THE ROAD TO RUIN? SEQUENCES OF INITIATION TO DRUG USE AND OFFENDING BY YOUNG PEOPLE IN BRITAIN *

Stephen Pudney
Public Sector Economics Research Centre
Department of Economics, University of Leicester

April 2001

ABSTRACT: The routes by which young people develop offending behaviour are very varied and strongly influenced by family background. A good understanding of the temporal sequences of first experiences of illicit drug use and other offending behaviour is needed before any plausible attempt can be made to investigate causal "gateway" effects. In this paper we develop and apply a statistical method for analysing the behavioural sequences observed in the 1998 Youth Lifestyles Survey.

KEYWORDS: illicit drugs, gateway effect, youth crime, random effects.

JEL CLASSIFICATION: I120, K420

ADDRESS FOR CORRESPONDENCE: Stephen Pudney, Department of Economics, University of Leicester, University Road, Leicester LE1 7RH, UK; e-mail: sep2@le.ac.uk.

* I am grateful to the Home Office for seedcorn funding for this research and for providing access to the Youth Lifestyles Survey data. All errors are mine, as are the views expressed here.

1 Introduction

The strongly rising trends in truancy, crime and illicit drug use by young people constitute one of the most important social developments of the post-war world. One of the most disturbing aspects of this development is the trend towards much earlier onset of these patterns of behaviour (see Stratford and Roth, 1999 and Flood-Page *et. al.,* 2000 for evidence on and discussion of this in the UK context). Developments in public policy have been influenced by these alarming trends. In Britain, the government has adopted a very ambitious target of reducing the availability and use by young people of certain types of drug use by 25% by 2005 and 50% by 2008 (UKADC, 2000). Effective anti-drugs policy may need to go beyond general targeting of this kind to much more specific action. If there is indeed a 'slippery slope' from early minor offending through soft drugs to hard drugs and serious crime, then we need to ask whether there are critical stages in this causal chain, against which policy is best directed.

However, all such policy initiatives are presently based on rather limited knowledge of the behaviour underlying these trends. It is no easy matter to study these issues. Illicit behaviour is inherently difficult to observe by means of conventional survey instruments. Particular problems include the possibility of misreporting by survey subjects and of non-response causally related to the behaviours in question. Even with suitable data, it is hard to resolve the dynamic causal structure underlying observed sequences of initiation to different types of offending and drug use because of the pervasive role of common unobservable psychological and social factors. Although there is a substantial research literature dealing with the dynamics of drug use and criminal activity at the individual level (see Flood-Page *et. al.* (2000) and Kenkel *et. al.* (2001) for surveys of this literature), few studies concentrate explicitly on the age of initiation into crime and drug use and on the sequences in which these initiation events occur. It is the aim of this paper to use recent British youth survey data to examine the pathways along which early drug/crime careers evolve.

2 A picture of drug use: the 1998 Youth Lifestyles Survey (YLS)

The 1998 YLS is an extended version of a youth survey first conducted in 1993. It covers the 12-30 age group, who were identified through one or other of two methods. A core sample of 3643 young people was identified from households participating in the 1998 British Crime Survey (BCS). This sample was then topped up by screening the occupants of addresses adjacent to those of the core sample to identify further subjects in the target age group. To ensure adequate coverage of high-crime areas, this top-up sample was deliberately biased towards areas identified by the BCS as having high victimisation rates. This over-sampling raised the coverage of high-crime areas from 27.5% in the core sample to 35.4% in the top-up sample.

Fieldwork took place between October 1998 and January 1999. Interviewing was subject to written consent from the parents of subjects aged under 16. Face-to-face computer assisted personal interviewing (CAPI) and computer assisted self interviewing (CASI) were used for different parts of the data gathering process, with CASI employed for the sensitive topics of drug use and criminal activity. The response rate was 69.1%, yielding a final usable sample of 3901 respondents.[1] Further detail on the design and conduct of the survey can be found in Stratford and Roth (1999) and Flood-Page *et. al.* (2000). The YLS questionnaire gives considerable detail on respondents' family circumstances, both currently and at age 15. Many aspects of experience at school are also recorded. Appendix Table A1 summarises the variables we use to describe individual characteristics and family background.

Our focus is on drug use and its relation to truancy and criminal activity. Drug use is here interpreted broadly to cover each of a set of 12 illicit substances together with consumption of alcohol and tobacco.[2] Importantly for our purposes, the questionnaire asks for the age at which each of these substances was first consumed. The basic sample characteristics of the age of

[1] Altogether there were 4848 respondents, but not all were interviewed about drug use and criminal activity.

[2] The YLS also contains questions about a non-existent drug "semeron", included to test response reliability and questions about anabolic steroids. The number of respondents claiming experience of semeron is very small and such cases have been dropped. Anabolic steroids have been excluded from our analysis because of the very low level of prevalence and the rather different use to which steroids are put.

onset for each category are summarised in Table 1. Three summary statistics are given for each drug: the sample percentage reporting any previous use; the mean age of first use for those who had used the drug; and the proportion of users who had begun before their 16th birthday. All of these statistics are weighted to be representative of the 12-30 age group in the population.

A clear pattern emerges from Table 1. The drugs with earliest onset, around age 14, are alcohol, tobacco and glue/solvents. Over three-quarters of the people who report experience of these substances commenced use before the age of 16. There is then a gap of around $2\frac{1}{2}$years before the mean age of first use of cannabis and amyl nitrite. A little later, at age 17-18, comes the first use of hard drugs (heroin and crack) and other substances (amphetamines, LSD, mushrooms, tranquilisers). The most "adult" drugs are methadone, ecstasy and finally cocaine, which has a mean age of first use of almost 20. It is dangerous to generalise about patterns of bahaviour, but there seems to be a natural division of drugs into five groups: (i) early onset legal substances (alcohol, tobacco); (ii) glue/solvents; (iii) early/middle onset soft drugs (amphetamines, cannabis, LSD, mushrooms, tranquilisers, amyl nitrite); (iv) early/middle onset hard drugs (heroin, crack, methadone); (v) late onset recreational drugs (ecstasy, cocaine).

Table 1 Age of first use by drug type

Event	**% prevalence**	**Mean age**	**% under 16**
Amphetamines	19.66	17.83	22.06
Cannabis	38.63	16.60	41.76
Cocaine *	7.49	20.22	7.40
Crack *	1.50	18.37	18.20
Ecstasy	9.47	18.87	13.35
Heroin *	1.18	17.51	25.21
LSD	11.19	17.23	29.03
Magic mushrooms	9.06	17.32	32.34
Methadone *	0.73	18.42	23.21
Tranquilisers	3.60	18.15	19.56
Amyl nitrite	15.72	16.89	33.47
Glue/solvents	7.84	14.12	83.17
Any drug	42.68	16.20	47.55
Any hard drug	8.11	19.86	11.18
Alcohol	90.17	13.75	76.84
Tobacco	71.40	14.01	76.41
Truancy	32.09	13.78	90.56
Minor crime	43.41	14.46	69.01
Serious crime	9.36	14.52	65.63

* denotes substances treated as hard drugs

Crime is represented by participation in either of two groups of offences. The first is a group of 18 "minor" offences (criminal damage, arson, theft, dealing in stolen goods, cheque and credit card offences, fraud and public fighting) and 9 "serious" crimes (theft of vehicles, robbery, breaking and entering and assault). The full set of 27 offences identified by the YLS is given in Flood-Page *et. al.* (2000, appendix B). There is some evidence of a progression from truancy to minor crime to serious crime. This progression tends to occur early relative to most drug use.

The sequencing of drug use events within the larger process of offending and truancy behaviour is summarised in Table 2, which gives weighted sample frequencies of the logically possible event sequences. The two columns of Table 2 correspond to two alternative definitions of crime and drug use: the

first covers all drugs (excluding alcohol and tobacco) and all crime; the second covers only hard drugs (cocaine, crack, heroin, methadone) and serious crime.

This simple tabulation exercise is revealing. There is a clear tendency towards a chain of events beginning with petty crime and truancy, and only later developing into drug use. Sequences of offending beginning with drug use have a significantly smaller sample frequency than sequences beginning with truancy or crime, and this is particularly true when we consider only hard drugs and serious crime. If we were prepared to assume that this tendency has causal significance then we might conclude that a policy addressing truancy and other problems at school might be more effective than a policy attacking drug use directly. We now examine this issue in more detail by estimating conditional models of drug use and offending behaviour.

Table 2 Sequences of illicit behaviour

Sequence	% frequency (all crimes and drugs)	% frequency (serious crimes and hard drugs)
No offending or drug use	34.86	61.76
Truancy only	6.33	22.96
Crime only	11.55	3.24
Drugs only	8.83	2.57
Truancy→drugs	4.65	3.33
Truancy→crime	2.76	3.08
Crime→drugs	8.82	0.14
Crime→truancy	2.48	1.57
Drugs→truancy	1.67	0.10
Drugs→crime	5.85	0.20
Truancy→crime→drugs	5.48	1.50
Truancy→drugs→crime	5.06	0.53
Crime→truancy→drugs	6.39	0.49
Drugs→crime→truancy	1.84	0.14
Drugs→truancy→crime	2.49	0.13
Crime→drugs→truancy	3.38	0.11
Drugs→other offences	11.85	0.57
Crime→other offences	21.07	2.31
Truancy→other offences	17.95	8.44

Note: tied events are double-counted; alcohol and cigarettes are not included in drug use

3 Availability, demonstration and price effects on demand

3.1 Social externalities

Economists tend to emphasise individual decision-making in isolation from the social context. The theory of rational addiction (Becker and Murphy, 1988; Grossman and Chaloupka, 1998; Kenkel, Mathios and Pacula, 2001) is an example of this: drug users are seen as rational, forward-looking individuals pursuing a planned course of action that takes full account of possible

future consequences of current actions. It is very easy to ridicule an approach like this when applied to a pattern of behaviour that often involves severe distress and a departure from normal psychological and social functioning. Nevertheless, the core idea of rational individual choice is an important one that has a place in the study of drug use. Equally, it is also important to take account of social externalities (corresponding to the idea of a 'drug culture'). Manski (2000) gives a good survey of the importance of and analytical difficulties introduced by these social interactions.

To illustrate the impact of these external influences on an economic model of drug use, consider the following generic demand model:

$$\delta = \delta(p, u, \varphi\Lambda(p, u, \varphi)) \tag{1}$$

where δ is the individual's demand for the illicit drug, p is its price, u is a variable distinguishing the different types of individual in the population, Λ is a variable representing the external effects influencing individuals of type u and φ is a non-negative parameter introduced to represent responsiveness to these influences. The function δ has partial derivatives with respect to p and φ which are negative and positive respectively. The variable u has a population distribution $G(u)$ which could be multivariate; u may include observable elements, such as income and location, and also unobservable psychological characteristics, such as risk aversion or ability.

