the highest proportion of these immigrants is found among the non-users. Further, the data confirms that respondents living in cities have easier access to drugs compared to subjects living in more remote rural areas of Norway - both physical and cultural: A larger proportion of respondents living in cities claim to be able to obtain illegal drugs within 3 days (72 vs 57%) and have been offered drugs (78 vs 59%). The obtain variable also differs substantially by cannabis intake, see Appendix B. Employing these variables directly, however, may cause severe endogeneity issues, so instead we use the obtain information to create location specific obtain variables for cannabis and hard drugs. The obtain variables in Table 1 are defined as the percentage of *non-users* that can obtain cannabis or hard drugs within 3 days in each of the 19 counties in Norway. As an additional indicator of cultural accessibility, we have included variables for the mean county prevalence of cannabis and hard drug. Even though the "prevalence" and the "obtain" variables do not show any great variation across the groups in Table 1, they seem to influence the cannabis and hard drug uptake (see Table 2).

Accessibility related data from two supplementary data sources have also been included. The "seizure" variable is based on the mean number of amphetamine and heroin seizures by customs and police for each county. In our analysis we use this variable as a proxy for the geographical variation in hard drug supply. The highest number of seizures per 100,000 inhabitants is found among the group of hard drug users. Finally, drug prices are potentially important in explaining drug uptake (economic accessibility). Given the illegality of drug markets, reliable and detailed price data are rare. We are fortunate to have data on cannabis and amphetamine prices that were collected through personal interviews with drug addicts. Since 1993 more than 4000 interviews have been conducted with people attending the main needle exchange service in Oslo (see Bretteville-Jensen and Biorn (2004) for more details of this data collection). The prices for hard drugs have shown a falling trend throughout the period and the amphetamine price series is used as a proxy for all hard drug prices. Since we have cross-sectional data, every person is assigned the drug price that prevailed when he/she statistically had the highest risk of taking up the different drugs (drug price and seizure variables are divided by 1000 in the estimations). The police confirm that drug prices outside the capital are higher than in Oslo, so a dummy for Oslo is included among the set of covariates. However, if there is another cannabis or hard drug culture in Oslo compared to other parts of Norway, this too will be picked up the Oslo dummy.

4 Empirical Analysis

4.1 Modeling Cannabis and Hard Drug Intake

In this section we formulate a model to investigate the effect of prior cannabis use, accessibility and proneness factors on hard drug initiation. As mentioned before we cannot rule out the presence of unobserved proneness factors (such as genetic endowments) that affect both a subject's likelihood to use cannabis and later hard drugs. We therefore proceed with our analysis under a joint model for cannabis use and subsequent hard drug uptake. To account for the sequential initiation pattern of the drug use and the role of cannabis as a gateway drug, $c_i = 0, 1$ indicates a subject's cannabis use prior to hard drug use $h_i = 0, 1$. To allow for an extended gateway effect, we model hard drug intake as a flexible function of previous cannabis intake in the spirit

of an endogenous switching regression model so that $h_i = h_{0i} + (h_{1i} - h_{0i})c_i$, where h_{0i} and h_{1i} refer to hard drug uptake without and with previous cannabis use, respectively.

We start by proposing a probit model for hard drug intake of ith subject, $h^*_{j,i}$ as a function of previous cannabis use h_{ji}, where $j = 0, 1$ indicates the subject's the previous cannabis use $c_i = j$. Our specification extends the typical hard drug model used in the literature by explicitly modeling the effects of proneness and accessibility, as well as by modeling the effect of previous cannabis use in a more general way through a switching regression type formulation that is motivated by the hypothesized transmission channels. As previous papers we include an indicator for previous cannabis use to control for any psychological and physiological need for harder drug that arises from cannabis use together with controls for accessibility, proneness and demographic factors. The indicator would also pick up the increased accessibility to hard drugs for cannabis users implied by transmission channel 4. If cannabis use lowers the threshold for hard drug use and makes them seem less harmful (channels 2 and 3), then the same degree of accessibility should lead to a higher hard drug uptake for cannabis users than non-users. We capture these two channels by modeling the effect of the accessibility factors as a function of previous cannabis use.

To formulate our probit model for hard drug uptake in the more convenient common latent variable formulation (which will also be exploited in the fitting of the model), we define the latent continuous hard drug use variable $h^*_{j,i}$ that is related to the observed binary hard drug use in the usual way through the identity function as $h_{ji} = I[h^*_{ji} > 0]$. We specify the latent hard drug uptake in terms of a vector of hard drug specific cultural and physical accessibility variables $\mathbf{a}_{hi}$, hard drug price (economic accessibility) p_{hi}, and a vector of proneness related variables $\mathbf{pr}_{hi}$ as:

$$h^*_{j,i} = \mathbf{w}'_{1,hi}\boldsymbol{\beta}_j + \mathbf{w}'_{2,hi}\boldsymbol{\alpha} + \varepsilon_{j,i}, \quad \varepsilon_{ji} \sim N(0, 1) \tag{4.1}$$

where $\mathbf{w}_{1,hi} = [\mathbf{a}_{hi}, p_{hi}]$ and $\mathbf{w}_{2,hi} = [const, g_i, \mathbf{pr}_{hi}, \mathbf{d}_{hi}]$. In the latter expression g_i indicates a subject's prior (gateway) use of cannabis and $\mathbf{d}_{hi}$ is a vector of demographic variables. The coefficient on g_i captures the constant effect previous cannabis use on hard drug uptake. The model also captures what we call an extended gateway effect through the coefficients on the

accessibility variables through the cannabis state dependent parameter vector $\boldsymbol{\beta}_j$. To benchmark our coefficient estimates on the gateway variable, we also consider a more traditional specification which only considers the direct gateway effect of cannabis and where $\boldsymbol{\beta}_0 = \boldsymbol{\beta}_1$.

Given our switching regression formulation of the hard drug model, the error term $(\varepsilon_{j,i})$ is specified as a function of previous cannabis use. This allows for the possibility that cannabis users and non-users face different unobserved random shocks that are relevant for hard drug intake and/or react differently to the unobserved shocks. The raw data shows that cannabis users and non-users differ significantly in terms of their observed characteristics and thus, very likely also in terms of the random shocks and unobserved factors that affect hard drug initiation. For example, we cannot rule out that cannabis users differ in terms of their unobserved proneness characteristics or socioeconomic background from the non-cannabis users in the population. These factors may affect cannabis and hard drug uptake, but not necessarily in the same way, which can be captured by different values for ρ_0 and ρ_1.

To specify a joint model, we formulate a probit model for cannabis intake in terms of the latent continuous cannabis use (c_i^*), where $c_i = I[c_i^* > 0]$. Similar to the hard drug case, we model cannabis use as a function of demographic and relevant accessibility $(\mathbf{a}_{ci}, p_{ci})$ and proneness variables $\mathbf{pr}_{ci}$. By modeling cannabis intake in such a way we also provide a benchmark for the interpretation of the estimates from the hard drug model and a more differentiated picture of the hard drug versus the cannabis market. In particular, we assume the following model for the latent cannabis intake:

$$c_i^* = \mathbf{w}_{c,i}'\boldsymbol{\gamma} + u_i, \quad u_i \sim \mathcal{N}(0,1) \tag{4.2}$$

where $\mathbf{w}_{ci} = [const, \mathbf{a}_{ci}, p_{ci}, \mathbf{pr}_{ci}, \mathbf{d}_{ci}]$.

