Published in final edited form as:

Ann Epidemiol. 2007 January ; 17(1): 74–80.

Ecstasy and Gateway Drugs: Initiating the Use of Ecstasy and Other Drugs

Lesley W. Reid, Kirk W. Elifson, and Claire E. Sterk

Abstract

Purpose—The main purposes of this study are to examine if, and to what extent, ecstasy use serves as a gateway to the use of hard drugs such as cocaine, heroin, and methamphetamine and to compare the age of onset of alcohol and marijuana use and subsequent use of cocaine, heroin, and methamphetamine among young adult ecstasy users.

Methods—Face-to-face surveys were conducted with 268 young adult ecstasy users in Atlanta, Georgia. Subjects were solicited using the community identification process, including targeted sampling and guided recruitment. Data analysis involved discrete-time, event history analysis.

Results—Results suggest that the age of onset of ecstasy use influences the initiation of cocaine and methamphetamine for our sample of active ecstasy users. In addition, alcohol and marijuana use precedes the initiation of cocaine and methamphetamine, but only marijuana influences the initiation of heroin.

Conclusions—The sequential progression of drug use proposed in the gateway literature is not immutable. Researchers must take into account the changing popularity of drugs over time, such as the emergence of ecstasy use, when identifying patterns of drug use onset.

Keywords

MDMA; Ecstasy (Drug); 3,4-Methylenedioxymethamphetamine; street drugs

The nature of the developmental progression of drug use trajectories remains a hotly debated topic, often focusing on the validity of the gateway theory (1). This theory assumes that the use of specific drugs is related to a sequential pattern of initiation, although the causal mechanisms that drive this sequential trajectory are often unclear (2). Kandel's seminal research identified marijuana as the key gateway substance between adolescent cigarette smoking and alcohol consumption and the subsequent use of hard drugs (1,3–6). Other scholars have provided further support for the gateway theory as it pertains to other drug pathways (7–15).

Studies guided by the gateway theory are not without critics. One critique is that such studies are often derived from population-based samples, which are known to exhibit low prevalence of hard drug use (12,16–17). When examining findings from targeted samples of drug users, results show that hard drug users often do not initiate their trajectory with alcohol use, but with marijuana (18–21). A second critique of studies based on the gateway theory is that most are limited to adolescents. There is little evidence that adolescents who initiate the use of hard drugs necessarily become regular users (16,21). A final critique of the theory is that there is

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

substantial ambiguity over how research findings should be interpreted, with similar findings leading to divergent conclusions (2).

Despite critiques of research guided by gateway theory, the theory is used to set policies and develop educational campaigns that emphasize alcohol and tobacco abstinence to prevent future use of and addiction to hard drugs (19,22). Drug researchers have critiqued such policies for failing to consider the complex underlying causal mechanisms that lead to both hard drug use and subsequent addiction (2,23). Further, such policies inadequately recognize the impact of local drug markets, which to a large extent determine what drugs are available and how they are used (17,24). Finally, policies typically do not respond to period and cohort effects in drug use initiation (20,25). As the drug scene evolves, gateway drugs may change as well (18).

In this paper we focus on ecstasy (3,4-Methylenedioxymethamphetamine or MDMA) and whether it serves as a gateway in the path to other drug use within a sample of young adult ecstasy users. We do not argue that ecstasy use may physiologically cause more serious drug use, however the social context of ecstasy use may set the stage for the subsequent use of other drugs. Within the social context of drug use, ecstasy is largely perceived to be a safe drug, with roughly one-half of adults age 19 to 30 years old seeing little risk in ecstasy (26). In addition, there is a relatively high prevalence of ecstasy use. Among individuals between the ages of 18 and 25, 15.1% reported lifetime ecstasy use, 5.8% reported past year ecstasy use, and 1.1% reported past month ecstasy use (27). Combining the popular perception of the relative safety of ecstasy and the prevalence of its use amongst young adults, the drug may play a “new” role in the sequential use trajectory as described by the gateway theory.