The phenomena represented by Λ include social externalities (demonstration effects, peer pressure, etc.) and local availability through the medium of drug-using social contacts. The externality function Λ is defined as follows:

$$\Lambda(p, u, \varphi) = \int \delta(p, v, \varphi\Lambda(p, v, \varphi))\theta(u, v)dG(v) \tag{2}$$

where $\theta(u, v)$ is some non-negative measure of social distance or of the influence exerted by someone of type v on someone of type u. Note that, in Manski's (1993, 2000) terminology, (1) and (2) embody the notion of endogenous interactions, where each person's behaviour depends on that of the others. Contextual and correlated effects can also be captured in this framework through the variable u which can represent similar exogenous characteristics and common factors operating within reference groups.

A social demand equilibrium is then a pair of functions $\delta(.)$, $\Lambda(.)$ satisfying the equations (1)-(2). Aggregate demand is:

$$D(p, \varphi) = \int \delta(p, u, \varphi\Lambda(p, u, \varphi))dG(u) \tag{3}$$

Let the aggregate supply function be $S(p,t)$ where t represents an autonomous expansion in supply. Market equilibrium implies $D(p,\varphi) = S(p,t)$. The partial derivatives of supply are $S_p > 0$ and $S_t > 0$. Demand is more complex. Omitting unnecessary arguments and using subscripts to denote partial derivatives:

$$D_p = \int \left[\delta_p(u) + \varphi\delta_{\varphi\Lambda}(u)\Lambda_p(u)\right] dG(u) \tag{4}$$

$$D_\varphi = \int \left[\delta_{\varphi\Lambda}(u) + \delta_{\varphi\Lambda}(u)\left(\Lambda(u) + \varphi\Lambda_\varphi(u)\right)\right] dG(u) \tag{5}$$

where

$$\Lambda_p(u) = \int \left[\delta_p(v) + \varphi\delta_{\varphi\Lambda}(v)\Lambda_p(v\right] \theta(u,v) dG(v)$$

$$\Lambda_\varphi(u) = \int \left[\delta_{\varphi\Lambda}(v)\left(\Lambda(v) + \varphi\Lambda_\varphi(v)\right)\right] \theta(u,v) dG(v)$$

Make the reasonable assumption that individual demands are downward-sloping after allowing for external effects. Then $\Lambda_p(v) < 0$ for all v and thus $D_p < 0$. Similarly, make the reasonable assumption that increasing the strength of the demonstration effect always has a positive impact on individual demands, so that $\Lambda_\varphi(v) > 0$ for all v and thus $D_\varphi > 0$. Note that, from (4), the effect of the social externality is to increase the price elasticity of demand. In essence, what happens is that social externalities amplify the effect of price changes: as price falls, there is a direct increase in the individual's demand, but the consequent general increase in consumption also strengthens the demonstration effect, which stimulates individual demand still further. This process continues until a new equilibrium is reached.

The effect of an expansion in supply is therefore to reduce the fall in price that would otherwise occur ($p^{2\varphi} - p^1$ rather than $p^{20} - p^1$) and to increase the equilibrium level of consumption ($Q^{2\varphi} - Q^1$ rather than $Q^{20} - Q^1$). This is illustrated in figure 1, where S^1 and S^2 are the supply curves before and after the supply change; D^0 is the demand curve with no externality effects (i.e. $\varphi = 0$) and D^φ is the full demand curve with $\varphi > 0$. The important point here is that if social interactions do exist, then they have potentially important consequences for the economics of illicit drugs markets and should be incorporated in attempts at modelling.

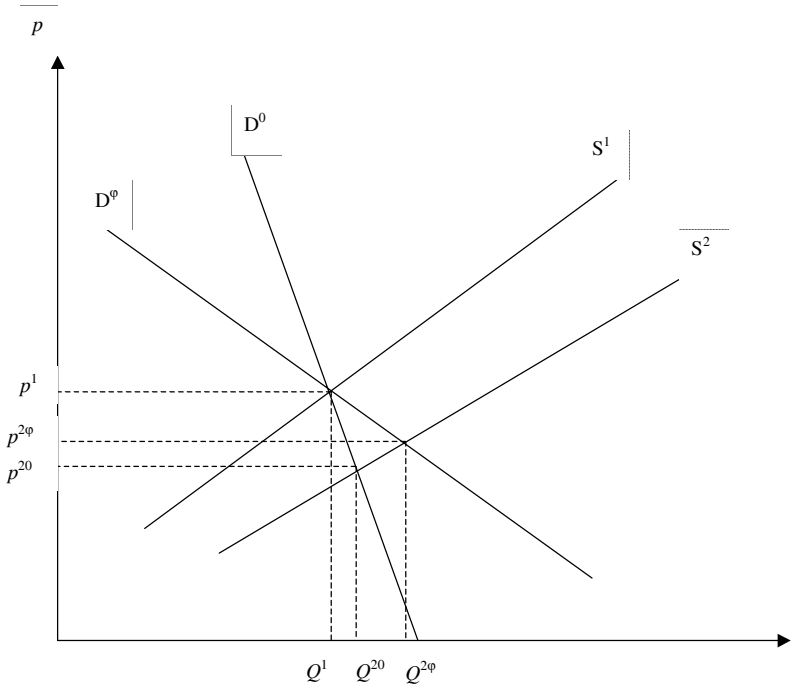


Figure 1 The impact of consumption externalities on market response to a supply shift

There are serious identification issues to be overcome in modelling the impact of social externalities: referred to by Manski (1993) as the reflection problem. In simple terms, since group norms are defined as averages of individual outcomes, it is impossible to distinguish the effect of that average from the other factors underpinning those outcomes. In dynamic models where there is a lag in the formation of group norms, the set of possible observable outcomes is richer and the identification problem is resolved (Manski, 1993; Brock and Durlauf, 2000). This dynamic setting is inherent to the behaviour studied here. We are concerned with the timing of first use by individuals and thus any demonstration effect influencing the drug 'novices' in our sample must by definition be the result of past behaviour of others.[3] We do not pursue this identification issue formally, but rely on the dynamics of the drug use process to justify the approach used here.

[3]There are further issues introduced by time aggregation which we do not pursue here.

It is difficult to capture empirically the influence of social externalities without having detailed information on the behaviour of subjects' social contacts. Instead, we use a macro-level proxy for the external influences acting on the individual at the time when s/he is of age t:

$$\Lambda_t(p_t, u, \varphi) \approx f(z, A_t) \tag{6}$$

where z is a set of observable variables governing the individual's social location and A_t is a macro-level index of drug use in society at large at the time that the individual is at age t. Suitable indices of prevalence have been constructed by Pudney (2001) for a subset of the drugs considered here, using a multiple-indicator latent variable approach based on time-series data on: drug seizures; numbers of new addicts; numbers of drug-related convictions; and BCS prevalence rates. These indicators are plotted in Figure 2 and in some cases (notably amphetamines, LSD and cannabis) follow a path that would be difficult to capture using simple time trends. In the econometric modelling discussed later, the relevant prevalence index is used in logarithmic form.[4]

[4]For crack and ecstasy, we assume a prevalence of 0.5% of the 1995 level for the period prior to 1989, during which the recorded indicators of drug use were non-existent or too low to permit a positive estimate of prevalence.

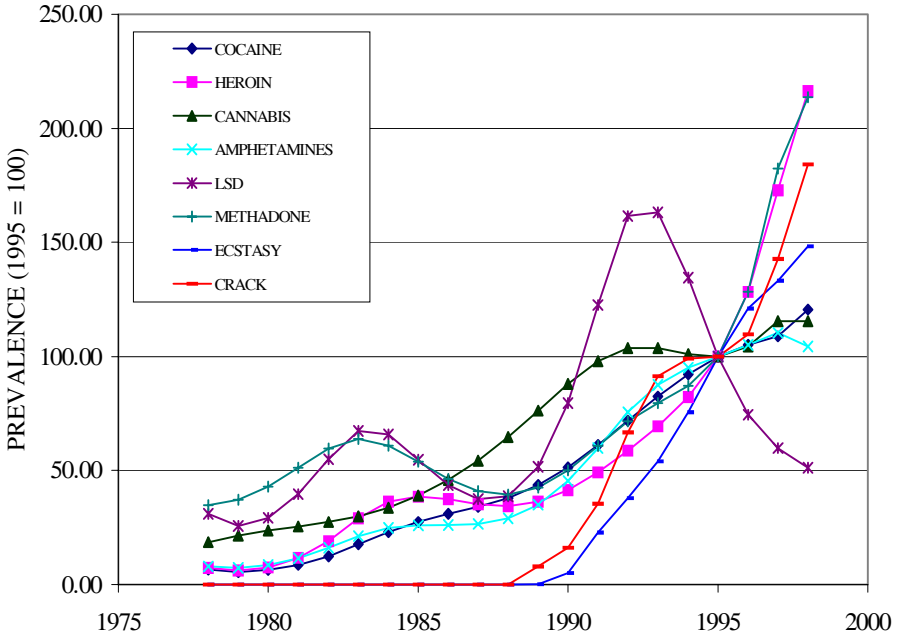


Figure 2 Indices of prevalence by drug type (source: Pudney, 2001)

3.2 Prices and supply constraints

Drugs are goods like any other and it is likely that drug use is responsive to price variations. However, price effects raise difficult empirical problems. The available data on street prices of illicit drugs are sparse and not very reliable. They fall far short of the quality of a conventional price index and are only available in anything like a consistent form for the period since 1988. The main source of information is the UK National Criminal Intelligence Service NCIS), which provides rough ranges of typical street prices in a few particular locations. Figure 3 plots these price series in real terms for the London drug market.[5] There are two major problems with these price data for our purposes. Firstly, to incorporate price effects explicitly, we would require a sequence of past prices covering the relevant past of people aged up to 30 in 1998. This would involve price series going back to perhaps

[5]To construct Figure 3, we have taken the mid points of quoted price ranges and deflated by the Retail Price Index.

1978 but only half of that period is available. Secondly, given the inherent unreliability of price data, it is not reasonable to infer more from Figure 3 than that there has been a steady downward trend in the real price of the major illicit drugs over the 1978-98 period of something like 3% per year. It would be rash to attribute much significance to the large year-to-year swings around this common trend.

A further issue is supply constraints. Drugs are illicit commodities which are not routinely available in the same way as other commodities. It is very likely that many individuals in the YLS sample will have been supply constrained for significant periods. This is particularly important in the early part of their drug use careers, which are are the focus of our study. Given the incomplete and unreliable price data and the unobserved but probably widespread quantity constraints on demand, there is little point in attempting a standard type of demand analysis with explicit use of price variables. Instead, we rely on the constructed prevalence indices to act as proxies for consumption externalities, availability and also price movements.