To complete the joint model specification, we assume that (ε_{ji}, u_i) are jointly normally distributed with mean zero and correlation matrix

$$\boldsymbol{\Omega}_j = \begin{pmatrix} 1 & \rho_j \\ \rho_j & 1 \end{pmatrix}. \tag{4.3}$$

The correlation parameter ρ_j will correct for, among others, unobserved accessibility and proneness factors, such as genetic factors, that are not captured by the limited accessibility and proneness controls that are included in the covariate vectors. The above assumptions imply

that the joint model for the latent hard drug and cannabis use for cannabis users is the bivariate Normal distribution $\mathcal{N}_2(h^*_{ji}, c^*_i|\mathbf{W}'_{ji}\boldsymbol{\delta}, \boldsymbol{\Omega}_j)$, where

$$\mathbf{W}_{ji} = \begin{pmatrix} \mathbf{w}'_{1,hi} \times (1-j) & \mathbf{w}'_{1,hi} \times (j) & g_i \sim \mathbf{w}'_{2,hi} & 0 \\ 0 & 0 & 0 & \mathbf{w}'_{ci} \end{pmatrix}.$$

and $\boldsymbol{\delta} = (\boldsymbol{\beta}_0, \boldsymbol{\beta}_1, \boldsymbol{\alpha}, \boldsymbol{\gamma})$. For our empirical analysis we specify the variable vectors in the cannabis and hard drug equations in terms of the corresponding accessibility and proneness and demographic variables as

$$\mathbf{w}_{1,hi} = [\mathbf{a}_{hi}, p_{hi}],\ \mathbf{w}_{2,hi} = [const, c_i, \mathbf{pr}_i, \mathbf{d}_i],\ \mathbf{w}_{ci} = [const, \mathbf{a}_{ci}, p_{ci}, \mathbf{pr}_i, \mathbf{d}_i]$$

where

$$\mathbf{a}_{hi} = \text{[country,oslo,obtain hard,seizure,hard prevalence,non-western]}$$

$$\mathbf{a}_{ci} = \text{[country,oslo,obtain can,can prevalence,non-western]}$$

$$\mathbf{pr}_i = \text{[police,parents,school,friends,leave school,alc young,smoker]}$$

$$\mathbf{d}_i = \text{[male,age2124]},\ p_{hi} = \text{[amphetamine price]},\ \ p_{ci} = \text{[cannabis price]}$$

including the same proneness and demographic variables in the cannabis model and the hard drug model.

4.2 Prior Posterior Analysis

In the Bayesian estimation framework all information about the vector of model parameters, $\boldsymbol{\theta} = (\boldsymbol{\beta}_0, \boldsymbol{\beta}_1, \boldsymbol{\alpha}, \boldsymbol{\gamma}, \rho_0, \rho_1)$, is summarized in the posterior distribution of the model parameters conditional on the observed data $\pi(\boldsymbol{\theta}|\mathbf{h}, \mathbf{s}, \mathbf{W})$. The posterior distribution combines the information from the data, via the likelihood, and prior knowledge about the parameters, via the prior distribution. By Bayes theorem, the posterior distribution is proportional to the product of the prior distribution $\pi(\boldsymbol{\theta})$ and the likelihood function $p(\mathbf{h}, \mathbf{c}|\mathbf{W}, \boldsymbol{\theta}) = \prod_{i=1}^{n} \mathsf{Pr}(h_i = l, c_i = j|\mathbf{W}_i, \boldsymbol{\theta})$. From the modeling assumptions in the previous section it follows directly that the likelihood contribution $\mathsf{Pr}(h_i = l, c_i = j|\mathbf{W}_i, \boldsymbol{\theta})$ of the ith subject can be written in terms of the bivariate normal distributions of the latent drug variables (h^*_i, c^*_i) as

$$\mathsf{Pr}(h_i = l, c_i = j|\mathbf{W}_i, \boldsymbol{\theta}) = \int_{A_l} \int_{A_j} \mathcal{N}_2(h^*_i, c^*_i|\mathbf{W}'_{ji}\boldsymbol{\delta}, \boldsymbol{\Omega}_j)\, ds^*_i\, dh^*_i$$

where $l = 0, 1$, and the integration regions are $\{-\infty, 0\}$ for $l, j = 0$ and $\{0, +\infty\}$ for $l, j = 1$.

The prior posterior analysis provides us with a tool to incorporate prior knowledge about the model parameters, via the prior distributions of the parameters. Since the specific problem and data has not been previously analyzed in the literature we use information from the 2002 drug survey of SIRUS to help specify our prior assumptions. The posterior distribution in our case is not available in closed form, so we develop an estimation algorithm based on Markov Chain Monte Carlo methods (Chib, 2001). We employ the approach discussed in Albert and Chib (1993) to deal with the binary variables by including the latent drug use variables into the parameter space. The details of the algorithm we use to generate draws from the posterior distribution of the parameters are given in the appendix. Of particular interest for our analysis are inferences for each parameter by itself, which is given by the marginal posterior distribution of the parameter. These can be easily obtained from the draws of the joint posterior distribution. By construction of the MCMC algorithm, the draws for each parameters come from the marginal posterior distribution of that particular parameter. We can thus summarize the posterior distribution of the model parameters based on the corresponding draws. Usually this is done in terms of the means and standard deviations of the marginal posterior distribution and/or directly in graphical form.

4.3 Predictive Analysis

While the inferences about coefficients from the prior-posterior analysis will provide us with some information about the effects of the accessibility and proneness related variables and previous cannabis use, the coefficients of the probit model are not easily interpreted. Further, the coefficient estimates will not allow us to assess the overall impact of the observed accessibility and proneness related factors on a subject's probability of hard drug uptake compared to previous cannabis use. To address this issues we employ a predictive analysis of the probability of hard drug uptake.

The approach draws inferences about the hard drug initiation of a random future observation from the population (denoted with $n+1$) based on our observed sample data $(\mathbf{c}, \mathbf{h}, \mathbf{W})$ and the inferences about the model parameters summarized in the posterior distribution of the model

parameters (from the model fitting). In our particular context we are interested in the probability that a random subject from the population will initiate hard drug uptake under no and previous cannabis use. We then explore how these probabilities are affected by accessibility and proneness related factors. We also explore the direct effect of prior cannabis intake use in the hard drug probability of cannabis users.

The marginal predictive probabilities of hard drug use $\mathsf{Pr}(h_{j,n+1} = 1|\mathbf{c}, \mathbf{h}, \mathbf{W})$, $j = 0, 1$ can be computed based on the marginal predictive distribution of the latent hard drug intake for a subject as

$$\mathsf{Pr}(h_{j,n+1} = 1|\mathbf{c}, \mathbf{h}, \mathbf{W}) = \mathsf{Pr}(h^*_{j,n+1} > 0|\mathbf{c}, \mathbf{h}, \mathbf{W}) \tag{4.4}$$

We compute this quantity of interest from the marginal distribution of hard drug intake conditional on the model parameters $p(h^*_{j,n+1}|\mathbf{h}, \mathbf{s}, \mathbf{W}, \mathbf{w}_{h,n+1}, \boldsymbol{\beta}_j, \boldsymbol{\alpha})$ by integrating over the model parameters using the posterior distribution of the model parameters from the model fitting and over the covariates using the empirical distribution from the sample (see appendix for details).

To see how we use the predictive approach to assess the effect of accessibility, proneness and the direct gateway effect on the predictive probability, we express the marginal predictive distribution as the following integral based on the conditional predictive distribution $p(h^*_{j,n+1}|\mathbf{h}, \mathbf{s}, \mathbf{W}, \mathbf{w}_{h,n+1}, \boldsymbol{\beta}_j, \boldsymbol{\alpha})$:

$$\int p(h^*_{j,n+1}|\mathbf{h}, \mathbf{s}, \mathbf{W}, \mathbf{w}_{h,n+1}, \boldsymbol{\beta}_j, \boldsymbol{\alpha})\pi(\boldsymbol{\beta}_j, \boldsymbol{\alpha}|\mathbf{h}, \mathbf{s}, \mathbf{W})p(\mathbf{w}_{h,n+1}|\mathbf{h}, \mathbf{s}, \mathbf{W})\ d\boldsymbol{\beta}_j\ d\boldsymbol{\alpha}\ d\mathbf{w}_{h,n+1}$$

From the model assumptions in Section 4.2 it follows that the likelihood of the future observation $n + 1$, the latent hard drug intake $h^*_{j,n+1}$, is just a Normal distribution $\mathcal{N}(h^*_{j,n+1}|\boldsymbol{\mu}_{j,n+1}, 1)$ where the mean is a function of cultural and physical accessibility factors, prices, proneness and demographic factors:

$$\boldsymbol{\mu}_{j,n+1} = \mathbf{a}'_{h,n+1}\boldsymbol{\beta}^a_j + \mathbf{p}'_{h,n+1}\boldsymbol{\beta}^p_j + g_{n+1}\alpha^g + \mathbf{pr}'_{h,n+1}\boldsymbol{\alpha}^{pr} + \mathbf{d}'_{n+1}\boldsymbol{\alpha}^d$$

where $g_{n+1} = 1$ if $j = 1$ and $g_{n+1} = 0$ for the $j = 0$ case. To assess how a subject's probability of hard drug uptake is influenced by the various effects we also compute the predictive probabilities setting $\boldsymbol{\beta}^a_j = 0$, $\boldsymbol{\beta}^p_j = 0$, $\alpha^g = 0$ or $\boldsymbol{\alpha}^{pr} = 0$ (see Table 3). For example, comparing the obtained predictive probabilities without the effects of observed accessibility with the total predictive

probability allows us to evaluate the average marginal effect of accessibility in predicting a subjects hard drug uptake. Since this calculation can only be based on observed accessibility, these results should be interpreted with care and understood more as a lower limit of the role of accessibility effects play in hard drug uptake.