METHODS

Study Procedures

Between September 2002 and April 2004, a cross-sectional survey was conducted of 268 active ecstasy users between the ages of 18 and 25 in Atlanta, Georgia. To be eligible, respondents had to have used ecstasy at least three times in the past 90 days (on separate occasions), not be in drug treatment or another institutional setting, and not be intoxicated or otherwise cognitively impaired at the time of interview. Initial participant recruitment involved the community identification (CID) process, a mapping method to record epidemiological indicators of the prevalence of ecstasy use, expert opinions, and ethnographic information from local researchers (28–29). It is important to note that this sample is not generalizable to the general population or even to a population of active ecstasy users. As has been done in similar studies (30), we concentrated on a specific sample of ecstasy users to reinforce the internal validity of our data. Our subsequent results must be interpreted taking into account the specificity of our sample. Further, since all of our respondents use ecstasy, we cannot establish causality as we may have been able to had our sample included non-users. However, this sample is important in its own right, despite these weaknesses, because drug use trajectories of active ecstasy users may differ from those of experimental or non-users (12).

Using a short form, potential respondents were initially screened in the setting where they were actively recruited such as at clubs and near college dorms. In addition, we actively solicited respondents via referrals. We limited referral chains to two referrals per chain. Further, we limited the number of respondents from any one setting to eight in order to avoid saturation. Passive recruitment, involving the posting of flyers in locales with heavier concentrations of young adults, was also utilized. Passively recruited respondents were initially screened, when they called the phone number listed on the flyers, using the same short form as actively recruited respondents. Prior to each interview, a follow-up screening with a detailed focus on ecstasy use was conducted. Questions were asked about the local price of ecstasy, the local ecstasy taxonomy, and the ecstasy high. Very few individuals failed the second screening.

Individuals who passed both screenings were provided with information on the nature of the study, including details about informed consent and other confidentiality procedures. Data collection involved an interviewer-administered computerized survey. The interviews took place at a mutually convenient location such as one of the project offices or a local coffee shop. The average length of time to complete the survey was two hours. Respondents were reimbursed \$25 for their participation.

Analytic Technique

We use logistic regression of discrete-time models (31–32), to examine the effects of the timing of the onset of ecstasy use on the onset of cocaine, heroin, and methamphetamine use. Discrete-time event history analysis is the most appropriate approach to studying processes in which individuals change from one state to another (e.g. from not having used a given drug to having used that drug), in which change occurs at set intervals (as in this case where respondents report the age, in years, at which they first used each drug), and in which the determinants of change are time-constant and/or time-variant (33–36).

Discrete-time event history models require a unique type of data. The data used in these analyses were collected as a cross-sectional dataset in which each case represents a single individual. To estimate these models using discrete-time analysis, the original data were transformed into a person-year dataset in which each case represents a separate observation of each individual for each year in which they were at risk of having used a given drug. Admittedly using years as a unit of time is a rough interval, however it is often used in social research due to a lack of more discrete measures and to avoid recall problems endemic of retrospectively collected data (37). For each person-year case, a dummy variable, indicating whether or not the individual used a drug in that year, was added, along with a dichotomous event indicator to specify whether the event of interest occurred at the time of the last observation or the case was censored. This event indicator was regressed using logistic regression on the predictor variables and the series of dummy variables in each person-year. Every respondent within our sample either experienced the onset of a given drug or was censored at the time of the survey, within a very small number of possible years. Hence there are many cases that are tied (i.e. multiple subjects report having used a given drug for the first time at the same age). The presence of ties leads to bias in continuous-time models but not discrete-time models (36).

We chose to use discrete-time models in lieu of continuous-time models for two reasons. First discrete-time models can approximate continuous-time models (39). The transition from never using a given drug to having used that drug can occur at any finite point in time. Using retrospective data, however, limits the ability of the respondent to recall the precise timing of their transition from one state to another. The data used in this study were collected so that the respondents indicated their age when they first used a given drug. We, therefore, use discrete-time event history analysis to model a phenomenon that is in reality continuous, but that is measured discretely. Second, continuous-time models assume that an event does not happen at precisely the same time for multiple subjects.