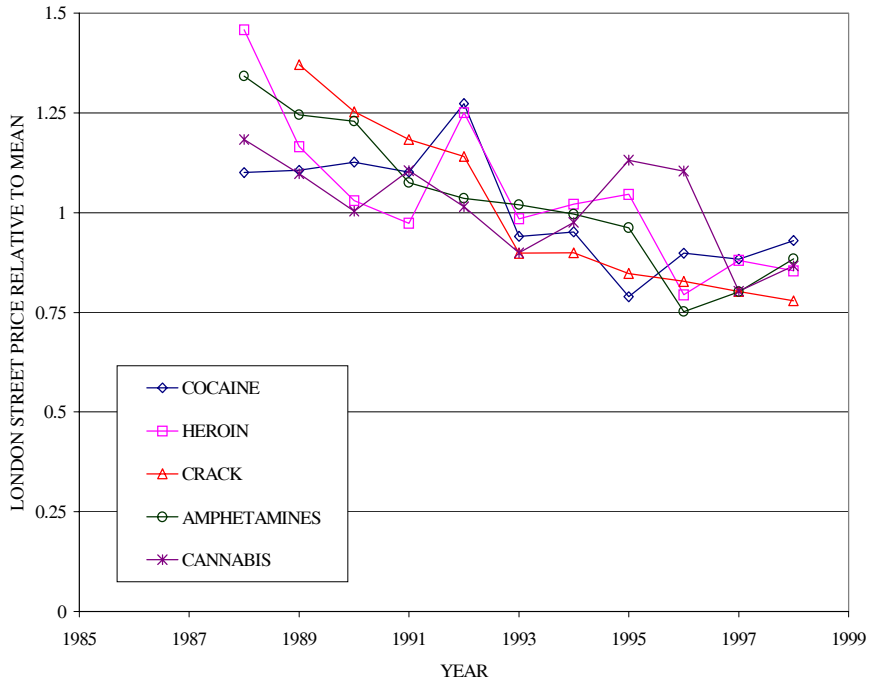


Figure 3 Real London street prices for illicit drugs
(source: National Criminal Intelligence Service)

4 Sequential modelling

The analysis used here is essentially survival analysis. In that sense, it is comparable with the work of Fergusson and Horwood (2000) who have used prior experience of cannabis as an explanatory factor in a proportional hazards duration model of the age of onset of use of other illicit drugs. However, there are some complicating features. Firstly, the risk of onset of drug use and other types of offending varies greatly over age, in a non-monotonic fashion. Many of the widely-used parametric survival models, such as the Weibull used by Fergusson and Horwood may therefore be inappropriate. Secondly, age of onset is recorded only as an integer, with the consequence that there are very large numbers of 'tied' durations, causing difficulties for the semi-parametric Cox regression model. Thirdly, the rapidly-changing prevalence of drug use introduces time-varying covariates linked to calendar time rather

than age. Fourthly, there is a possibility of dynamic causal feedback from one type of offending to another. Finally, there may be persistent unobservable individual-specific effects which complicate the problem of inferring causal processes from observed drug use/offending histories. The more ambitious model of van Ours (2000), dealing with the dynamic interrelation between alcohol, tobacco, cannabis and cocaine, deals with the functional form and heterogeneity issues in a continuous-time duration model. Our approach is a little different. We analyse a wider range of behaviours, including crime as well as drug use. We also consider only the timing of onset and leave aside the issue of exit from these behavioural patterns.[6] A third, more minor difference, is that it is slightly more convenient with the YLS data to use a discrete time approach.

For any given individual, consider an observation period that covers the years from some initial age T_0 to the current observed age T_1. Let there be J different types of first-occurrence events. These events are the first use of each of the set of different drugs, the first episode of truancy and the first criminal offence of two types: minor and serious. Denote the ages at which these events occur by $\tau_1 ... \tau_J$. If event j is not observed within the observation period, then τ_j is censored at the arbitrary value $T_1 + 1$.

4.1 Single-equation modelling

Consider first the case of a single event type j, analysed in isolation. The analysis is conditioned on all other aspects of the individual's history and thus implicitly adopts a very simple view of causality. Define the hazard rate at age t for event j as the probability that event j occurs at age t conditional on no occurrence of the event prior to t. This probability is also conditional on the past history of the $J-1$ other event types. Let $\mathbf{x}_{jt}$ be a vector of explanatory covariates relevant to event j at time t. The vector $\mathbf{x}_{jt}$ will in general contain variables describing aspects of the individual's history relevant to event j and also the proxy for availability, A_t. We model the hazard rate as a conventional probit structure:

$$\Pr(\text{event } j \text{ occurs at age } t \mid \text{history}) = \Phi(\mathbf{x}_{jt}\boldsymbol{\beta}_j) \tag{7}$$

[6] It is very difficulty both conceptually, and within the YLS questionnaire structure, to define a date of exit from crime or drug use. For many people, these are infrequent activities and there is no obvious date at which they can be said to have been "given up".

where $\Phi(.)$ is the cdf of the N(0,1) distribution.

The case of independent random effects can be dealt with on a single-equation basis using standard software. The log-likelihood function for equation j of this model is:

$$L(\boldsymbol{\beta}_j) = \sum_{i=1}^{n} \ln \left\{ \Phi(\mathbf{x}_{j\tau_j} \boldsymbol{\beta}_j)^{d_{ij}} \prod_{t=T_0}^{\tau_{ij}-d_{ij}} \left[1 - \Phi(\mathbf{x}_{jt}\boldsymbol{\beta}_j)\right] \right\} \tag{8}$$

where d_{ij} is a binary indicator for uncensored observations such that $d_{ij} = 1$ if $\tau_{ij} \leq T_1$ and 0 if $\tau_{ij} > T_1$. Note that (8) is the standard log-likelihood function for a probit model, estimated from a set of $N = \sum_i (\tau_{ij} - T_0 + 1)$ observations. We include the relevant (log) prevalence variable as a covariate in the models for cannabis, amphetamines, ecstasy, LSD, cocaine, crack, heroin and methadone. For the remaining six substances and for truancy and crime, we use a quadratic time trend to approximate the effect of changing conditions over time. In every case where it is available, the use of the prevalence variable resulted in a better fit that the time trend. Full results for this model are given in Appendix Tables A1-A2.[7]

Note that the YLS is not a full longitudinal survey, so characteristics which summarise family background, and which are potentially variable over time, are only observable at one point in time. This reference period is defined as the time of the respondent's fifteenth year of age or the time of the survey, whichever is the earlier. These variables record whether or not the mother or father was absent from the family and also the employment status of each parent. Other variables describing the neighbourhood (inner city and/or socially deprived); any family history of trouble with the police; and any religious affiliation, are observable only at the time of interview. As one might expect, females and those claiming some religious activity tend to have a lower risk of drug use and offending. Contrary to some popular stereotypes, whenever estimated ethnic differentials are significant, blacks and Asians are found to have lower rates of offending.

Table 3 summarises the pattern of estimated lagged responses in schematic form. It is striking that the group of 'minor' vices (tobacco, alcohol, cannabis,

[7]The single-equation results were computed using STATA 7.0; the quoted standard errors are calculated using robust formulae that take account of the clustering of years within individuals. Attempts to allow for Gaussian random effects within these single equation models were unsuccessful, since the random effects variances were estimated at zero in each case.

truancy and minor crime) tend to be associated with subsequent engagement in 'middle level' vices (amphetamines, ecstasy, LSD, magic mushrooms, tranquilisers, amyl nitrite) and to a lesser extent *vice versa*. However, there is little evidence of a direct link between minor vices and the most serious ones (cocaine, crack, heroin, methadone and serious crime), with the exception of a strong tendency for serious crime to be preceded by alcohol use, truancy and minor crime and a link between cannabis and cocaine. The middle-rank drugs are more strongly linked to subsequent use of hard drugs. Note that, in terms of the similarity of the pattern of responses, there is a strong case for regarding amphetamines, LSD, magic mushrooms, amyl nitrite and (arguably) tranquilisers as 'soft' drugs similar to cannabis.

Table 3 The sign pattern of significant lagged responses (95% significance level)

	Impact on occurrence probability of ...																	
	Tob	Alc	Glu	Can	Tru	Min	Amp	Ecs	Lsd	Mus	Tra	Amy	Coc	Cra	Her	Met	Ser
Tob		+	+	+	+	+	+	+	+	+		+		+			
Alc	+		+	+		+	+	+	+	+	+	+	+				+
Glu				+			+		+			+					
Can	+	+			+	+	+	+	+	+	+	+	+				
Tru			+	+		+	+	+	+	+							+
Min	+	+	+	+	+		+		+		+	+	+				+
Amp						+		+	+	+	+	+	+	+			+
Ecs			-						+	-	+		+		+		
LSD							+	+		+			+			+	
Mus							+										
Tra		-	+			+								+	+		
Amy				+			+	+	+	+							
Coc			+											+			
Cra															+		
Her					+						+			+		+	
Met							+	-									
Ser			+			+	+										

5 Joint estimation

The results summarised in Table 3 suggest the possibility of extensive dynamic links between types of illicit behaviour. It leaves open the possibility of elaborate causal chains going from smoking, drinking, truancy, etc. to soft drug use and on to hard drugs and serious crime. However, these links may have no causal significance and might stem from the common effect of unobserved psychological and sociological characteristics. If an individual is predisposed towards illicit behaviours by some personal characteristic, then there may be a tendency to observe simultaneous involvement in truancy, crime and drug use even without any direct causal connection between them. The unobservable characteristics underlying these spurious associations might include such features as a disturbed family background, an under-developed ability to appreciate the long-term consequences of current actions, or low ability leading to under-achievement and alienation. Modelling the effect of unobservable characteristics is inherently difficult and can only generate clear results under strong assumptions. Nevertheless, it is worth attempting since the results can give a good indication of the potential importance of unobservable factors.

We assume throughout that the occurrences of events $1...J$ are contemporaneously independent conditional on $\{\mathbf{x}_{jt}, u_j\}$. However, this still permits considerable dependence through lagged effects embodied in $\mathbf{x}_{jt}$ and through correlation in the joint distribution of $u_1...u_J$. The probability of the observed joint event $(\tau_1...\tau_J)$ is:

$$\Pr(\tau_1...\tau_J|\mathbf{X}) = \int \mu_i(u_j) dG(\mathbf{u}) \tag{9}$$

where $\mu_i(\mathbf{u})$ is the conditional probability $\Pr(\tau_1...\tau_J|\mathbf{X}, \mathbf{u})$:

$$\mu(\mathbf{u}) = \prod_{j=1}^{J} \prod_{t=1}^{\tau_j - d_j} \left[1 - \Phi(\mathbf{x}_{jt}\boldsymbol{\beta}_j + u_j)\right] \Phi(\mathbf{x}_{j\tau_j}\boldsymbol{\beta}_j + u_j)^{d_j} \tag{10}$$

where $\mathbf{X} = \{\mathbf{x}_{jt}\,,\ j = 1...J;\ t = 1...T\}$. We allow the random effects $u_1...u_J$ to have different variances and to be cross-correlated. We permit this by expressing the u_j as linear combinations of a set of underlying independent standardised variates as follows:

$$\mathbf{u} = \mathbf{R}\boldsymbol{\varepsilon} \tag{11}$$

where $\mathbf{R}$ is a $J \times J$ loading matrix which is subject to a set of $J(J-1)/2$ normalising restrictions. We normalise $\mathbf{R}$ to be a lower-triangular matrix, which is equivalent to working with the Choleski decomposition of the covariance matrix of the random vector $\mathbf{u}$.[8]