5 Estimation Results

5.1 Results from Prior-Posterior Analysis

In this section we discuss the results from the prior-posterior and predictive analysis of the Norwegian drug use data based on the extended gateway model that we described in the previous section. In Table 2 we summarize the estimation results for the extended gateway model in terms of the means and standard deviations of the posterior distributions of the parameters. Our results are based on 10,000 iterations of the MCMC algorithm (see Appendix) after a burn-in phase of 1,000 iterations. For comparison, we also provide selected results from the fitting of a restricted version with only a constant effect of previous cannabis use on hard drug uptake (restricted gateway model).

A comparison of the first block of results for the hard drug model (columns 2 and 3) indicates that accessibility matters and that the effect of the accessibility related variables on hard drug initiation varies by previous cannabis use. For example, we see that living in the country or in a city other than Oslo increases the likelihood of a cannabis user to take up hard drugs, as does living in an area with high supply (seizure), high prevalence and being of non-western background. The latter has a negative effect for non-cannabis users as does living outside Oslo. The effects of the other variables are small and estimated with a lower precision for non-cannabis users as indicated by the high posterior standard deviation. The low precision is not surprising as there is only a small number of hard drug users without previous cannabis use in the data (n=102). Further, our estimates show no effect of amphetamine price on the probability of hard drug uptake of cannabis users, while the effect is positive but estimated with a low precision for non-users.

For cannabis uptake (column 4) the coefficient on cannabis price is negative, but also im-

	Extended Gateway Model			Restricted Gateway Model
	Hard uptake		Can uptake	Hard uptake
	no can	can		
Country	0.12 (0.19)	0.37 (0.18)	-0.27 (0.10)	0.17 (0.14)
Oslo	0.46 (0.28)	-1.11 (0.46)	-0.03 (0.11)	-0.06 (0.25)
Obtain	0.48 (0.99)	-0.02 (1.06)	0.15 (0.71)	0.05 (0.75)
Seizure	0.11 (0.67)	3.21 (1.11)	-	0.97 (0.61)
Prevalence	0.20 (1.31)	2.46 (1.97)	2.26 (0.91)	1.47 (1.23)
Non-Western	-0.31 (0.19)	0.48 (0.29)	-0.54 (0.13)	-0.08 (0.15)
Amphetamine Price	0.47 (0.41)	0.04 (0.45)	-	0.23 (0.35)
Cannabis Price	-	-	-0.62 (1.38)	-
Cannabis Use	1.20 (0.54)		-	1.10 (0.41)
Police prob.	0.90 (0.22)		0.62 (0.19)	0.89 (0.21)
Parents prob.	0.45 (0.11)		0.42 (0.08)	0.46 (0.10)
School prob.	-0.02 (0.13)		0.25 (0.11)	0.01 (0.13)
Friends prob.	-0.04 (0.16)		-0.09 (0.13)	-0.02 (0.15)
Leave School early	0.52 (0.12)		0.19 (0.11)	0.50 (0.12)
Alcohol young	0.28 (0.11)		0.41 (0.09)	0.31 (0.11)
Smoker	0.42 (0.15)		1.17 (0.06)	0.41 (0.16)
Male	0.28 (0.07)		0.43 (0.05)	0.28 (0.08)
Age2124	-0.01 (0.10)		0.03 (0.06)	-0.02 (0.09)
Intercept	-2.81 (0.48)		-2.27 (0.39)	-2.65 (0.37)
Correlation	0.34 (0.23)	0.06 (0.20)		0.26 (0.14) 0.15 (0.21)

Table 2: *Posterior means and standard deviations (parentheses) for parameters in the cannabis and hard drug equation from the prior posterior analysis of the extended gateway model.*

precisely estimated. This suggests that prices (as measured here) are not of great importance for drug initiation. However, we cannot rule out that this is, at least partly, due to the poor data situation (only data for Oslo is available) and to how the price variable is constructed. For example, Van Ours and Williams (2007) find a negative effect of cannabis price on cannabis initiation. The remaining coefficient estimates for the accessibility variables in the cannabis model suggest that accessibility factors also matter for cannabis intake. Especially cannabis prevalence, obtainability and non-western cultural background affect cannabis uptake, while living in Oslo has no effect. The negative effect of the Oslo variable on the hard drug uptake of cannabis users versus the positive effect for non-users suggests that a cultural acceptance of cannabis use may not translate into a cultural acceptance of hard drug uptake.

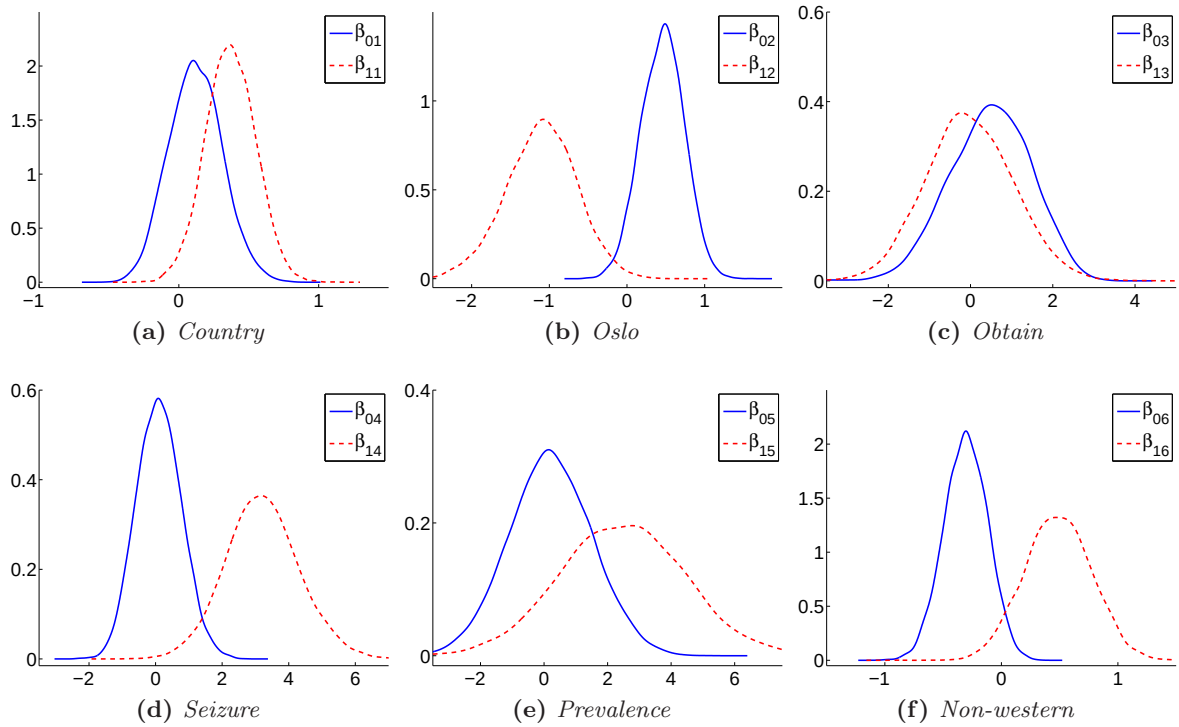
We now turn to the estimates of the direct effect of previous cannabis use and proneness factors. We estimated a large positive effect of previous cannabis use on the probability of hard

drug uptake. Further, we find a large positive effect of childhood problems with police and parents, leaving school early, early alcohol use and smoking. These factors also increase the likelihood of cannabis use. In contrast, problems in school and with friends do not have seem to have an effect on hard drug uptake. We find the same results for hard drug uptake in terms of previous cannabis use and the proneness variables in our estimation of the simple model (see last column of Table 2), with the difference that the gateway effect is estimated with a higher precision. This is not the case for the results on the accessibility and price variables and we see that the coefficient on Oslo, Non-Western and the price variables are now close to zero. Except for living in the country, which has a positive effect, the coefficients on the remaining variables are poorly estimated.

Our findings with regard to unobserved confounders are expressed in terms of the correlation coefficients presented in the bottom row of the table. For non-users of cannabis our estimation provides evidence for a positive correlation, while the results for cannabis users do not. Previous work that have used a more restrictive model formulation has found positive correlation for the overall population (Bretteville-Jensen et al 2008), and under such restrictions we too estimate a positive correlation for both groups.