The discrete-time logistic regression model takes the following form:

$$\log_e \left(\frac{h_{ij}}{1 - h_{ij}} \right) = (\alpha_1 D_{1ij} + \alpha_2 D_{2ij} + \dots + \alpha_J D_{Jij}) + (\beta_1 Z_{1ij} + \beta_2 Z_{2ij} + \dots + \beta_J Z_{Jij})$$

where D_{1ij} , D_{2ij} , ..., D_{Jij} represent a series of dummy variables indexing time periods, the intercept parameters (α_1 , α_2 ... α_J) represent the baseline level of hazard in each time period, and the slope parameters (β_1 , β_2 ... β_J) describe the effects of the predictors on the baseline hazard function. This equation predicts the conditional log-odds that a given event (using a certain drug) will occur in a given time period, j . In interpreting the coefficients from discrete-

time survival models, the estimated percentage change in the hazard rate of having experienced a given event for a positive change in each covariate is calculated as: (38)

$$(e^{\beta} - 1) * 100$$

Measures

Dependent Variables—The three dependent variables in this analysis are the risk of using *cocaine*, *methamphetamine*, and *heroin* (i.e. the rate of transition from having never used each drug to having used it). The dependent variable in each model is coded 0 for each year in which a respondent is at risk of using a drug and is censored for those years that the respondent is no longer at risk because the event has already occurred (i.e. they have used the given drug).

Independent Variables—Our key independent variable is a time-dependent measure of *ecstasy use*. We also include measures of the timing of first *alcohol use* and *marijuana use*. We integrate time-varying independent variables into our models via the method of episode splitting (32). Episode splitting is used when covariates change their value at some discrete point in time, such as over the course of a given year as is the case here. We have integrated episode splitting by substituting each original episode with a set of sub-episodes that split whenever the respondent experienced the onset of one of the independent variables. Hence, in our analyses, each time-varying independent variable is coded categorically as onset having occurred or not having occurred, as was the dependent variable (32).

Beyond time-dependent covariates, we include a static measure of self-control to assess differences in propensity for drug use (see Table 1). We measured *low self-control* with a modified version of Grasmick et al.'s (39) often-used scale comprised of twenty questions across five indicators: Impulsivity, preference for simple tasks, tendency toward risk seeking behavior, self-centeredness, and having a short temper (39, see also Gottfredson and Hirschi [40]). The result is an additive scale of the standardized scores of responses coded so that higher values indicate greater levels of low self-control ($KR_{20}=0.80$).

We also include a number of demographic characteristics as control variables. These are *educational level* (in years), *socioeconomic status* (self-identifying as low socioeconomic status coded as one), *gender* (female coded as zero), *age* (in years), and *race/ethnicity* (African American or being some other race/ethnicity, with being white serving as the excluded reference category).

We conducted diagnostic analyses of our data, with special attention to issues of multicollinearity. We find that none of the correlations between the independent variables are problematic (none exceed $r=0.62$). The highest variance inflation factor in our models is 1.95, below the limit of 2.50 suggested by Allison (41).

RESULTS

The onsets of alcohol and marijuana use occur at earlier ages than the onset of any other substance use among our respondents (Table 2). Moreover, in this sample the onset of marijuana use precedes the onset of alcohol use by almost one year. Further, a larger proportion of our respondents reports never having used alcohol (5.6%) than never having used marijuana (1.5%). Although all respondents are active ecstasy users, the age of onset of ecstasy use occurs quite late in the drug use queue. On average, the respondents report using cocaine (17.4 years of age) before ecstasy (17.8 years of age) and first using heroin at the same age as ecstasy. Methamphetamine has the oldest age of onset at 18.2 years of age.

Univariate Kaplan-Meier survival curves for each of the drug use variables show the probability of *not* experiencing the onset of a given behavior at a given age (Figure 1). As expected from the results presented above, the probability of not initiating alcohol and marijuana use appear lower at younger ages than the probability of not using any hard drug, including ecstasy. The probability of having not used either alcohol or marijuana drops below 50% by the age of 15. At that age, the use of all other drugs is just emerging. The probability of having not used ecstasy does not drop below 50% until roughly age 17. This trend is followed by cocaine and methamphetamine at approximate ages of 19 and 22, respectively. Since the vast majority of our sample had not used heroin at the time of the interview, the probability of surviving the onset of heroin use never drops below 50%.

Our multivariate analyses indicate that the estimated risk of using cocaine is influenced by a number of factors (Table 3). Of greatest interest to this study, however, is the use of ecstasy and the so-called gateway substances, alcohol and marijuana. As would be expected from the gateway literature, later onset of alcohol and marijuana is associated with later onset of cocaine use in our sample. For each year that a respondent delays initial use of alcohol, the use of cocaine is delayed by 65% in this sample. Likewise, later onset of marijuana delays the onset of cocaine by use 97%. These results are expected from the literature and from the survival curves presented in Figure 1. However, neither clearly indicated that delaying the onset of ecstasy use might also influence the onset of cocaine use among our respondents. That appears to be the case in these multivariate analyses. For each year later that a respondent initiates ecstasy use, the initiation of cocaine use is delayed by 52%. While the effect is not of the same magnitude as the effects of alcohol and marijuana, ecstasy appears to hold a position in the sequential progression of cocaine use.