The parameters are estimated by maximising the following objective function, which is based on a second-order expansion of the log of the simulated likelihood function.[9]

$$\ln L = \sum_{i=1}^{n} \left\{ \ln(\overline{\mu}_i) + \frac{1}{2Q} \frac{s_i^2}{\overline{\mu}_i^2} \right\} \tag{12}$$

where $\overline{\mu}_i$ and s_i^2 are the mean and variance across replications of the ith likelihood element:

$$\overline{\mu}_i = \frac{1}{Q} \sum_{q=1}^{Q} \frac{(\mu_i(\boldsymbol{\varepsilon}_q) + \mu_i(-\boldsymbol{\varepsilon}_q))}{2}$$

$$s_i^2 = \frac{1}{Q} \sum_{q=1}^{Q} \left[\frac{(\mu_i(\boldsymbol{\varepsilon}_q) + \mu_i(-\boldsymbol{\varepsilon}_q))}{2} - \overline{\mu}_i \right]^2$$

where $\mu_i(\boldsymbol{\varepsilon}_q) = \Pr(\tau_{i1} ... \tau_{iJ} | \mathbf{X}_i, \boldsymbol{\varepsilon}_q)$, Q is the number of Monte Carlo replications used and $\boldsymbol{\varepsilon}_q$ is a vector of independent pseudo-random variates drawn from the assumed standard normal distribution for $\boldsymbol{\varepsilon}$. This SML estimator is consistent and asymptotically normal with covariance matrix given by the usual inverse Hessian expression provided Q goes to infinity at least as fast as n. We use $Q = 50$ replications in our calculations, which experience with similar models suggests is adequate to make SML approximate true ML very closely even without the second-order bias correction (see Mealli and Pudney, 1996, for an example of this).

Joint modelling of this kind encounters the curse of dimensionality. As we increase the number J of types of event, the number of possible interactions

[8] We do not give a formal analysis of the identifiability of this model. However, the theoretical results of Abbring and van den Berg (2000) indicate that the identification of endogenous treatment effects is considerably less problematic in a duration setting than in the usual 2-period discrete setting. In general, in their bivariate framework, identification is achievable without the exclusion restrictions required in the conventional selection models.

[9] See Gouriéroux and Monfort 1996, page 45, but note the minor error in their equation 3.4.

between them increase in proportion to J^2. Estimation of a model with as many as eleven different drugs/offences is infeasible, so we now work with a simpler structure in which the categories are condensed into six. Even so, the full 6-factor model would involve 156 parameters. The construction of the aggregate offending categories is based on the information on age of onset given in Table 1 and the single equation results summarised in Table 4. In particular, we distinguish between solvents and other soft drugs because of the early age of onset for the former and thus its potentially important role in initiation to drug use. Ecstasy and cocaine are included in a single category because of their relatively high age of onset and their role as 'social' drugs. This approach views cocaine as a drug with a much more socially acceptable image than heroin and crack. The six categories finally specified are: (i) *glue/solvent abuse*; (ii) *soft drug use* (cannabis, amphetamines, LSD, magic mushrooms, amyl nitrite); (iii) *'social' drug use* (ecstasy or cocaine); (iv) *hard drug use* (heroin, crack, methadone); (v) *minor offending* (truancy, criminal damage, arson, theft, dealing in stolen goods, cheque and credit card offences, fraud and public fighting); (vi) *serious crime* (theft of vehicles, robbery, breaking and entering and assault).

The SML estimator for this model is computationally demanding. Our strategy is to begin with the simplest 1-factor model in which $\boldsymbol{\varepsilon}$ contains a single random factor and the matrix $\mathbf{R}$ is a column vector. Then a sequence of generalised models is estimated, with the number of random factors in $\boldsymbol{\varepsilon}$ increased by 1 at each step. This process is terminated when the addition of an extra factor leads to an insignificant improvement according to a simulated likelihood ratio criterion. In practice, the 1-factor model was preferred to the 2-factor by this criterion and the random effects estimates discussed below correspond to the 1-factor specification. For this case, a comparison of the likelihood values computed at a representative point confirmed that our simulation approach delivers numerical accuracy comparable with the Gauss-Legendre quadrature used by Butler and Moffitt (1982) for an analogous multinomial probit model.[10]

The full parameter estimates are given in appendix Table A3. We interpret these results in detail in the next two sections, but it is worth noting

[10]If we accept the result of 40-point quadrature as fully accurate, the simulation approach with $Q = 50$ gives a roughly similar degree of accuracy to 20-point quadrature. Standard statistical software often uses quadrature based on as few as 12 points.

immediately that the estimated effects of past behaviour on the hazards of drug onset are generally smaller than in the non-heterogeneous model. In particular, there is no significant impact of early soft drug use on the hazard for hard drugs (crack, heroin, methadone), although there is still a significant impact of soft drugs on the hazard for truancy and minor crime. In contrast, the estimated impact of social and family background and of general drug prevalence is stronger in the random effects model.

5.1 The impact of personal characteristics

To illustrate the implications of the model, we use stochastic simulation to summarise the estimated effects of personal characteristics on behaviour. Table 4 is based on the non-heterogeneous model in which $\mathbf{u}$ is restricted to be $\mathbf{0}$ during estimation. Tables 5 and 6 are based on the random effects model estimated by maximising (12) numerically. Table 5 simulates the model for a hypothetical class of individuals with zero individual effects, while the simulations in Table 6 allow the values of $\mathbf{u}$ to vary randomly across replications, thus representing a cross-section of individuals. The stochastic simulations are done by generating 50,000 sets of 6×18 pseudo-random numbers for the six offending categories and 18 years (from age 12 to 29). These are then used to generate 50,000 histories for each of five different hypothetical individuals. The random effects $\mathbf{u}$ are either held fixed at their mean value $\mathbf{0}$ (Tables 4 and 5) or are sampled from the distribution $G(\mathbf{u}) = N(\mathbf{0}, \mathbf{R}\mathbf{R}')$ (Table 6). The baseline individual is a white male with a favourable family and social background (both parents present and in work, no family history of trouble with police, resident in non-deprived non-inner-city area) and living through a stable period with low prevalence of drug use (10% soft drugs; 0.5% ecstasy/cocaine; 0.1% hard drugs). The other four hypothetical individuals are simple deviations from this base: (i) a disadvantaged background (absent non-working father, working mother, family history of trouble with police, resident in deprived inner-city area); (ii) female; (iii) Asian; (iv) High prevalence of drug use (50% of the population having ever used soft drugs, 8% ecstasy/cocaine and 2% hard drugs). For the baseline, Table 4 gives the proportion ($\widehat{P}_j$) of the replications yielding experience of drug or offence j and the average age of onset ($\overline{\tau}_j$) in those cases. For the other four cases, the figures quoted are the difference in prevalence and average age with respect to the baseline. A disadvantaged social/family background is clearly

the dominant influence on drug use and offending, with general drug culture (as measured by prevalence) also extremely important. With the exception of a small but statistically significant rise in the hazard rate for serious crime for blacks, the influence of gender and ethnicity is to reduce the incidence of drug use and offending in comparison with the baseline white male group. In the case of ethnicity, these estimates are based on small sample numbers and therefore possibly not very robust, but they suggest that common racial stereotypes of drug users are seriously in error.

Table 4 Predicted % prevalence ($\widehat{P}_j$) and mean age of onset ($\overline{\tau}_j$) for baseline individual and differences ($\Delta\widehat{P}_j$, $\Delta\overline{\tau}_j$) relative to the baseline for other individual types (model without random effects; 50,000 replications)

		Solv.	Soft	C & E	Hard	Minor	Serious
Baseline	$\widehat{P}_j$	4.6	46.2	13.5	1.6	68.6	9.2
white male	$\overline{\tau}_j$	14.4	17.0	19.8	19.0	14.6	15.2
Disadvant-	$\Delta\widehat{P}_j$	+26.1	+48.4	+28.6	+25.3	+31.1	+55.3
aged	$\Delta\overline{\tau}_j$	+0.4	-1.6	-0.5	+0.1	-2.1	+0.1
Female	$\Delta\widehat{P}_j$	-1.7	-14.0	-8.1	-0.9	-16.7	-7.5
	$\Delta\overline{\tau}_j$	-0.2	+0.1	-0.1	-1.0	+0.2	-0.5
Asian	$\Delta\widehat{P}_j$	-2.2	-34.0	-10.7	+0.5	-20.7	-4.1
	$\Delta\overline{\tau}_j$	-0.3	+0.3	-0.4	-2.0	+0.0	-0.7
Black	$\Delta\widehat{P}_j$	-3.7	-19.2	-8.5	-1.2	-2.2	4.8
	$\Delta\overline{\tau}_j$	-0.0	+0.2	-0.0	-1.0	-0.1	-0.3
High	$\Delta\widehat{P}_j$	+10.6	+49.0	+43.5	+34.9	+19.2	+22.8
prevalence	$\Delta\overline{\tau}_j$	+0.5	-1.8	-0.8	-0.0	-0.1	+0.9

If the simulation results in Table 4 can be given a causal interpretation, they lend strong support to indirect policy directed initially at reducing social exclusion and disadvantage. However, true causation is virtually impossible to establish in this non-experimental setting and the best we can do is to explore the degree of robustness of these results to changes in specification

designed to allow for possible non-causal association. The simulations presented in Tables 5 and 6 are based on the estimated random effects model and thus make some allowance for unobservable factors that might be responsible for a predisposition of some individuals towards patterns of drug use and offending. The prevalence of drug use and offending among the simulated individuals is generally much higher in Table 6 (which allows for random variation in $\mathbf{u}$) than in Table 5 (which holds $\mathbf{u}$ fixed at $\mathbf{0}$). This underlines the potential importance of these factors and the consequent difficulty of drawing causal inferences. Comparing the results from the heterogeneous model (Table 6) with the non-heterogeneous model (Table 4), there does appear to be a fair degree of robustness. Both Tables give a broadly similar picture of the influence of social, family and personal characteristics on the drugs/crime hazard, but there are important differences of detail. Most noticeably, the influence of social disadvantage and general prevalence on the hazard rates for harder drugs (cocaine, ecstasy, heroin, crack, methadone) are rather larger in the random effects model.