For a more detailed summary of the accessibility related coefficients in the hard drug model we take a closer look at the posterior distributions of the accessibility coefficients from the extended gateway model. In Figure 2 we provide plots of the posterior distributions of the coefficients for accessibility variables in vectors $\boldsymbol{\beta}_0$ and $\boldsymbol{\beta}_1$. The solid line refers to no previous cannabis use and the dashed line to previous cannabis use. Except for the case of the obtain variable (third figure in 1st row), the two lines have different regions of support (some overlapping). These graphs thus provide further evidence for the extended gateway effect in that previous cannabis use affects hard drug uptake via accessibility variables, and we therefore proceed with our further analysis under the extended gateway model. In section 6 where we consider a range of alternative model specifications, e.g. alternative definitions for the dependent variables and alternative vectors of covariates.

Note that our estimates for the accessibility variables in the two hard drug models, presented



(a) *Country* **(b)** *Oslo* **(c)** *Obtain*

(d) *Seizure* **(e)** *Prevalence* **(f)** *Non-western*

Figure 2: *Plots of the Posterior Distributions for the coefficients on oslo, prevalence and non-western under no cannabis use (blue line) and cannabis use (red dashed line).*

in Table 2 and Figure 2, are interesting in regard to the possible transmission mechanisms of cannabis use. In particular, the fact that physical accessibility, measured by the seizure and prevalence variables, has a large positive effect under previous cannabis use compared to almost no effect under no previous cannabis use may be seen as evidence of the transmission channel 2 and 3. Further, we find that previous cannabis use has a strong positive direct positive effect on hard drug uptake. We interpret this as evidence for transmission channels 1 and/or 4. According channel 1 the coefficient reflects an increase physiological and physical need for stronger drugs. Under channel 4 the positive coefficient reflects a better knowledge of the drug market. We further explore these issues in our sensitivity analysis Section 6 where we consider a range of alternative model specifications.

5.2 Results from Predictive Analysis

We employ the predictive analysis to explore the importance of accessibility and proneness related factors, in addition to that of previous cannabis intake, on a subject's probability of hard

drug uptake. We first investigate the effects for a random (average) subject from the population. From the first row in Table 3 we see that the subject's probability to take up hard drugs is 7.6% under no previous cannabis use and 36.2% under previous cannabis use. As discussed in Section 4.3, these two probabilities refer to the two counterfactual scenarios if we were to observe the same subject under no and previous cannabis use. The estimates are based on the marginal model of hard drug uptake and are corrected for unobserved confounding (selection on unobservables). If we assume that we have successfully accounted for all unobserved confounders

Predictive Probabilities for Extended Gateway Model					
		informed prior		ignorant prior	
		no cannabis	cannabis	no cannabis	cannabis
Total	$\mathsf{Pr}(h_{j,n+1} = 1)$	0.076	0.362	0.081	0.351
Partial	w/o Access*	0.036	0.164	0.040	0.136
		(4pp)	(19.8pp)	(4.1pp)	(21.5pp)
	w/o Prices	0.053	0.363	0.060	0.350
		(2.3pp)	(-0.1pp)	(2.1pp)	(1.0pp)
	w/o Gateway	-	0.094	-	0.130
		-	(26.8pp)	-	(22.1pp)
	w/o Proneness	0.035	0.257	0.036	0.243
		(4.1pp)	(10.5 pp)	(4.5pp)	(10.8pp)

Table 3: *Estimates for total and partial marginal predictive probabilities of hard drug uptake based on the extended model under two different prior assumptions. The informative prior refers to priors based on the 2002 data as discussed in Section 4.2. For the ignorant prior we centered the prior distributions for all parameters at zero. *As before access includes all access related variables from main specification other than prices*

through our flexible joint model, the difference of 28 percentage points can be interpreted as the causal effect of cannabis use on hard drug uptake.

Table 3 also reports the estimated marginal predictive probabilities without the effect of accessibility, price and proneness factors as well as the direct "gateway" effect (under previous cannabis use only). As discussed in Section 4.3, these "partial" probabilities are obtained by setting the corresponding coefficients to zero. Within each row, the difference between the partial and the total probabilities (reported in parentheses) can be interpreted as a kind of average marginal effects. For example, without the effect of accessibility related factors, and holding everything else constant, the probability of a random subject to take up hard drugs by drops by 4 percentage points, from 7.6% to 3.6%, if we assume the subject had no previous

cannabis use. Under previous cannabis use the probability would drop by 19.8 percentage points, from 36.2% to 16.4%. The average marginal effect of proneness on the probability of hard drug uptake is 4.1 percentage points under no previous cannabis use and 10.5 percentage points under previous cannabis use. The largest effect is from the direct gateway effect with 26.8 percentage points. As expected from the estimates on the price coefficient the effect of prices is the smallest.

Finally, we also report the predictive results for the analysis of the extended gateway model under alternative prior assumptions that are not based on the 2002 data (ignorant prior) in Table 3. The estimates of the predictive probabilities as well as those of the model parameters (see Table 10 in appendix) are comparable to those from the analysis under the informed priors. We do observe a smaller decrease in the probability due to the direct gateway effect. This is driven by a smaller mean of the posterior distribution of the coefficient on previous cannabis use. Overall the estimates in Table 3 suggest that accessibility and proneness both play an important role in hard drug uptake of subjects. Both proneness and accessibility have a larger effect on the probability of hard drug uptake for cannabis user, which contributes to the observed pattern of higher hard drug use among cannabis users. Since we can only assess the effects based on the available limited data on proneness and accessibility, it is very likely that our estimates correspond to a lower boundary of the effects. We also note that especially the effect of accessibility related variables is much higher under previous cannabis use. This is to a large extent driven by the different coefficient estimates on accessibility factors under the two cannabis use states that we discussed in previous section. We also observe a larger marginal average effect of proneness under previous cannabis use which is a result of the non-linear transformation of in the probit model.

In Table 4 we provide an alternative summary of the effects of previous cannabis use, accessibility and proneness for a male and female living in Oslo. The tables shows the predicted probability for an average male (female) in Oslo, as well as for a male (female) under high and low accessibility, hard drug prices and proneness. As expected given the positive coefficient on male in the hard drug model, males have a higher probability of hard drug uptake than females, as seen in the first row of results. The remaining results in the table show how these base

Predictive Probabilities for Various Scenarios				
	Male		Female	
	cannabis	no cannabis	cannabis	no cannabis
Benchmark	0.269	0.126	0.196	0.081
high access	0.458	0.134	0.349	0.085
low access	0.051	0.116	0.031	0.071
high prices	0.271	0.140	0.199	0.091
low prices	0.267	0.114	0.195	0.073
high proneness	0.842	0.809	0.783	0.733
low proneness	0.185	0.062	0.127	0.036

Table 4: *Predictive probabilities of hard drug uptake for males and females under various scenarios based on the extended gateway model.*

probabilities for males and females are affected by changes in accessibility, prices and proneness. The access scenario explores the effect of a move from an area with 50% increase in seizure and prevalence of hard drugs compared to an area with a 50% decrease. The proneness scenarios explore the effect of a 50% increase in having problems in police and parents versus having no problems. The estimates show that changes in accessibility and proneness can have a very large impact on the hard drug uptake both of cannabis users and non-users.

6 Sensitivity Analysis

In this section we present selected results from the prior posterior and predictive analysis from different specifications of the extended gateway model. We consider different frequency definitions of the cannabis and hard drug variables as well as different specifications of the variable vector in the hard drug equation. The purpose is 1) to check the robustness of our findings from the previous section regarding the direct and extended gateway effects and the importance of accessibility, proneness and previous cannabis use for hard drug uptake; and 2) to aid the interpretation of our results with respect to the gateway mechanisms.