Alcohol, marijuana, and ecstasy onset have similar effects on the onset of methamphetamine use (Table 4). For each year that the onset of alcohol is delayed, the risk of using methamphetamine declines by 60% in this sample. For marijuana, each one year delay in onset lessens the risk of methamphetamine use by 97%. Likewise, for each year later that an individual in this sample initiates ecstasy use, the risk of initiating methamphetamine use declines by 57%. As was the case for the initiation of cocaine use, the initiation of methamphetamine use appears to be influenced by ecstasy.

Alcohol, marijuana, and ecstasy use do not have the same effects on heroin use as they did on cocaine and methamphetamine use among our respondents (Table 5). In fact, only marijuana use has a significant effect on heroin use. For each year older an individual is before trying marijuana, their risk of using heroin declines by 93%. Neither the onset of ecstasy use or the onset of alcohol use has a significant effect on the onset of heroin use.

DISCUSSION AND CONCLUSIONS

In this study, we explored if ecstasy may occupy a position between alcohol/marijuana use and the use of other hard drugs in the developmental trajectory of drug use. We explored this proposition by using discrete-time hazard models to examine how the timing of initial onset of ecstasy use influenced the onset of other hard drugs in a unique sample of active ecstasy users.

Very little previous research has explored ecstasy's role in the initiation of other drugs. And to our knowledge no other research has specifically examined the effects of the age of onset of ecstasy use on the age of onset of other hard drugs. Therefore our findings regarding ecstasy are revealing. The analyses indicate that ecstasy may have an influence on the use of other hard drugs, specifically cocaine and methamphetamine. While those in our sample of active ecstasy users tend to initiate ecstasy at a later age than many other substances, once key variables are

controlled for, it appears that initiating ecstasy at a later age may lower the risk of initiating the use of cocaine and methamphetamine, although not heroin.

There are a number of reasons to expect that ecstasy use may trigger the initiation of other hard drug use. While not without risk, ecstasy tends to be perceived by drug users as less risky than other hard drugs (42). Because perception of risk influences drug use (43), individuals may be more likely to try ecstasy. In the early years of ecstasy, use tended to be restricted to a rave-scene that was purist in nature with participants restricting the use of drugs to ecstasy (44). However, in recent years, ecstasy use has expanded far beyond this specific drug culture (45). The expanded use of ecstasy may have lessened its potential protection against the use of other, more stigmatized, hard drugs.

Further, the current use of ecstasy in a poly-drug culture may have increased the exposure of ecstasy users to other hard drugs (46). Users may begin to use ecstasy because they perceive it as having less risk than other hard drugs. However the initiation of ecstasy use may also be an initiation into a poly-drug culture where the availability of other hard drugs is greater and the use such drugs is normalized. In other words, individuals begin to use ecstasy because they perceive it to be low-risk, but they then put themselves at risk of using other drugs because they have now entered an environment where poly-drug use is common, and an environment where ecstasy may be adulterated with a variety of other substances unbeknownst to the user.

It is important to note that our research simply establishes the existence of temporal trajectories from ecstasy use to the use of other drugs. Our research cannot explicate the causal mechanisms by which this occurs. The ideas we present as to how ecstasy may operate as a gateway to the use of other drugs are offered simply for discussion. We leave to other research the task of identifying the causal mechanisms that underpin this drug sequence. We also leave to other research the task of exploring how ecstasy operates as part of the temporal progression of drug use within other populations. Our research is based upon a unique sample of young adult, active ecstasy users. For this reason our results cannot be generalized to other drug users or to the population in general. Ecstasy may not operate in the same way in the general population where there are a greater number of people who may have experimented with ecstasy, but would not be considered active ecstasy users. Finally, our sample was restricted to those ages 25 or younger. It may be that some of our respondents initiated the use of certain drugs at some later point in life outside the bounds of this study.

In summary, this research suggests that the sequential progression of drug use proposed in the gateway literature is not immutable. As different drugs rise and fall in popularity, it is important to revisit the patterns of initiation of substance use. Policy and prevention efforts based on drug sequences identified in the literature of the past may be misidentifying the typical progression of those initiating drug use in the contemporary context. Hence the initiator role of ecstasy and more recent entries into youth drug cultures, such as methamphetamine, must be investigated further.