Table 5 Predicted % prevalence ($\widehat{P}_j$) and mean age of onset ($\overline{\tau}_j$) for baseline individual and differences ($\Delta\widehat{P}_j$, $\Delta\overline{\tau}_j$) relative to the baseline for other individual types (random effects model; $\mathbf{u} = \mathbf{0}$; 50,000 replications)

		Solv.	Soft	C & E	Hard	Minor	Serious
Baseline	$\widehat{P}_j$	1.2	36.6	1.7	0.0	67.1	5.5
white male	$\overline{\tau}_j$	14.7	18.2	20.6	16.7	14.8	15.2
Disadvant-	$\Delta\widehat{P}_j$	+25.5	+63.3	+62.4	+13.9	+32.9	+60.9
aged	$\Delta\overline{\tau}_j$	-0.3	-3.6	-1.4	+2.5	-2.7	-0.4
Female	$\Delta\widehat{P}_j$	-0.6	-16.9	-1.5	-0.0	-19.1	-4.8
	$\Delta\overline{\tau}_j$	-0.2	+0.1	-0.2	+0.3	+0.2	-0.6
Asian	$\Delta\widehat{P}_j$	-0.7	-33.4	-1.7	+0.0	-23.4	-3.3
	$\Delta\overline{\tau}_j$	-0.1	+0.1	-0.8	+0.6	+0.2	-0.5
Black	$\Delta\widehat{P}_j$	-1.0	-21.7	-1.6	-0.0	-1.7	+2.8
	$\Delta\overline{\tau}_j$	+0.1	+0.2	-0.7	-16.7	-0.0	+0.1
High	$\Delta\widehat{P}_j$	+1.4	+60.6	+17.0	+3.8	+13.5	+11.2
prevalence	$\Delta\overline{\tau}_j$	-0.1	-2.2	-0.3	+2.1	-0.2	+0.2

Table 6 Predicted % prevalence ($\widehat{P}_j$) and mean age of onset ($\overline{\tau}_j$) for baseline individual and differences ($\Delta\widehat{P}_j$, $\Delta\overline{\tau}_j$) relative to the baseline for other individual types (random effects model; $\mathbf{u} \sim N(\mathbf{0}, \mathbf{I})$; 50,000 replications)

		Solv.	Soft	C & E	Hard	Minor	Serious
Baseline	$\widehat{P}_j$	7.0	43.7	13.1	2.6	64.7	9.9
white male	$\overline{\tau}_j$	14.3	16.9	19.3	18.5	14.5	15.2
Disadvant-	$\Delta\widehat{P}_j$	+27.5	+48.0	+43.6	+28.3	+34.5	+52.7
aged	$\Delta\overline{\tau}_j$	-0.6	-2.3	-1.7	-1.7	-2.2	-0.9
Female	$\Delta\widehat{P}_j$	-2.5	-11.1	-6.8	-1.4	-15.1	-7.9
	$\Delta\overline{\tau}_j$	+0.1	+0.3	+0.4	+0.1	+0.3	-0.0
Asian	$\Delta\widehat{P}_j$	-2.4	-30.0	-10.1	+0.4	-18.9	-4.7
	$\Delta\overline{\tau}_j$	+0.2	+0.9	+0.5	-0.5	+0.3	-0.1
Black	$\Delta\widehat{P}_j$	-4.5	-15.5	-9.1	-2.0	-1.2	+3.9
	$\Delta\overline{\tau}_j$	+0.2	+0.5	+0.4	+0.1	+0.0	+0.0
High	$\Delta\widehat{P}_j$	+2.7	+38.4	+17.5	+15.4	+10.0	+12.5
prevalence	$\Delta\overline{\tau}_j$	-0.1	-1.5	-0.7	-1.1	-0.2	-0.2

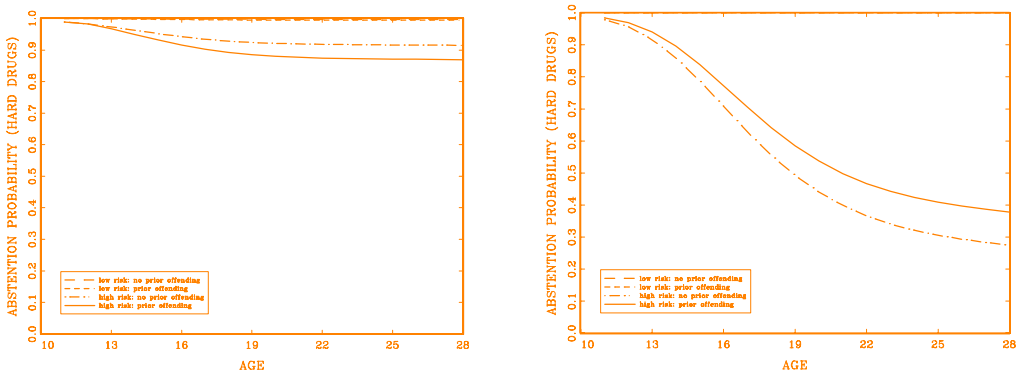
6 The gateway effect

The "gateway" or "slippery slope" effect is the increase in hazard rate for onset of hard drug use which is induced by prior use of soft drugs. It is important that this comparison is made holding constant all personal characteristics, observed and unobserved, to avoid selection bias. We illustrate the estimated gateway effect and its sensitivity to selection bias in Figures 4-8. For any behaviour type j, these show the abstention or survivor probability $P_j(t|\mathbf{x}_{j1}...\mathbf{x}_{jt}, u_j)$ plotted against age t, where:

$$\begin{aligned} P(t|\mathbf{x}_{j1}...\mathbf{x}_{jt}, u_j) &= \Pr(\tau_j > t|\mathbf{x}_{j1}...\mathbf{x}_{jt}, u_j) \\ &= \prod_{s=1}^{t} \left[1 - \Phi(\mathbf{x}_{js}\boldsymbol{\beta}_j + u_j)\right] \end{aligned} \tag{13}$$

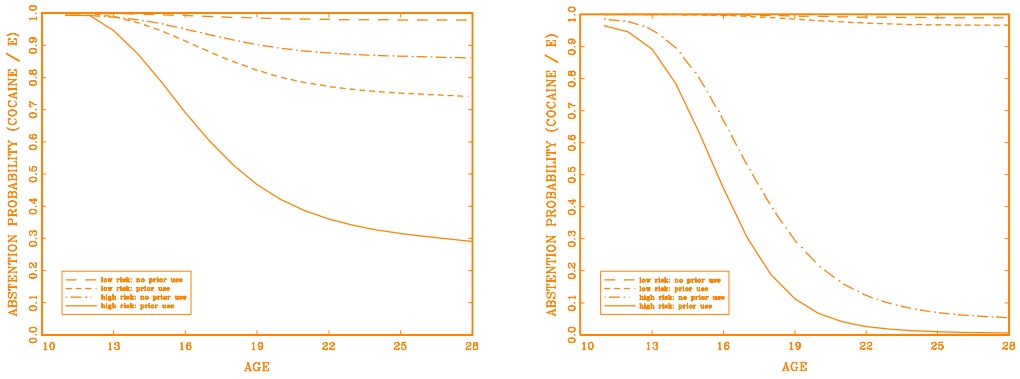
Each panel shows four cases: (i) a white male with two working parents living in a 'good' neighbourhood with low drug prevalence and no previous experience of drug use or offending; (ii) the same circumstances, except for

early (age 12) experience of a particular drug or offence; (iii) a white male with absent father and working mother, living in a deprived inner-city area at a time of high drug prevalence but no personal history of drug use or offending; and (iv) the same individual but with early experience of a particular drug or offence. Note that there are thirty possible plots corresponding to the effect of each of the six drugs/offences on the other five. Of these, we plot only the five for which there is a significant positive gateway effect in the random effects model. For each plot, two separate panels give the survivor probabilities calculated using (a) the model estimated without random effects and (b) the preferred random effects specification, but with the u_j set to their means of zero when calculating (13). A comparison of (a) and (b) gives an indication of the sensitivity of the results to selection bias. In almost all cases the estimated impact of past behaviour on subsequent behaviour is considerably smaller for the random effects model. Thus selection bias tends to exaggerate the estimated gateway effect.



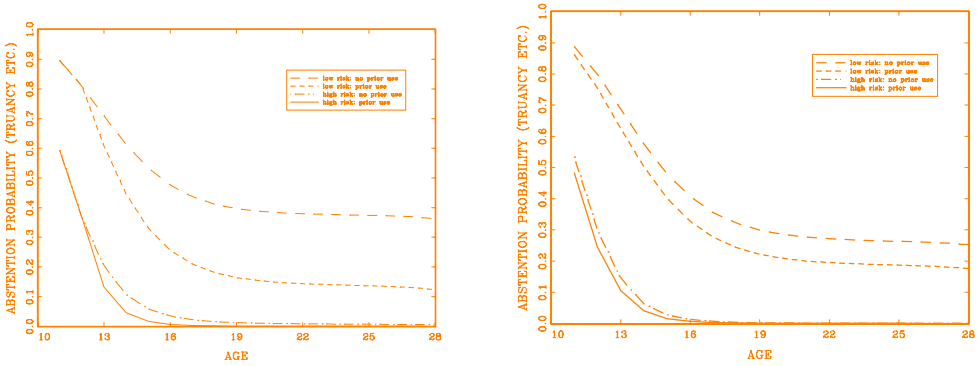
(a) non-heterogeneous model **(b)** random effects model

Figure 4 The effect of prior truancy/minor crime on the abstention probability for hard drugs



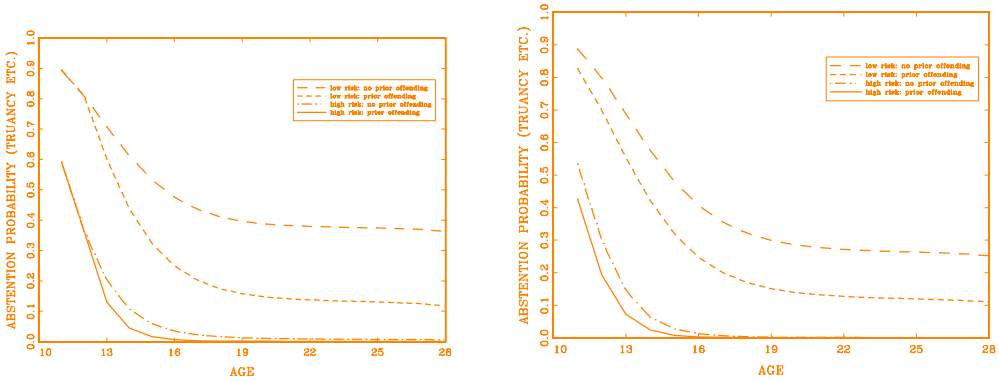
(a) non-heterogeneous model **(b)** random effects model

Figure 5 The effect of prior soft drug use on the abstention probability for cocaine/E



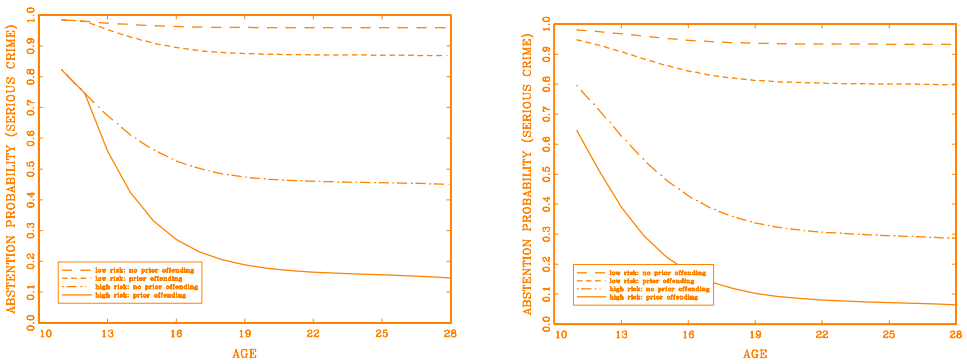
(a) non-heterogeneous model **(b)** random effects model

Figure 6 The effect of prior soft drug use on the abstention probability for truancy/minor crime



(a) non-heterogeneous model **(b)** random effects model

Figure 7 The effect of prior serious crime on the abstention probability for truancy/minor crime



(a) non-heterogeneous model **(b)** random effects model

Figure 8 The effect of prior truancy/minor crime on the abstention probability for serious crime

Figures 4-8 give a good impression of the estimated direct impact of the past use of one category of drug on the current use of another. However, they ignore the indirect effects that might arise through feedback from the impact on the use of other categories. Tables 7 and 8 give a fuller picture of these impacts using stochastic simulation. We first simulate a complete offending history for the age range 11-30 for each individual in the YLS sample Then the simulation is repeated (re-using the same set of pseudo-random variates), but with the hazard rate for one of the drug categories constrained to be zero. This is repeated for each category in turn; each set of results is then compared with the baseline in terms of prevalence among the simulated individuals and the average age of onset. Table 7 summarises the simulation results produced using estimates from a non-heterogeneous version of the model, from which random effects are excluded. Table 8 gives the simulation results for the random-effects model. To make the results representative of the YLS target population, we use a fresh draw of the random effect $\mathbf{u}$ for each individual (held constant across all simulations for that individual). To the extent that the model is correctly specified, comparisons of the baseline and perturbed simulations give an assessment of the causal impact, or gateway effect, of each type of offending on other forms of offending, after controlling for the influence of unobservables.