In Tables 5 and 6 we provide results from the fitting and the predictive analysis of the extended gateway model under two alternative frequency definitions of the cannabis and hard drug variables, c_i and h_i. So far we have defined a cannabis user ($c_i = 1$) as a person that has used cannabis at least 5 times (and $c_i = 0$ otherwise) and a hard drug user ($h_i = 1$) as a person who has used hard drugs at least once (and $h_i = 0$ otherwise). Under Specification 1 we now

Some Results for Various Specifications of Variable Vectors						
	Spec 1		Spec 2		Spec 3	
	Hard uptake		Hard uptake		Hard uptake	
	no can	can	no can	can	no can	can
Country	0.03 (0.24)	0.23 (0.16)	0.05 (0.24)	0.25 (0.19)	0.13 (0.16)	0.41 (0.17)
Oslo	0.48 (0.34)	-0.73 (0.38)	0.27 (0.36)	-1.12 (0.50)	0.46 (0.28)	-1.06 (0.44)
Obtain	0.21 (1.09)	0.03 (0.91)	-0.37 (1.10)	-0.18 (1.13)	-	-
seizure	-0.05 (0.78)	2.38 (0.92)	0.25 (0.83)	2.78 (1.19)	0.10 (0.64)	3.00 (1.03)
Prevalence	-0.16 (1.34)	2.83 (1.84)	0.03 (1.36)	2.55 (1.99)	0.18 (1.34)	2.15 (1.89)
Non-Western	-0.08 (0.22)	-0.10 (0.21)	-0.22 (0.24)	0.37 (0.27)	-0.34 (0.20)	0.60 (0.29)
Amph. Price	0.49 (0.50)	-0.08 (0.37)	0.54 (0.55)	-0.18 (0.50)	-	-
Can. Use	0.93(0.48)		1.24(0.57)		1.35(0.51)	
Intercept	-2.91(0.54)		-2.81(0.57)		-2.12(0.23)	
smoking	included		included		-	
Other*	included		included		included	

Table 5: *Specifications 1 and 2 refer to the main model with the different frequency definitions of the depended variables, freq11 and freq22. *All specifications include the standard proneness and demographic variables from the main estimation.*

define a person as cannabis (hard drug) user if he/she has used cannabis (any hard drug) at least once. Under Specification 2 we define a person as cannabis (hard drug) user if he/she has used cannabis (any hard drug) at least five times. We find that the gateway coefficient varies from 0.93 for Specification 1 to 1.24 for Specification 2 compared to 1.20 in the main model. The predicted probabilities for these specifications differ somewhat more from the main model (Table 6), as also the raw data on the frequencies of use suggests (see Appendix B). As before, accessibility and proneness contribute significantly to hard drug uptake in addition to previous cannabis use, composed of a direct effect and a extended effect via the accessibility variables.

We need to acknowledge potential endogeneity issues with respect to some of our control variables, in particular for the lifetime smoking and the obtain variable, which may bias our results. To address this concern, we have estimated the extended gateway model without these variables (Specification 3). Again we find that our results do no change dramatically compared to those in the main model. As expected, the exclusion of the smoking variable from the proneness vector leads to a smaller overall effect of proneness on hard drug uptake (Table 6). We do observe a higher total predictive probability of hard drug uptake for cannabis users in this more restricted specifications.

A comparison of the coefficient estimates (posterior means) from the fitting of Specifications

Predictive Probabilities Under Various Specifications								
	Spec 1		Spec 2		Spec 3		Spec 4	
	no can	can	no can	can	no can	can	no can	can
Total	0.057	0.218	0.035	0.246	0.063	0.543	0.088	0.303
w/o Access	0.040	0.092	0.038	0.142	0.032	0.293	0.051	0.161
w/o Prices	0.039	0.234	0.024	0.279	-	-	0.067	0.259
w/o Gateway	-	0.071	-	0.053	-	0.131	-	0.185*
w/o Proneness	0.020	0.128	0.011	0.154	0.050	0.517	0.032	0.199

Table 6: *Predictive Probabilities for various specifications of the extended gateway model. *Gateway here does not include the early use effect.*

1 in Table 5 to those from the main model in Table 2 yields some potentially interesting insights and interpretations and speculations regarding the transmission channels of previous cannabis use on hard drug uptake. First, we find that the posterior mean of the coefficient on previous cannabis use decreases from 1.20 to 0.90 if we include the lowest frequency cannabis user (between one and four times) into the group of cannabis users. This observations is coherent with the fact that one would expect the strength of the transmission channels 1 and 4 to be lower for accidental and low frequency cannabis users as they not have developed a strong psychological or physiological need for harder drugs and are less knowledgeable about the drug market. Further, we don't see large changes in the coefficients on the accessibility variables. This observations is consistent with channels 2 and 3. Using cannabis only once or twice is sufficient for a subject to pass the threshold to illegal drugs and to be less concerned about their potential harmful effects, increasing the probability that these take up hard drugs given the opportunity.

Another issue that we should address here is that of early cannabis use. It has been suggested in the literature that starting with cannabis at a young age will reinforce the gateway effect (see e.g. Fergusson et al. 2006). In addition to the indicator for previous use we therefore consider a specification (4) with an additional dummy variable for early cannabis use in the hard drug model (results not shown in Table 5). The variable is set equal to 1 if the respondents report to have used cannabis before they turn 16. We find that early cannabis use has a positive effect on hard drug uptake (0.71), while we now observed a lower mean of the posterior distribution of previous cannabis use of 0.60 compared the estimate of 1.20 for the main specification (Table 2).

One might speculate that this implies that the estimated direct gateway effect to a large extent is driven by early cannabis use. However, the early use variable might be associated with endogeneity issues as we cannot rule out that early use is at least partially driven by unobserved proneness factors that also drive hard drug uptake (We do observe an increase in the gateway coefficient if we omit the smoking variable in Spec 4). If, on the other hand, the early use coefficient reflects mainly unobserved proneness that for some reason has not been captured by our joint modeling, then this would imply a smaller gateway effect than the one presented in Table 2. Finally, we should note that the inferences of the other coefficients remain fairly stable and we again observed an extended gateway effect. This is also born out in the estimates from the predictive analysis for Specification 4 presented in Table 6.

7 Policy Implications

A robust finding that emerges from our previous analysis is that in addition to previous cannabis use, accessibility and proneness related factors play an important role in explaining hard drug uptake among cannabis users and contribute to the observed higher hard drug uptake among them. We have also found an interesting link between previous cannabis use and the effect of accessibility factors. In this section we explore possible policy implications of our empirical analysis using the model specification from main empirical section (Table 2). In Table 7 we report the predictive probabilities of hard drug uptake under various hypothetical scenarios. For example, we consider a scenario where the availability of hard drugs (measured by seizure and prevalence) will change in response to policy interventions. Our results suggest that a 50% decrease in availability would lead to a 36% reduction in the overall probability of hard drug uptake (from 0.134 to 0.086, see last column of Table 7). As expected, increased accessibility will have the opposite effect (up 19%, from 0.134 to 0.165). While changes in accessibility will hardly affect non-users of cannabis, the impact on cannabis users is relatively large.

We also examine two different scenarios for changes in the proneness variables. First, if the sample prevalence of problems with parents and police doubles our estimations suggests that the overall probability increases to 0.144, whereas setting the two problem variables to

Predicted Hard Drug Uptake for Policy Scenarios			
	Cannabis	No Cannabis	Overall
Base	0.362	0.076	0.134
high hard seiz and prev	0.545	0.076	0.165
low hardseiz and prev	0.236	0.074	0.086
more problems pol an par	0.378	0.085	0.144
no problems pol an par	0.341	0.067	0.116
more early alc and leave school	0.377	0.083	0.151
no early alc and leave school	0.346	0.068	0.124
high can prev	-	-	0.156
low can prev	-	-	0.116
high can price	-	-	0.129
low can price	-	-	0.137
high gateway	-	-	0.188
lower gateway	-	-	0.089

Table 7: *Predicting Probabilities for different scenarios for males and females using estimation results from the extended gateway model.*

zero reduces the corresponding probability to 0.116. A successful policy towards reducing early alcohol initiation and school drop-outs would have a somewhat smaller effect and reduce the overall probability to 0.124. Note that these changes are based only on the model for hard drug intake and do not directly consider the interaction between cannabis and hard drug use. This means that if the policy scenarios discussed here also influence the probability of cannabis uptake, the results are likely to underestimate the overall effect on hard drug use in the population.

Given the link between cannabis and hard drug use, however, a very interesting policy question is how changes in the accessibility of *cannabis* affect hard drug uptake. By employing the conditional predictive probability of hard drug uptake (instead of the marginal predictive probability), we can explore the question. The idea behind this measure is to first predict a random subject's cannabis intake and then, conditional on the predicted cannabis use, predict hard drug uptake (see Appendix A). Lowering the prevalence of cannabis use by 50% (everything else held constant) would decrease the probability of hard drug uptake (to 0.116), while a 50% increase in the prevalence raises the probability to 0.156. Changes in the cannabis price, on the

other hand, do not seem to affect the probability of hard drug uptake to a large extent.