Acknowledgements

This research was supported by the National Institute on Drug Abuse (RO1DA-14232, PI: Sterk) and the Emory Center for AIDS Research. The views presented in this paper are those of the authors and do not represent those of the funding agency. We thank Miriam W. Boeri for the coordination of the data collection and all the field staff and the participants who made this study possible.

References

1. Kandel DB. Stages in Adolescent Involvement in Drug Use. *Science* 1975;190:912–914. [PubMed: 1188374]

2. MacCoun, R. In What Sense (If Any) Is Marijuana A Gateway Drug? The FAS Drug Policy Analysis Bulletin 1998. [accessed May 11, 2006]. <http://www.fas.org/drugs/issue4.htm#gateway>
3. Kandel DB, Faust R. Sequence and Stages in Patterns of Adolescent Drug Use. *Archives of General Psychiatry* 1975;32:923–932. [PubMed: 1156108]
4. Kandel DB, Kessler RC, Margulies RZ. Antecedents of Adolescent Initiation into Stages of Drug Use: A Developmental Analysis. *Journal of Youth and Adolescence* 1978;7:13–39.
5. Yamaguchi K, Kandel DB. Patterns of Drug Use from Adolescence to Young Adulthood: II. Sequences of Progression. *American Journal of Public Health* 1984a;74:668–672. [PubMed: 6742252]
6. Yamaguchi K, Kandel DB. Patterns of Drug Use from Adolescence to Young Adulthood: III. Predictors of Progression. *American Journal of Public Health* 1984b;74:673–681. [PubMed: 6742253]
7. Kane RJ, Yacoubian GS. Patterns of Drug Escalation Among Philadelphia Arrestees: An Assessment of the Gateway Theory. *Journal of Drug Issues* 1999;29:107–120.
8. Stenbacka M, Allebeck P, Robelsjo A. Initiation into Drug Use: The Pathway from Being Offered Drugs to Trying Cannabis and Progression to Intravenous Drug Abuse. *Scandinavian Journal of Social Medicine* 1993;21:31–39. [PubMed: 8469942]
9. Werch CE, Anzalone D. Stage Theory and Research on Tobacco, Alcohol, and Other Drug Use. *Journal of Drug Education* 1995;25:81–98. [PubMed: 7658298]
10. Fergusson DM, Horwood LJ. Does Cannabis Use Encourage Other Forms of Illicit Drug Use? *Addiction* 2000;95:505–520. [PubMed: 10829327]
11. Lo CC. Timing of Drinking Initiation: A Trend Study Predicting Drug Use among High School Seniors. *Journal of Drug Issues* 2000;30:525–554.
12. Mackesy-Amiti ME, Fedrich M, Goldstein PJ. Sequence of Drug Use among Serious Drug Users: Typical versus Atypical Progression. *Drug and Alcohol Dependence* 1997;45:185–196. [PubMed: 9179520]
13. Abdel-Ghany M. Contemporaneous and Intertemporal Relationship Between the Consumption of Licit and Illicit Substances by Youth. *Journal of Family and Economic Issues* 2003;24:281–289.
14. Best D, Rawaf S, Rowley J, Floyd K, Manning V, Strang J. Drinking and Smoking as Concurrent Predictors of Illicit Drug Use and Positive Drug Attitudes in Adolescence. *Drug and Alcohol Dependence* 2000;60:319–321. [PubMed: 11053768]
15. Yu J, Williford WR. The Age of Alcohol Onset and Alcohol, Cigarette, and Marijuana Use Patterns: An Analysis of Drug Use Progression of Young Adults in New York State. *The International Journal of Addictions* 1992;27:1313–1323.
16. Golub A, Johnson BD. The Shifting Importance of Alcohol and Marijuana as Gateway Substances among Serious Drug Abusers. *Journal of Studies on Alcohol* 1993;55:607–614. [PubMed: 7990471]
17. Sterk, C. *Fast Lives: Women Who Use Crack Cocaine*. Philadelphia, PA: Temple University Press; 1999.
18. Ginzler JA, Cochran BN, Domenech-Rodriguez M, Cause AM, Whitbeck LB. Sequential Progression of Substance Use Among Homeless Youth: An Empirical Investigation of Gateway Theory. *Substance Use and Misuse* 2003;38:725–758. [PubMed: 12747403]
19. Golub A, Johnson BD. The Misuse of the ‘Gateway Theory’ in US Policy on Drug Abuse Control: A Secondary Analysis of the Muddled Deduction. *International Journal of Drug Policy* 2002;13:5–19.
20. Golub A, Johnson BD. Cohort Differences in Drug-Use Pathways to Crack among Current Crack Abusers in New York City. *Criminal Justice and Behavior* 1994;21:403–422.
21. Labouvie E, Bates ME, Pandina RJ. Age of First Use: Its Reliability and Predictive Utility. *Journal of Studies on Alcohol* 1996;58:638–643. [PubMed: 9391924]
22. Golub A, Johnson BD. Alcohol is not the Gateway to Hard Drug Abuse. *Journal of Drug Issues* 1998;28:971–985.
23. Kenkel D, Matios AD, Pacula RL. Economics of Youth Drug Use, Addiction, and Gateway Effects. *Addiction* 2001;96:151–164. [PubMed: 11177526]
24. Sterk-Elifson C, Elifson K. The Social Organization of Crack Cocaine Use: The Cycle in One Type of Base House. *Journal of Drug Issues* 1993;23:429–441.