Table 7 Impact of early experience of solvent abuse, soft drugs, truancy or crime on subsequent behaviour (model without random effects)

Effect on use of ...		Solv.	Soft	E/coc	Hard	Tru.	S.crime
Baseline case	$\widehat{P}_j$	8.3	41.7	11.7	1.9	57.3	15.3
	$\overline{\tau}_j$	15.3	16.8	19.3	19.0	14.3	15.4
Effect of removal of risk arising from ...							
...Glue/solvents	$\Delta\widehat{P}_j$	-	-1.8	-0.9	-0.3	-0.5	-0.3
	$\Delta\overline{\tau}_j$	-	+0.1	-0.0	-0.5	-0.0	-0.1
...Soft drugs	$\Delta\widehat{P}_j$	-0.9	-	-8.3	-1.3	-3.0	-1.2
	$\Delta\overline{\tau}_j$	-0.4	-	-0.9	-2.9	-0.2	-0.5
...Ecstasy/cocaine	$\Delta\widehat{P}_j$	+0.0	-0.7	-	-0.8	-0.1	-0.1
	$\Delta\overline{\tau}_j$	+0.0	-0.0	-	-1.9	-0.0	-0.1
...Hard drugs	$\Delta\widehat{P}_j$	-0.0	-0.1	-0.0	-	-0.0	-0.0
	$\Delta\overline{\tau}_j$	+0.0	-0.0	-0.0	-	-0.0	-0.1
...Truancy, minor	$\Delta\widehat{P}_j$	-4.1	-10.8	-5.0	-0.9	-	-4.6
crime	$\Delta\overline{\tau}_j$	-1.1	-0.1	-0.3	-1.6	-	-1.9
...Serious crime	$\Delta\widehat{P}_j$	-0.6	-0.8	-0.6	-0.2	-0.5	-
	$\Delta\overline{\tau}_j$	-0.1	-0.0	-0.0	-0.2	+0.0	-

Table 8 Impact of early experience of solvent abuse, soft drugs, truancy or crime on subsequent behaviour (random effects model)

Effect on use of ...		Solv.	Soft	E/coc	Hard	Tru.	S.crime
Baseline case	$\widehat{P}_j$	8.2	40.2	11.7	2.5	55.9	9.1
	$\overline{\tau}_j$	14.1	16.7	19.2	17.9	14.0	14.9
Effect of removal of risk arising from ...							
...Glue/solvents	$\Delta\widehat{P}_j$	-	-0.2	+1.1	+0.6	+0.0	+0.1
	$\Delta\overline{\tau}_j$	-	+0.0	-0.2	+0.2	+0.0	+0.0
...Soft drugs	$\Delta\widehat{P}_j$	+0.7	-	-3.8	-0.2	-0.6	+0.2
	$\Delta\overline{\tau}_j$	+0.2	-	-0.5	-0.4	-0.0	+0.1
...Ecstasy/cocaine	$\Delta\widehat{P}_j$	+0.2	+0.0	-	-0.3	+0.1	+0.0
	$\Delta\overline{\tau}_j$	+0.0	-0.0	-	-0.6	+0.0	+0.0
...Hard drugs	$\Delta\widehat{P}_j$	+0.0	+0.1	+0.2	-	+0.0	+0.1
	$\Delta\overline{\tau}_j$	+0.0	-0.0	-0.0	-	-0.0	+0.1
...Truancy, minor	$\Delta\widehat{P}_j$	-2.0	-1.3	+0.9	+0.8	-	-3.3
crime	$\Delta\overline{\tau}_j$	-0.2	+0.0	-0.0	+0.1	-	-1.0
...Serious crime	$\Delta\widehat{P}_j$	-0.2	+0.2	+0.3	+0.1	-0.2	-
	$\Delta\overline{\tau}_j$	+0.0	+0.0	-0.1	+0.2	-0.0	-

In both the non-heterogeneous and heterogeneous models, gateway effects are generally moderate. For the former model however, there are large gateway effects for truancy and minor crime, with particularly large reductions in the prevalence of all types of drug use. After allowing for unobservable random effects, these impacts, more or less disappear. Although there are statistically significant positive dynamic effects in 8 of the 30 possible cases, these impacts are small and there is little convincing evidence of important causal pathways leading from one type of drug use to others.

7 Conclusions

We have applied discrete statistical duration methods to data from the 1998 Youth Lifestyles Survey to investigate the age of onset of various types of

crime and illicit drug use. The YLS data suggest at first sight that there are widespread and strong links between early experience of minor offending/drug use and later involvement in more serious crime and drug abuse. However, this turns out not to be a very robust finding. After making allowance for unobservable individual-specific random effects, the estimates of these dynamic impacts are reduced considerably and remain small even where statistically significant. This is in line with the results of analogous work by van Ours (2000) using data from Amsterdam.

The estimated impacts of social, family and cultural factors are much more important. The background 'drug culture' of society, as proxied by aggregate drug prevalence trends, is also a dominant influence although the YLS data is not adequate to allow us to distinguish externalities such as demonstration effects from price and income effects.

The policy implications of our findings are important. There is little support here for a policy directed at reducing early exposure to soft drugs. Our best-fitting statistical model suggests that the impact of eliminating soft drug use completely would only be to reduce prevalence of the most damaging category of drugs by some 8%[11] - a statistically insignificant figure. Our results suggest instead that an effective policy directed at reducing the extent of social deprivation may have a better chance of success. In either case, it seems inevitable that progress will be slow.

References

[1] Abbring, J. H. and van den Berg, G. J. (2000). The non-parametric identification of treatment effects in duration models. Working paper, Free University Amsterdam.

[2] Becker, G. S. and Murphy, K. M. (1988). A theory of rational addiction. *Journal of Political Economy* **96**, 675-700.

[11] A 0.2% reduction from a 2.5% base level of those who have used crack, heroin or methadone by age 30. However, note that there is a larger (and statistically significant) reduction of around one third on the prevalence of cocaine / ecstasy.

[3] Brock, W. and Durlauf, S. (2000). Interactions-based models, in *Handbook of Econometrics, vol. 5*, J. Heckman and E. Leamer (eds.). Amsterdam: North-Holland.

[4] Butler, J. S. and Moffitt, R. (1982), "A Computationally Efficient Quadrature Procedure for the One-Factor Multinomial Probit Model", *Econometrica*, 50, 761-764.

[5] Fergusson, D. M. and Horwood, L. J. (2000). Does cannabis use encourage other forms of illicit drug use? *Addiction* **95**, 505-520.

[6] Flood-Page, C., Campbell, S., Harrington, V. and Miller, J. (2000). *Youth Crime: Findings from the 1998/99 Youth Lifestyles Survey*. London: Home Office Research Study no. 209.

[7] Gourieroux, C. and Monfort, A. (1996) *Simulation-Based Econometric Methods* (Oxford University Press, Oxford).

[8] Grossman, M. and Chaloupka, F. J. (1998). The demand for cocaine by young adults: a rational addiction approach, *Journal of Health Economics* **17**, 427-474.

[9] Kandel, D. B., Yamaguchi, K. and Chen, K. (1992). Stages of progression in drug involvement from adolescence to adulthood: further evidence for the gateway theory, *Journal of Studies on Alcohol* **53**, 447-457.

[10] Kenkel, D., Mathios, A. D. and Pacula, R. L. (2001). Economics of youth drug use, addiction and gateway effects, *Addiction* **96**, 151-164.

[11] Manski, C. F. (1993). Identification of social effects: the reflection problem, *Review of Economic Studies* **60**, 531-542.

[12] Manski, C. F. (2000). Economic analysis of social interactions, *Journal of Economic Perspectives* **14**, 115-136.

[13] Maughan, B., Pickles, A., Rowe, R., Costello, E. and Angold, A. (2000). Developmental trajectories of aggressive and non-aggressive conduct problems, *Journal of Quantitative Criminology* **16**, 199-221.

[14] Mealli, F. and Pudney, S. E. (1996), "Occupational Pensions and Job Mobility in Britain: Estimation of a Random-Effects Competing Risks Model", *Journal of Applied Econometrics*, 11, 293-320.

[15] Pacula, R. (1997). The modelling of the gateway effect, *Health Economics* **6**, 521-524.

[16] Pudney, S. E. (2001).The growth of illicit drugs markets in the UK 1978-99. *Mimeo*, University of Leicester: Public Sector Economics Research Centre.

[17] Stenbacka, M., Allebeck, P. and Romelsjo, A. (1993). Initiation into drug use: the pathway from being offered drugs to trying cannabis and progression to intravenous drug abuse, *Scandinavian Journal of Social Medicine* **21**, 31-39.

[18] UKADC (2000). United Kingdom's Anti-Drugs Co-ordinator's Second National Plan 2000/2001. London: Cabinet Office.

[19] van Ours, J. C. (2000). On the dynamics in the consumption of alcohol, tobacco, cannabis and cocaine. Working paper, Tilburg University.

[20] Stratford, N. and Roth, W. (1999). *The 1998 Youth Lifestyles Survey Technical Report*, London: National Centre for Social Research.

[21] Yamaguchi, K. and Kandel, D. B. (1984a). Patterns of drug use from adolescence to young adulthood II. Sequences of progression, *American Journal of Public Health* **74**, 668-672.

[22] Yamaguchi, K. and Kandel, D. B. (1984b). Patterns of drug use from adolescence to young adulthood III. Predictors of progression, *American Journal of Public Health* **74**, 673-681.