Lastly, we consider a change in the strength of the direct effect of previous cannabis use. For instance, a policy aiming at separating the markets for cannabis and hard drugs (in line with what is found in the Netherlands), may reduce the effect of previous cannabis use on hard drug initiation if the policy change reduces the social interaction between different groups drug users (mechanism 4 of those listed in section 1). In contrast, a more profound gateway effect than the one estimated here, can also be a possible future scenario as improved cultivation techniques have resulted in cannabis with higher THC levels, i.e. more potent cannabis. More potent cannabis could imply stronger addiction to the drug and more craving also for other, and stronger, drug experiences (mechanism 1 in section 1). According to the World Drug Report 2007 (UN 2007), the prevalence of this high-potent cannabis has increased substantially in recent years. The bottom rows of Table 7 show that changes by 50% in the coefficient on previous cannabis use are the scenarios with the largest effect on hard drug initiation. A decrease will reduce the probability from 0.134 to 0.089 and an increase will raise it to 0.188.

8 Discussion

Extensive use of hard drugs is associated with substantial health risks and adverse consequences for educational achievements, social relationships, employment, careers etc. Most countries therefore rely on a drug policy aiming to reduce the use and misuse of substances such as cocaine, amphetamine, ecstasy and heroin and many uphold also a strict cannabis regime. Still, global estimates suggest that currently there are about 160 millions cannabis users and also millions of hard drug users (UN 2007). To improve drug policy means a better understanding of the factors influencing hard drug uptake is needed.

Using data from a representative sample of Norwegian adults (21-30 years) and a novel modeling approach, we have examined the influence of previous cannabis use, accessibility and proneness factors on the probability of hard drug uptake. To the best of our knowledge, this paper is the first attempt in the literature to examine the three hypotheses simultaneously and to quantify the relative importance of the three effects on the probability of hard drug uptake. Our

results show that all three factors matter, with the effect of previous cannabis use dominating the effects from the observed accessibility and proneness factors. Our predictive analysis suggests that, for a random person, the probability of taking up hard drugs increases from roughly 8 per cent, if he/she has not previously used cannabis, to 36 per cent for cannabis users. Both proneness and accessibility have larger effects for cannabis users than for non-cannabis users, with the overall effects, measured as marginal average effects, from accessibility being more important than that of proneness. This is partly due to the fact that even though many of the proneness variables proved important, relatively few individuals reported problems with police and parents or started to drink alcohol before they turned 13. As shown in the sensitivity analysis, the findings are robust across various model specification (alternative definitions of the dependent variables and sets of covariates), all taking account also of unobserved factors.

Further, our model of hard drug uptake as a function of cannabis use (motivated by the hypothesized transmission channels) helps us to gain some insights about the suggested transmission mechanisms that could underpin the reported gateway effect. First, for cannabis to cause a psychological and/or physiological need for stronger drug experiences one would assume that the drug is used with some frequency. In line with this we find that the gateway coefficient increases with frequency of use (Table 5). It has also been suggested that starting with cannabis very young could reinforce the gateway effect, and animal studies (Ellgren et al. 2007) and twin studies examining same-sex twin pairs discordant for early cannabis initiation (see e.g. Lynskey et al. 2006) seem to support this. The results from Specification 4 in the sensitivity analysis suggest the same, namely that starting with cannabis before turning 16 has a positive effect on the probability of hard drug uptake.

Second, both the mechanism regarding lower credibility in warnings and that having used one illegal drug reduces the costs of also trying another, imply a lower threshold for hard drugs after cannabis has been initiated. While a more restricted hard drug model, with only a dummy for previous cannabis use, cannot isolate potential threshold effects, we find support the hypothesis in terms different effects of the accessibility variables for cannabis users versus non-users in our extended gateway model: Given the same degree of physical accessibility, cannabis users,

who have passed the threshold/have lower costs, are more likely to take up hard drugs. The accessibility coefficients show little variation across the different frequency specification, which indicates that once one has passed the threshold, frequency of use is not of great importance for the accessibility effects.

Finally, without being able to include any variables reflecting individual variation in social interaction or market knowledge in the estimations, the fourth mechanism is hard to examine separately. The direct gateway effect we report may reflect this transition channel but may also reflect psychological and/or physiological effects. As mentioned, however, the raw data strongly suggest that there are differences between cannabis users and non-users with regard to reported obtainability of hard drugs. The table presented in Appendix B shows that while only 31, 23, 19 and 29 per cent of non-users claim to be able to obtain amphetamine, cocaine, heroin and ecstasy, respectively, within three days the corresponding numbers for cannabis users are 62, 57, 33 and 48 per cent. Also more cannabis users report that they have been offered hard drugs (80 vs 26%). Probable endogeneity problems refrained us from including these variables in the estimations. Based on our findings, however, we would argue that there are support in the data for all the suggested mechanisms.

When it comes to policy implications, the finding of a substantial gateway effect suggests that any intervention successful in reducing cannabis use also will have a positive effect on the uptake of hard drugs. Previous cannabis use is the single most important variable for the increased probability of starting to use drugs like amphetamine and cocaine. Further, for any given level of cannabis use, means for weakening the transition mechanisms should be of interest to policy makers. For instance, a separation of markets for cannabis and hard drugs may prove effective in reducing hard drugs uptake if that would reduce the influence hard drug users and knowledge of those markets might have on current cannabis users. Increased credibility of health warnings, something which might be achieved by providing more drug specific information instead of focusing on illegal drugs in general, is another means to weaken the transition mechanisms. The recent increase in the prevalence of high-potent cannabis may, on the other hand, strengthen the effect of cannabis use. Further, our findings regarding observed proneness factors indicate that

intervention directed against high risk groups, e.g. early detection and adequate interference towards subgroups with high prevalence of the problem indicators (early alcohol debut, low educational achievements, problems with parents and police), will reduce hard drugs initiation. Finally, our results suggest that means aiming at reducing the cultural and physical accessibility of drugs are likely to be more successful than actions aiming at increasing drug prices.

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9 Appendix A: Markov Chain Monte Carlo Methods

9.1 Prior-Posterior Analysis

Due to the structure of the likelihood function, the posterior distribution $\pi(\boldsymbol{\theta}|\mathbf{h},\mathbf{c},\mathbf{W})$ of the vector of model parameters $\boldsymbol{\theta} = (\boldsymbol{\beta}_0, \boldsymbol{\beta}_1, \boldsymbol{\alpha}, \boldsymbol{\gamma}, \rho_0, \rho_1)$ is not readily estimable via Markov Chain Monte Carlo (MCMC) methods. We can however, exploit the ideas in Albert and Chib (1993) and include the latent drug intake variables in the parameter space and work with the augmented posterior distribution $\pi(\boldsymbol{\theta}, \mathbf{c}_i^*, \mathbf{h}_{ji}^*|\mathbf{h},\mathbf{c},\mathbf{W})$. which is given by the expression

$$\pi(\boldsymbol{\theta}, \mathbf{h}_{ji}^*, \mathbf{c_i}^*|\mathbf{h},\mathbf{c},\mathbf{W}) \propto \pi(\boldsymbol{\theta}) p(\mathbf{h},\mathbf{c},\mathbf{h_i}^*,\mathbf{c_i}^*|\mathbf{W},\boldsymbol{\theta}),$$

where the joint likelihood of the augmented parameter space $p(\mathbf{h},\mathbf{c},\mathbf{h}_{ji}^*,\mathbf{c_i}^*|\mathbf{W},\boldsymbol{\theta})$ takes the form

$$\prod_{i=1}^{N} \mathcal{N}_2(h_{ji}^*, c_i^*|\mathbf{W}'_{ji}\,\boldsymbol{\delta}, \boldsymbol{\Omega}_j)\,[I\{c_i^* \le 0\}^{1-c_i} + I\{c_i^* > 0\}^{c_i}]\,[I\{h_i^* \le 0\}^{1-h_i} + I\{h_i^* > 0\}^{h_i}] \quad (9.1)$$

$h_i = h_{0i}$ if $c_i = 0$ and $h_i = h_{1i}$ if $c_i = 1$. The prior distribution is given by expression (9.2). The resulting joint posterior distribution is of a type that can be efficiently processed by MCMC methods. We propose the following a six step MCMC chain to generate draws from the posterior distribution. We let $\mathbf{c} = (\mathbf{c}_0, \mathbf{c}_1)$ where $\mathbf{c}_0 = (c_i : i \in N_0)$ and $\mathbf{c}_1 = (c_i : i \in N_1)$ represent the cannabis observations under the two possible cannabis states; with a similar convention for the latent cannabis intake is $\mathbf{c}^* = (\mathbf{c}_0^*, \mathbf{c}_1^*)$. We propose the following algorithm to generate draws from the posterior distribution for the extended gateway model proposed in Section 4.1-2. The algorithm is run for M (for example $M = 10,000$) iterations. A burn-in phase of 1,000 iterations is used to ensure that MCMC chain has converged to the posterior distribution of interest.