25. Boeri MW, Sterk CE, Elifson KW. Babyboomer Drug Users: Career Phases, Social Control and Social Learning Theory. *Sociological Inquiry* 2006;28:264–291.
26. Johnston, L.; O'Malley, P.; Bachman, J. Monitoring the Future National Survey Results on Adolescent Drug use: Overview of Key Findings. Bethesda, MD: National Institute on Drug Abuse; 2003.
27. Substance Abuse and Mental Health Services Administration (SAMHSA). Results from the 2002 National Survey on Drug Use and Health: National Findings. Rockville, MD: SAMHSA; 2003.
28. Tashima N, Crain C, O'Reilly K, Sterk-Elifson C. The Community Identification (CID) Process: A Discovery Model. *Qualitative Health Research* 1996;6:23–48.
29. Watters JK, Biernacki P. Targeted Sampling: Options for the Study of Hidden Populations. *Social Problems* 1989;36:416–430.
30. Sommers I, Baskin D. Methamphetamine Use and Violence. *Journal of Drug Issues* 2006;36:77–96.
31. Allison, PD. Event History Analysis: Regression for Longitudinal Event Data. Newbury Park: Sage; 1984.
32. Rohwer, G.; Potter, U. TDA User's Manual. Bochum, Germany: Ruhr University, Bochum; 1998.
33. Coleman, J. Longitudinal Data Analysis. New York: Basic Books; 1981.
34. Guilkey DK, Rindfuss RR. Logistic Regression Multivariate Life Tables: A Communicable Approach. *Sociological Research and Methods* 1987;16:276–300.
35. Singer JD, Willett JB. It's About Time: Using Discrete-Time Survival Analysis to Study Duration and the Timing of Events. *Journal of Educational Statistics* 1993;18:155–195.
36. Yamaguchi, K. Event History Analysis. Newbury Park: Sage; 1991.
37. Bradburn, NM. Response Effects. In: Rossi, PH.; Wright, JD.; Anderson, AB., editors. *Handbook of Survey Research*. New York: Academic Press; 1983. p. 289–328.
38. Teachman JD, Hayward MD. Interpreting Hazard Rate Models. *Sociological Methods and Research* 1993;21:340–371.
39. Grasmick HG, Tittle CR, Bursik RJ, Arneklev BJ. Testing the Core Implications of Gottfredson and Hirschi's General Theory of Crime. *Journal of Research in Crime and Delinquency* 1993;1993;30:5–29.
40. Gottfredson, M.; Hirschi, T. A General Theory of Crime. Palo Alto: Stanford University Press; 1990. p. 10
41. Allison, PD. Multiple Regression. Thousand Oaks, CA: Pine Forge Press; 1998.
42. Solowij N, Hall W, Lee N. Recreational MDMA Use in Sydney: A Profile of 'Ecstasy' Users and their Experiences with the Drug. *British Journal of Addiction* 1992;87:1161–1172. [PubMed: 1354992]
43. Shewan D, Dalgarno P, Reith G. Perceived Risk and Risk Reduction among Ecstasy Users: The Role of Drug, Set, and Setting. *International Journal of Drug Policy* 2000;10:431–453.
44. Collin, M. *Altered State: The Story of Ecstasy Culture and Acid House*. London: Serpent's Tail; 1998.
45. Boeri MW, Sterk CE, Elifson KW. Rolling Beyond the Raves: Ecstasy Use Outside the Rave Setting. *Journal of Drug Issues* 2004;34:831–860.
46. Sterk C, Theall K, Elifson K. Young Adult Ecstasy Use Patterns: Quantities and Combinations. *Journal of Drug Issues* 2006;36:201–228.