Appendix: Data characteristics and full estimates

Table A1 Definitions and sample means of explanatory variables

Variable	Description	Mean
Female	Dummy = 1 if female	0.498
Asian	Dummy = 1 if Indian, Pakistani or Bangladeshi	0.044
Black	Dummy = 1 if Afro-Caribbean, black African or other black	0.018
Religious	Dummy = 1 if respondent claims religious affiliation	0.135
Absent father	Father absent at age 15 or absent currently if below 16	0.065
Absent mother	Mother absent at age 15 or absent currently if below 16	0.012
Working father	Father in work at age 15 or currently if below 16	0.857
Working mother	Mother in work at age 15 or currently if below 16	0.716
Family Trouble	Dummy = 1 if parents have been in trouble with police	0.017
Inner City	Dummy = 1 if resident in inner-city area	0.146
Deprived area	Dummy = 1 if resident in a deprived neighbourhood	0.065
Age98	Age at time of interview	20.76

Table A2 Single-equation results for minor offences

Covariate	Tobacco	Alcohol	Glue	Cannabis	Truancy	Minor crime
Female	0.095	-0.086	-0.066	-0.150	-0.081	-0.317
	(0.025)	(0.025)	(0.052)	(0.030)	(0.032)	(0.026)
Asian	-0.288	-1.007	0.007	-0.395	-0.171	-0.112
	(0.069)	(0.081)	(0.134)	(0.117)	(0.092)	(0.073)
Black	-0.217	-0.403	-0.451	-0.122	-0.031	0.002
	(0.076)	(0.084)	(0.218)	(0.121)	(0.097)	(0.091)
Religious	-0.176	-0.055	0.091	-0.095	-0.172	-0.065
	(0.039)	(0.041)	(0.078)	(0.050)	(0.056)	(0.043)
Absent father	0.084	0.139	0.064	0.118	0.181	0.115
	(0.060)	(0.063)	(0.125)	(0.072)	(0.071)	(0.066)
Absent mother	0.059	-0.021	-0.148	0.152	0.175	-0.162
	(0.108)	(0.102)	(0.203)	(0.122)	(0.126)	(0.121)
Working father	0.058	0.180	0.008	0.020	-0.148	0.034
	(0.045)	(0.044)	(0.095)	(0.055)	(0.055)	(0.048)
Working mother	0.003	0.060	-0.041	0.107	-0.068	-0.000
	(0.028)	(0.029)	(0.058)	(0.034)	(0.036)	(0.030)
Family trouble	0.320	0.069	0.345	0.222	0.440	0.443
	(0.116)	(0.102)	(0.141)	(0.120)	(0.122)	(0.098)
Inner city	-0.024	-0.080	0.031	0.066	0.103	0.118
	(0.030)	(0.031)	(0.065)	(0.037)	(0.039)	(0.033)
Deprived area	0.061	-0.116	0.169	0.107	0.133	-0.059
	(0.044)	(0.047)	(0.087)	(0.055)	(0.057)	(0.050)
Initial period	0.498	0.950	0.421	0.179	0.339	0.230
(up to age 11)	(0.054)	(0.076)	(0.156)	(0.105)	(0.195)	(0.057)
Age/10	33.09	27.80	44.10	20.96	-163.0	5.295
	(2.02)	(6.07)	(8.42)	(1.95)	(57.7)	(1.806)
$(\text{Age}/10)^2$	-17.87	-12.09	-25.94	-10.88	136.2	-3.510
	(1.10)	(3.78)	(4.67)	(1.02)	(41.5)	(0.968)
$(\text{Age}/10)^3$	2.978	1.459	4.625	1.727	-36.87	0.630
	(0.190)	(0.770)	(0.804)	(0.172)	(9.91)	(0.167)
Prevalence index	-	-	-	0.570	-	-
				(0.035)		
Time	-0.416	-0.858	0.642	-	0.413	-0.044
	(0.114)	(0.116)	(0.255)		(0.162)	(0.127)
Time^2	0.244	0.613	-0.248	-	-0.263	0.145
	(0.052)	(0.053)	(0.112)		(0.074)	(0.056)

Table A2 (cont.) Single-equation results for minor offences

Covariate	Tobacco	Alcohol	Glue	Cannabis	Truancy	Minor crime
Tobacco	-	0.465	0.355	0.476	0.406	0.208
		(0.034)	(0.067)	(0.035)	(0.040)	(0.036)
Alcohol	0.335	-	0.306	0.538	0.046	0.247
	(0.034)		(0.068)	(0.040)	(0.042)	(0.038)
Glue	-0.249	0.091	-	0.545	0.200	0.113
	(0.150)	(0.140)		(0.084)	(0.119)	(0.095)
Cannabis	0.330	0.529	0.113	-	0.252	0.229
	(0.099)	(0.142)	(0.102)		(0.098)	(0.055)
Truancy	-0.019	0.087	0.292	0.115	-	0.164
	(0.048)	(0.047)	(0.071)	(0.035)		(0.038)
Minor crime	0.152	0.302	0.396	0.247	0.221	-
	(0.044)	(0.045)	(0.065)	(0.034)	(0.048)	
Amphet's	-0.311	-0.339	0.098	0.022	0.157	0.194
	(0.185)	(0.371)	(0.185)	(0.174)	(0.245)	(0.090)
Ecstasy	0.415	-0.312	-1.072	-0.101	0.327	-0.146
	(0.236)	(0.364)	(0.380)	(0.352)	(0.481)	(0.143)
LSD	-0.319	-0.499	-0.322	0.334	0.006	-0.183
	(0.251)	(0.286)	(0.208)	(0.208)	(0.253)	(0.113)
Mushrooms	-0.078	-0.052	0.323	0.209	-0.049	-0.068
	(0.210)	(0.353)	(0.185)	(0.120)	(0.280)	(0.106)
Tranqu's	-0.134	-1.326	0.639	0.512	-0.465	0.460
	(0.375)	(0.635)	(0.267)	(0.458)	(0.413)	(0.185)
Amyl nitrite	0.070	0.544	0.135	0.465	-0.159	0.151
	(0.147)	(0.339)	(0.150)	(0.089)	(0.191)	(0.083)
Cocaine	-0.195	0.027	0.650	-0.480	-	0.140
	(0.430)	(0.394)	(0.294)	(0.666)		(0.165)
Crack	0.267	-0.472	-0.591	-	-	-0.050
	(0.641)	(0.493)	(0.716)			(0.259)
Heroin	-0.660	-	0.485	0.636	0.913	-0.484
	(0.609)		(0.521)	(0.493)	(0.274)	(0.328)
Methadone	0.676	-	-0.133	0.213	0.758	-
	(0.637)		(0.609)	(0.114)	(1.134)	
Serious crime	-0.136	0.218	0.273	0.029	0.117	0.404
	(0.104)	(0.127)	(0.116)	(0.072)	(0.117)	(0.111)

Table A2 (cont.) Single-equation results for soft drugs

Covariate	Amph's	Ecstasy	LSD	Mushrooms	Tranqu's	Amyl N
Female	-0.133	-0.244	-0.201	-0.371	0.022	-0.195
	(0.040)	(0.055)	(0.049)	(0.053)	(0.074)	(0.041)
Asian	-	-0.224	-0.249	-0.288	0.296	-0.450
		(0.341)	(0.252)	(0.241)	(0.258)	(0.248)
Black	-0.328	-0.082	-0.892	-0.501	0.018	-0.131
	(0.151)	(0.200)	(0.447)	(0.302)	(0.291)	(0.137)
Religious	-0.118	0.022	-0.016	0.063	-0.029	-0.141
	(0.071)	(0.090)	(0.088)	(0.089)	(0.127)	(0.075)
Absent father	-0.064	-0.044	0.050	-0.058	0.036	0.019
	(0.098)	(0.129)	(0.104)	(0.115)	(0.140)	(0.106)
Absent mother	0.158	-0.098	0.141	0.209	0.316	0.157
	(0.143)	(0.231)	(0.180)	(0.172)	(0.192)	(0.156)
Working father	-0.083	-0.033	-0.116	-0.082	-0.200	0.094
	(0.072)	(0.097)	(0.084)	(0.088)	(0.115)	(0.080)
Working mother	0.042	0.118	0.065	-0.044	0.043	-0.010
	(0.044)	(0.064)	(0.057)	(0.055)	(0.086)	(0.045)
Family trouble	0.233	0.054	0.031	-0.111	0.066	0.040
	(0.123)	(0.160)	(0.158)	(0.193)	(0.185)	(0.154)
Inner city	0.042	-0.057	-0.003	-0.013	-0.103	-0.011
	(0.048)	(0.065)	(0.057)	(0.062)	(0.091)	(0.051)
Deprived area	0.039	0.152	-0.098	-0.032	0.198	0.126
	(0.069)	(0.002)	(0.091)	(0.093)	(0.116)	(0.070)
Initial period	-0.083	-	-	-0.021	-0.241	-0.073
(up to age 11)	(0.233)			(0.236)	(0.318)	(0.201)
Age/10	15.00	16.36	18.41	18.65	3.59	19.25
	(2.76)	(4.28)	(3.37)	(3.55)	(4.73)	(2.69)
$(\text{Age}/10)^2$	-7.55	-7.78	-9.26	-9.81	-2.15	-10.24
	(1.42)	(2.15)	(1.77)	(1.86)	(2.45)	(1.40)
$(\text{Age}/10)^3$	1.160	1.147	1.412	1.584	0.337	1.666
	(0.230)	(0.351)	(0.302)	(0.318)	(0.411)	(0.236)
Prevalence index	0.324	0.061	0.362	-	-	-
	(0.035)	(0.011)	(0.053)			
Time	-	-	-	1.316	1.592	2.832
				(0.380)	(0.757)	(0.422)
Time^2	-	-	-	-0.570	-0.474	-1.017
				(0.152)	(0.279)	(0.159)