MCMC Algorithm:

1. Initialize $\boldsymbol{\beta}_j, \boldsymbol{\alpha}, \boldsymbol{\gamma}, \rho_j, \{c_i^*\}$ for $j = 0, 1$

2. Sample $h_{j,i}^*|h_i, c_i, c_i^*, \boldsymbol{\beta}, \boldsymbol{\alpha}, \boldsymbol{\gamma}, \rho$ for all subjects in N_1 from the conditional truncated normal
   $N(h_{j,i}^*|\mathbf{w}'_{1h,i}\boldsymbol{\beta}_j + \mathbf{w}'_{2h,i}\boldsymbol{\alpha} + \rho_j(c_i^* - \mathbf{w}'_{c,i}\boldsymbol{\gamma}), 1-\rho_j^2)\,[I\{h_i^* \le 0\}^{1-h_i} + I\{h_i^* > 0\}^{h_i}]$

3. Sample $c_i^*|h_i, c_i, h_i^*, \boldsymbol{\beta}_j, \boldsymbol{\gamma}, \rho_j$ from the conditional truncated normal

   $N(c_i^*|\mathbf{w}'_{c,i}\boldsymbol{\gamma} + \rho_j(h_i^* - \mathbf{w}'_{1h,i}\boldsymbol{\beta}_j - \mathbf{w}'_{2h,i}\boldsymbol{\alpha}), 1-\rho_j^2)[I\{c_i^* \le 0\}^{1-c_i} + I\{c_i^* > 0\}^{c_i}]$.

4. Sample $\boldsymbol{\gamma}|\mathbf{h},\mathbf{c},\mathbf{h}^*,\mathbf{c}^*,\boldsymbol{\beta},\boldsymbol{\alpha},\rho$ from $\mathcal{N}_k(\boldsymbol{\gamma}|\hat{\boldsymbol{\gamma}},\mathbf{G})$, where
   $\hat{\boldsymbol{\gamma}} = \mathbf{G}[\mathbf{G}_0^{-1}\mathbf{g}_0 + \sum_{i\in N}\mathbf{w}_{c,i}\sigma_j^{-2}(c_i^* - \rho_j\hat{h_i}^*)]$,
   $\mathbf{G} = [\mathbf{G}_0^{-1} + \sum_{i\in N}\mathbf{w}_{c,i}\sigma_j^{-2}\mathbf{w}'_{c,i}]^{-1}$,
   with $\sigma_j^2 = (1-\rho_j^2)$ and $\hat{h_i}^* = h_i^* - \mathbf{w}'_{1h,i}\boldsymbol{\beta}_j - \mathbf{w}'_{2h,i}\boldsymbol{\alpha}$.

5. Sample $\boldsymbol{\beta}_j|\mathbf{h},\mathbf{c},\mathbf{h}^*,\mathbf{c}_1^*,\boldsymbol{\gamma},\rho$ from $\mathcal{N}_p(\boldsymbol{\beta}|\hat{\boldsymbol{\beta}},\mathbf{B})$, where

   $\hat{\boldsymbol{\beta}}_j = \mathbf{B}_j[\mathbf{B}_{j,0}^{-1}\mathbf{b}_{j,0} + \sum_{i\in N_j}\mathbf{w}_{1h,i}\sigma_j^{-2}(h_i^* - \mathbf{w}'_{2h,i}\boldsymbol{\alpha} - \rho_j\hat{c_i}^*)]$,
   $\mathbf{B}_j = [\mathbf{B}_{j,0}^{-1} + \sum_{i\in N_j}\mathbf{w}_{1h,i}\sigma_j^{-2}\mathbf{w}'_{1h,i}]^{-1}$ with $\sigma_j^2 = (1-\rho_j^2)$,
   and $\hat{c_i}^* = c_i^* - \mathbf{w}'_{c,i}\boldsymbol{\gamma}$

6. Sample $\boldsymbol{\alpha}|\mathbf{h}, \mathbf{c}, \mathbf{h}^*, \mathbf{c}_1^*, \boldsymbol{\gamma}, \rho$ from $\mathcal{N}_m(\boldsymbol{\alpha}|\hat{\boldsymbol{\alpha}}, \mathbf{A})$, where
$\hat{\boldsymbol{\alpha}} = \mathbf{A}[\mathbf{A}_0^{-1}\mathbf{a}_0 + \sum_{i\in N} \mathbf{w}_{2h,i}\sigma_j^{-2}(h_i^* - \mathbf{w}'_{1h,i}\boldsymbol{\beta}_j - \rho_j \hat{c_i}^*)]$,
$\mathbf{A} = [\mathbf{A}_0^{-1} + \sum_{i\in N_j} \mathbf{w}_{2h,i}\sigma_j^{-2}\mathbf{w}'_{2h,i}]^{-1}$ with $\sigma_j^2 = (1-\rho_j^2)$,

7. Sample $\rho_j|\mathbf{h}_j, \mathbf{s}_j, \mathbf{h}_j^*, \mathbf{s}_j^*, \boldsymbol{\beta}_j, \boldsymbol{\gamma}$, for $j = 0, 1$ from an MH step described below.

8. Goto 2

Since the full conditional distribution of the correlation coefficient ρ_j is not tractable, we update the parameters using the Metropolis Hastings algorithm. Following Chib and Greenberg (1995,1998) we generate proposal values ρ'_j from a tailored student-t density $t_\nu(\mu_j, V_j)$ where μ_j is the approximate mode of

$$\mathsf{ln}(\prod_{I\in I_j} \mathcal{N}(h_i^*, s_i^*|\mathbf{W}_i\boldsymbol{\delta}_j, \boldsymbol{\Omega}_j)$$

and V is the inverse Hessian of this density evaluated at μ_j. We accept the proposal value with probability of move α_j where

$$\alpha_j = \left\{1, \frac{\pi(\rho'_j)\prod_{i\in I_j}\mathcal{N}(h_i^*, s_i^*|\mathbf{W}_i\boldsymbol{\delta}_j, \boldsymbol{\Omega_j}) \times t_\nu(\rho_j|\mu_j, V_j)}{\pi(\rho_j)\prod_{i\in I_j}\mathcal{N}(h_i^*, s_i^*|\mathbf{W}_i\boldsymbol{\delta}_j, \boldsymbol{\Omega}_j) \times t_\nu(\rho'_j|\mu_j, V_j)}\right\}$$

9.2 Prior Posterior Analysis

To formulate our prior assumptions we first follow standard practice and specify normal prior distributions for the slope parameters and the correlation coefficient. For the correlation coefficient the normal prior is restricted to the region $R = \{-1 < \rho_j < 1\}$ to ensure the positive definiteness of the correlation matrix $\boldsymbol{\Omega}_j$. Assuming that the parameters are a priori independent, the prior distribution of the coefficient vector, $\boldsymbol{\theta}$ is given by

$$\pi(\boldsymbol{\theta}) = \mathcal{N}_m(\boldsymbol{\alpha}|\boldsymbol{a}_0, \mathbf{A}_0)\, \mathcal{N}_q(\boldsymbol{\gamma}|\boldsymbol{g}_0, \mathbf{G}_0)\, \prod_{j=1}^{1} \mathcal{N}_p(\boldsymbol{\beta}_j|\boldsymbol{b}_{j0}, \mathbf{B}_{j0})\, \mathcal{N}(\rho_j|r_{jo}, R_{j0}) \times R.$$

The prior means are set based on a fitting of our model to the 2002 survey data using these prior distributions centered at zero (to express our apriori ignorance) and with a prior variance of 5 for the slope parameters and 1 for the correlation parameters. We fit the model described in the previous section to the 2002 data without the non-western and price variables for which we have insufficient information.