LIST OF ABBREVIATIONS

MDMA

3,4-Methylenedioxymethamphetamine, ecstasy

TDA

Transition Data Analysis

Figure 1.
Survival Probability of Initiation of Substance Use

Table 1

Exogenous Variable Characteristics

	Mean or %
Low Self-Control	0.0
Educational Attainment (in years)	12.3
Socioeconomic Status (% self-reported as low status)	33.2
Gender	
Female (%)	30.6
Male (%)	69.4
Age	20.8
Ethnic/Racial Background	
White (%)	50.4
Black (%)	35.8
Other Ethnicity (%)	13.8

n=268

Table 2

Onset and Prevalence of Substance Use

Variable	Minimum	Maximum	Mean Age at Onset	% No Onset
Alcohol	8	22	15.3	5.6
Marijuana	7	25	14.6	1.5
Ecstasy	11	25	17.8	0.0
Cocaine (any form)	12	24	17.4	37.3
Methamphetamine	12	24	18.2	50.7
Heroin	14	24	17.8	84.3

n=268

Table 3

Logistic Coefficients from the Regression of the Timing of First Cocaine Use on Selected Independent Variables

Variable	Coefficient	Standard Error	% Change in Hazard Rate from Change in Variable
Alcohol	-1.046***	0.240	-64.866
Marijuana	-3.393***	0.362	-96.639
Ecstasy	-0.735***	0.191	-52.050
Low Self-Control	0.006	0.010	0.602
Education	-0.133*	0.058	-12.454
Low Socioeconomic Status	0.189	0.187	20.804
Gender	0.340	0.203	40.495
Age	-0.041	0.044	-4.017
Black	-1.228***	0.211	-70.712
Other Ethnicity	-0.089	0.252	-8.515
Constant	1.528	---	---
Cases	268	---	---
Number of person-years	803	---	---
Chi-square	507.379	---	---

*
 $p < .05$;**
 $p < .01$;***
 $p < .001$

Table 4

Logistic Coefficients from the Regression of the Timing of First Methamphetamine Use on Selected Independent Variables

Variable	Coefficient	Standard Error	% Change in Hazard Rate from Change in Variable
Alcohol	-0.909**	0.302	-59.707
Marijuana	-3.664***	0.501	-97.437
Ecstasy	-0.840***	0.208	-56.829
Low Self-Control	-0.009	0.011	-0.896
Education	-0.049	0.061	-4.782
Low Socioeconomic Status	0.446*	0.213	56.205
Gender	0.308	0.229	36.070
Age	-0.143**	0.048	-13.325
Black	-2.648***	0.359	-92.921
Other Ethnicity	-0.629	0.274	-46.688
Constant	2.256	---	---
Cases	268	---	---
Number of person-years	859	---	---
Chi-square	461.330	---	---

*
 $p < .05$;

**
 $p < .01$;

***
 $p < .001$

Table 5
Logistic Coefficients from the Regression of the Timing of First Heroin Use on Selected Independent Variables

Variable	Coefficient	Standard Error	% Change in Hazard Rate from Change in Variable
Alcohol	-0.766	0.546	-53.513
Marijuana	-2.638***	0.715	-92.850
Ecstasy	-0.676	0.358	-49.135
Low Socioeconomic Status	0.317	0.339	37.300
Education	-0.167	0.094	-15.380
Low Self-Control	0.317	0.339	37.300
Gender	-0.283	0.363	-24.648
Age	0.016	0.072	1.613
Black	-2.283***	0.609	-89.802
Other Ethnicity	-1.288*	0.616	-72.418
Constant	-0.863	---	---
Cases	268	---	---
Number of person-years	903	---	---
Chi-square	121.328	---	---

*
 $p < .05$;

**
 $p < .01$;

***
 $p < .001$