Table A2 (cont.) Single-equation results for soft drugs

Covariate	Amph's	Ecstasy	LSD	Mushrooms	Tranqu's	Amyl N.
Tobacco	0.188	0.181	0.155	0.205	-0.020	0.163
	(0.051)	(0.077)	(0.063)	(0.068)	(0.105)	(0.052)
Alcohol	0.476	0.481	0.210	0.221	0.373	0.525
	(0.069)	(0.122)	(0.076)	(0.081)	(0.157)	(0.064)
Glue	0.152	-0.028	-0.028	0.117	0.173	0.251
	(0.072)	(0.087)	(0.079)	(0.084)	(0.095)	(0.074)
Cannabis	0.577	0.460	0.559	0.439	0.572	0.452
	(0.051)	(0.071)	(0.067)	(0.072)	(0.121)	(0.054)
Truancy	0.241	0.120	0.151	0.202	-0.021	0.045
	(0.043)	(0.057)	(0.054)	(0.056)	(0.083)	(0.048)
Minor crime	0.116	0.062	0.150	0.105	0.256	0.194
	(0.044)	(0.058)	(0.055)	(0.057)	(0.086)	(0.046)
Amphet's	-	0.505	0.300	0.351	0.232	0.188
		(0.077)	(0.082)	(0.092)	(0.109)	(0.088)
Ecstasy	0.345	-	0.290	-0.279	0.259	0.008
	(0.195)		(0.136)	(0.141)	(0.115)	(0.133)
LSD	0.553	0.347	-	0.235	0.181	0.054
	(0.106)	(0.085)		(0.109)	(0.109)	(0.109)
Mushrooms	0.213	-0.016	0.095	-	0.197	-0.101
	(0.093)	(0.091)	(0.100)		(0.103)	(0.108)
Tranqu's	0.130	0.007	-0.101	-0.142	-	0.166
	(0.163)	(0.151)	(0.165)	(0.175)		(0.209)
Amyl nitrite	0.281	0.178	0.324	0.230	0.145	-
	(0.062)	(0.070)	(0.074)	(0.081)	(0.090)	
Cocaine	0.132	0.186	0.031	0.151	-0.193	0.002
	(0.250)	(0.149)	(0.189)	(0.186)	(0.157)	(0.200)
Crack	-	0.008	0.056	-0.308	-0.006	-0.255
		(0.303)	(0.352)	(0.317)	(0.276)	(0.278)
Heroin	-	0.218	0.265	-0.514	0.986	0.185
		(0.289)	(0.321)	(0.454)	(0.253)	(0.280)
Methadone	1.146	-0.737	0.651	-	0.242	-0.465
	(0.477)	(0.334)	(0.569)		(0.407)	(0.501)
Serious crime	0.213	-0.008	0.145	0.112	0.036	0.141
	(0.077)	(0.094)	(0.088)	(0.089)	(0.109)	(0.078)

Table A2 (cont.) Single-equation results for hard drugs & serious crime

Covariate	Cocaine	Crack	Heroin	Methadone	S. crime
Female	-0.150	-0.126	0.013	0.021	-0.512
	(0.068)	(0.116)	(0.135)	(0.158)	(0.053)
Asian	0.278	-0.804	0.465	0.557	-0.048
	(0.211)	(0.181)	(0.261)	(0.342)	(0.144)
Black	0.038	0.081	-	0.492	0.239
	(0.184)	(0.361)		(0.342)	(0.129)
Religious	0.086	0.070	0.085	-0.077	0.004
	(0.106)	(0.136)	(0.447)	(0.175)	(0.075)
Absent father	0.063	0.025	0.368	0.357	0.122
	(0.150)	(0.198)	(0.281)	(0.277)	(0.111)
Absent mother	-0.174	0.513	0.312	0.386	-0.107
	(0.257)	(0.214)	(0.339)	(0.355)	(0.214)
Working father	0.139	0.129	0.576	0.321	-0.057
	(0.118)	(0.167)	(0.300)	(0.299)	(0.086)
Working mother	-0.129	-0.039	-0.107	0.056	0.029
	(0.073)	(0.115)	(0.133)	(0.154)	(0.056)
Family trouble	0.120	-0.104	-0.166	0.689	0.508
	(0.187)	(0.278)	(0.269)	(0.200)	(0.118)
Inner city	0.023	0.108	0.124	-0.389	0.163
	(0.072)	(0.119)	(0.137)	(0.149)	(0.056)
Deprived area	0.199	0.281	-0.055	0.557	0.019
	(0.104)	(0.144)	(0.220)	(0.137)	(0.085)
Initial period	0.833	0.075	0.413	-0.147	0.510
(up to age 11)	(0.296)	(0.363)	(0.366)	(0.375)	(0.108)
Age/10	14.68	9.79	35.07	-6.19	15.38
	(4.64)	(6.71)	(7.52)	(10.1)	(3.46)
$(\text{Age}/10)^2$	-7.56	-5.45	-19.84	3.17	-9.22
	(2.28)	(3.37)	(3.83)	(5.30)	(1.85)
$(\text{Age}/10)^3$	1.228	0.886	3.423	-0.590	1.632
	(0.365)	(0.547)	(0.623)	(0.893)	(0.316)
Prevalence index	0.226	0.109	0.241	0.279	-
	(0.070)	(0.042)	(0.102)	(0.141)	
Time	-	-	-	-	-0.092
					(0.233)
Time^2	-	-	-	-	0.167
					(0.100)

Table A2 (cont.) Single-equation results for hard drugs & serious crime

Covariate	Cocaine	Crack	Heroin	Methadone	S. crime
Tobacco	0.182	0.425	0.097	-0.102	0.118
	(0.099)	(0.148)	(0.194)	(0.226)	(0.068)
Alcohol	0.335	0.313	0.219	-0.013	0.199
	(0.161)	(0.237)	(0.214)	(0.327)	(0.078)
Glue	0.086	0.031	0.214	-0.014	0.117
	(0.086)	(0.159)	(0.168)	(0.194)	(0.096)
Cannabis	0.446	-0.020	0.086	0.452	0.063
	(0.095)	(0.164)	(0.243)	(0.330)	(0.088)
Truancy	0.010	0.242	0.142	0.037	0.275
	(0.070)	(0.133)	(0.170)	(0.176)	(0.065)
Minor crime	0.211	-0.101	0.160	0.261	0.595
	(0.073)	(0.128)	(0.162)	(0.218)	(0.063)
Amphet's	0.539	0.543	0.505	-0.036	0.274
	(0.097)	(0.219)	(0.272)	(0.253)	(0.112)
Ecstasy	0.449	0.121	0.564	0.004	-0.041
	(0.091)	(0.183)	(0.208)	(0.194)	(0.140)
LSD	0.273	0.135	0.345	0.626	0.079
	(0.091)	(0.182)	(0.215)	(0.189)	(0.122)
Mushrooms	0.092	0.218	-0.063	0.154	0.013
	(0.086)	(0.146)	(0.198)	(0.152)	(0.110)
Tranqu's	0.110	0.425	0.618	0.245	0.100
	(0.134)	(0.172)	(0.201)	(0.215)	(0.207)
Amyl nitrite	0.113	0.164	0.219	0.213	-0.025
	(0.080)	(0.159)	(0.195)	(0.232)	(0.102)
Cocaine	-	0.587	0.220	-0.010	0.033
		(0.159)	(0.203)	(0.220)	(0.189)
Crack	0.220	-	0.784	-0.403	-0.001
	(0.366)		(0.263)	(0.423)	(0.246)
Heroin	-0.317	0.940	-	1.373	0.199
	(0.329)	(0.272)		(0.294)	(0.297)
Methadone	0.252	-0.344	0.210	-	0.122
	(0.385)	(0.395)	(0.454)		(0.342)
Serious crime	-0.132	-0.080	0.185	0.099	-
	(0.094)	(0.163)	(0.172)	(0.204)	

Table A3 Results for the multivariate random-effects model

Covariate	Solvents	Soft	Cocaine/E	Hard	Truancy, etc	Serious crime
Female	-0.212	-0.305	-0.550	-0.426	-0.255	-0.624
	(0.076)	(0.046)	(0.083)	(0.156)	(0.029)	(0.064)
Asian	-0.183	-0.940	-0.848	-0.015	-0.311	-0.146
	(0.225)	(0.175)	(0.412)	(0.546)	(0.079)	(0.174)
Black	-0.599	-0.405	-0.823	-0.732	-0.000	0.184
	(0.259)	(0.135)	(0.379)	(0.980)	(0.087)	(0.155)
Religious	0.051	-0.213	0.005	-0.120	-0.196	-0.030
	(0.113)	(0.073)	(0.131)	(0.281)	(0.045)	(0.096)
Absent father	0.358	0.380	0.300	0.246	0.173	0.232
	(0.147)	(0.096)	(0.168)	(0.338)	(0.064)	(0.113)
Absent mother	-0.171	0.329	0.197	0.542	0.093	-0.129
	(0.270)	(0.174)	(0.269)	(0.437)	(0.125)	(0.253)
Working father	0.078	0.087	0.128	0.153	-0.075	-0.049
	(0.112)	(0.074)	(0.128)	(0.271)	(0.048)	(0.088)
Working mother	0.017	0.151	0.119	0.026	-0.008	0.095
	(0.074)	(0.050)	(0.080)	(0.156)	(0.032)	(0.060)
Family trouble	0.844	0.837	1.015	0.999	0.723	0.783
	(0.202)	(0.163)	(0.245)	(0.374)	(0.102)	(0.149)
Inner city	0.068	0.074	0.146	0.182	0.112	0.206
	(0.082)	(0.052)	(0.086)	(0.175)	(0.035)	(0.064)
Deprived area	0.136	0.097	0.262	0.285	0.079	0.009
	(0.122)	(0.079)	(0.122)	(0.228)	(0.051)	(0.089)
Initial period	0.497	0.092	0.790	0.281	0.387	0.604
(up to age 11)	(0.179)	(0.133)	(0.401)	(0.486)	(0.061)	(0.140)
Age/10	43.62	26.11	21.36	15.35	26.76	15.39
	(5.49)	(2.44)	(4.77)	(9.53)	(1.97)	(3.95)
$(\text{Age}/10)^2$	-23.84	-11.69	-8.592	-6.899	-14.38	-8.376
	(3.01)	(1.24)	(2.284)	(4.945)	(1.08)	(2.110)
$(\text{Age}/10)^3$	4.035	1.634	1.074	0.974	2.397	1.387
	(0.535)	(0.205)	(0.360)	(0.841)	(0.190)	(0.364)
Prevalence index	-	0.704	0.258	0.476	-	-
		(0.053)	(0.047)	(0.182)		
Time	0.227	-	-	-	0.054	-0.169
	(0.359)				(0.131)	(0.287)
Time^2	-0.054	-	-	-	0.021	0.218
	(0.149)				(0.056)	(0.118)

Table A3 (cont.) Results for the multivariate random-effects model effects of prior offending history and of random effects

Prior use of...	Solvents	Soft	Cocaine/E	Hard	Truancy, etc	Serious crime
...Solvents	-	0.196	0.527	-0.375	-0.002	-0.053
		(0.106)	(0.113)	(0.184)	(0.130)	(0.109)
...Soft drugs	-0.240	-	0.399	0.061	0.127	-0.034
	(0.095)		(0.079)	(0.230)	(0.058)	(0.088)
...Cocaine / E	-0.533	0.023	-	0.229	-0.181	-0.033
	(0.338)	(0.364)		(0.160)	(0.139)	(0.140)
...Hard drugs	0.005	-0.327	-0.606	-	-0.389	-0.245
	(0.352)	(0.451)	(0.239)		(0.439)	(0.281)
...Truancy, etc.	0.347	0.093	-0.078	-0.162	-	0.440
	(0.073)	(0.049)	(0.092)	(0.170)		(0.068)
...Serious crime	0.104	-0.136	-0.207	-0.052	0.233	-
	(0.119)	(0.105)	(0.106)	(0.162)	(0.118)	
Scale parameters for random effects (**R**)						
	0.874	0.885	1.123	1.215	0.403	0.468
	(0.100)	(0.065)	(0.142)	(0.231)	(0.032)	(0.071)