For our analysis of the 2006 data we then specify the prior means of for $(\boldsymbol{\beta}_0, \boldsymbol{\beta}_1, \boldsymbol{\alpha}, \boldsymbol{\gamma})$ in terms of the means of the posterior distribution based on the 2002 data as follows:

$$\begin{aligned}
\boldsymbol{b}_{00} &= (-0.11, -0.36, -1.04, 1.61, -0.28, 0, 0),\ \boldsymbol{b}_{10} = (0.66, -0.43, -1.40, 2.21, 1.0, 0, 0),\\
\boldsymbol{a}_0 &= (1.29, -1.78, 0.78, 0.18, 0.32, -0.24, 0.62, 0.44, 0, 0.10, 0.15),\\
\boldsymbol{g}_0 &= (-1.22, -0.35, 0.04, 0.08, 0.40, 0, 0, 0.64, 0.47, 0.48, -0.19, 0.06, 0.62, 0, 0.25, 0.14).
\end{aligned}$$

These prior means reflect our updated prior assumptions about the coefficients in the cannabis and hard drug models after seeing the 2002 data. For example, our specification of $\boldsymbol{b}_{00}$ and $\boldsymbol{b}_{10}$ suggest that accessibility factors affect hard drug uptake, and do so in a different way depending on previous cannabis use. The lower prior means for the seizure and prevalence variables for

subjects without previous cannabis use $(1.61, -0.28)$ compared to those with prior use $(2.21, 1.0)$ express a prior belief that previous cannabis affects hard drub uptake via transmission channels 2 and 3. The positive prior mean of 1.29 on the gateway coefficient assumes that previous cannabis use affect hard drub uptake directly (via transmission channels 1 and/or 4). Note that we have set the prior means of the non-western and price variables at zero since we cannot update our prior ignorance based on the 2002 data. To ensure that our prior assumptions can be updated based on the new data in the form of the likelihood, we formulate flexible priors around these specified means. In particular, we set the variances for all slope coefficient at 2. Based on the 2002 data analysis, we center the prior distributions of the correlation parameters (ρ_0, ρ_1) at 0.15 and 0 with a variance of 1.

9.3 Predictive Analysis

The marginal predictive probabilities of hard drug use $\mathsf{Pr}(h_{j,n+1} = 1|\mathbf{c}, \mathbf{h}, \mathbf{W})$, $j = 0, 1$ can be computed based on the predictive distributions of the latent hard drug intake. To generate draws from $p(h^*_{j,n+1}|\mathbf{h}, \mathbf{s}, \mathbf{W}, \mathbf{w}_{h,n+1})$ we exploit the fact that the conditional predictive distribution $p(h^*_{j,n+1}|\mathbf{h}, \mathbf{s}, \mathbf{W}, \mathbf{w}_{h,n+1}, \boldsymbol{\beta}_j, \boldsymbol{\alpha})$ is of a known form. To obtain the marginal predictive distribution we integrate numerically over the parameters using the corresponding posterior distribution and over $\mathbf{w}_{h,n+1}$ using the empirical distribution of the data. This can be done in a straight forward manner by extending the model fitting algorithm, that generates draws of the model parameters from the posterior distribution, with the following steps at each iteration g of the algorithm:

- Sample $\mathbf{w}^{(g)}_{1h,n+1}$ and $\mathbf{w}^{(g)}_{2h,n+1}$ from the full set of covariates
- Draw $h^{*(g)}_{n+1}$ from $\mathcal{N}(h^{*(g)}_{j,n+1}|\mathbf{w}^{g'}_{1h,i}\boldsymbol{\beta}^{(g)}_j + \mathbf{w}^{g'}_{2h,i}\boldsymbol{\alpha}^{(g)}, 1)$ where $\boldsymbol{\beta}^{(g)}_j$ and $\boldsymbol{\alpha}^{(g)}$ are the parameter values at the current g'th iteration of the MCMC algorithm
- Store values

The resulting draws $[h^{*^1}_{j,n+1}, h^{*^2}_{j,n+1}, ..., h^{*^M}_{j,n+1}]$ are from the marginal predictive distribution of latent hard drug intake. From these draws we can immediately compute the probabilities of hard drug intake as

$$\mathsf{Pr}(h_{j,n+1} = 1|\mathbf{c}, \mathbf{h}, \mathbf{W}) = \frac{1}{M}\sum_{g=1}^{M} I[h^{*(g)}_{j,n+1} > 0]$$

To compute the partial predictive probabilities without accessibility effects etc we use the same approach and set the corresponding elements in $\boldsymbol{\beta}_j$ or $\boldsymbol{\alpha}$ to zero when drawing $h^{*(g)}_{j,n+1}$ from $\mathcal{N}(h^{*(g)}_{j,n+1}|\mathbf{w}^{g'}_{h,i}\boldsymbol{\beta}^{(g)}_j + \mathbf{w}^{g'}_{2h,i}\boldsymbol{\alpha}^{(g)}, 1)$.

To compute the overall probability of hard drug use in Table 7, which is based on the conditional probability $\mathsf{Pr}(h_{n+1} = 1|c_{n+1}, \mathbf{c}, \mathbf{h}, \mathbf{W})$, we use the following steps:

- Sample $\mathbf{w}^{(g)}_{1h,n+1}$, $\mathbf{w}^{(g)}_{2h,n+1}$ and $\mathbf{w}^{(g)}_{c,n+1}$ from the full set of covariates
- Draw $c^{*(g)}_{n+1}$ from $\mathcal{N}(c^{*(g)}_{n+1}|\mathbf{w}^{g'}_{c,i}\boldsymbol{\gamma}^{(g)}, 1)$ and set $j = I[c^{*(g)}_{n+1} > 0]$.
- Draw $h^{*(g)}_{n+1}$ from $\mathcal{N}(h^{*(g)}_{j,n+1}|\mathbf{w}^{g'}_{h,i}\boldsymbol{\beta}^{(g)}_j + \mathbf{w}^{g'}_{2h,i}\boldsymbol{\alpha}^{(g)} - \rho_j(c^{*(g)}_{n+1} - \mathbf{w}^{g'}_{c,i}\boldsymbol{\gamma}^{(g)}), 1 - \rho^2_j)$
- Store values

The draws on $h^{(g)}_{n+1}$ are then used to compute $\mathsf{Pr}(h_{n+1} = 1|c_{n+1}, \mathbf{c}, \mathbf{h}, \mathbf{W})$.

10 Appendix B: Additional Data Summaries and Estimation Results

Variable	No Cannabis Use ($n = 3191$)	Cannabis Use ($n = 973$)
obtain amphetamine	0.31	0.62
obtain cocaine	0.23	0.57
obtain heroin	0.19	0.47
obtain ecstacy	0.29	0.48

Table 8: *Means of obtain indicators (1 indicates that subjects can obtain the drug within 3 days) for various hard drugs in the data by cannabis intake.*

User Definitions		No Cannabis Use	Cannabis Use only	Hard Drug Use
Cannabis	Hard Drugs			
$freq_c \geq 1$	$freq_h \geq 1$	2548	1045	571
$freq_c \geq 5$	$freq_h \geq 1$	3089	504	696
$freq_c \geq 5$	$freq_h \geq 5$	3160	661	343

Table 9: *Drug Use in the sample based on different frequency of use definitions. The second definition is the one used in the main empirical section, while the one in line 1 refers to Specification 1 in the sensitivity section and line 3 to Specification 2 in the sensitivity section.*

	Extended Gateway Model with ignorant priors		
	Hard uptake		Can uptake
	no can	can	
Country	0.03 (0.18)	0.37 (0.18)	-0.29 (0.10)
Oslo	0.58 (0.26)	-1.11 (0.46)	-0.02 (0.11)
Obtain	0.63 (0.98)	0.23 (1.06)	0.06 (0.71)
Seizure	-0.37 (0.64)	3.21 (1.11)	-
Prevalence	0.43 (1.30)	2.53 (1.97)	2.09 (0.90)
Non-Western	-0.31 (0.19)	0.48 (0.29)	-0.54 (0.13)
Amphetamine Price	0.36 (0.41)	0.04 (0.44)	-
Cannabis Price	-	-	-0.75 (1.38)
Cannabis Use	0.93 (0.54)		-
Police prob.	0.88 (0.22)		0.61 (0.19)
Parents prob.	0.46 (0.10)		0.42 (0.08)
School prob.	0.03 (0.13)		0.25 (0.11)
Friends prob.	-0.04 (0.16)		-0.09 (0.13)
Leave School early	0.51 (0.12)		0.18 (0.11)
Alcohol young	0.29 (0.11)		0.40 (0.09)
Smoker	0.44 (0.16)		1.17 (0.06)
Male	0.29 (0.07)		0.43 (0.05)
Age2124	-0.02 (0.10)		0.02 (0.06)
Intercept	-2.70 (0.47)		-2.16 (0.39)
Correlation	0.37 (0.24)	0.08 (0.21)	

Table 10: *Posterior means and standard deviations (parentheses) of the model parameters from the prior posterior analysis of the extended gateway model and for the restricted gateway model (selected results only). Results are based on the prior assumptions informed by the 2002 data as discussed in Section 4.2.*