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# Examining the Gateway Hypothesis Stages and Pathways of Drug Involvement

Denise B. Kandel

The notion that there are developmental stages and sequences of involvement in drugs was first advanced a quarter of a century ago (Hamburg, Kraemer, & Jahnke, 1975; Kandel, 1975). According to this notion, there is a progressive and hierarchical sequence of stages of drug use that begins with tobacco or alcohol, two classes of drugs that are legal, and proceeds to marijuana, and from marijuana to other illicit drugs, such as cocaine, metamphetamines, and heroin. The basic premise of the developmental stage hypothesis is that involvement in various classes of drugs is not opportunistic but follows definite pathways; an individual who participates in one drug behavior is at risk of progressing to another. The notion of developmental stages in drug behavior does not imply, however, that these stages are either obligatory or universal, nor that all persons must progress through each in turn.

In the early 1980s, the term *Gateway drug* began to be used to refer to alcohol and cigarettes, the drugs that are used prior to the use of illicit drugs. Soon, the usage was extended to include the use of marijuana as a precursor to the use of other illicit drugs, such as cocaine or heroin, and more rarely even to cocaine as a precursor to heroin. It is not clear how the term *Gateway drug* originated and I could not locate any relevant literature. Robert Dupont seems to have coined this term when he was director of the National Institute on Drug Abuse, and Dupont certainly was responsible for later popularizing its use (Dupont, 1984) and for connecting it with the stage hypothesis. The Gateway Hypothesis is implicit in the Gateway term and in the concept that certain drugs serve as gateways for the use of other drugs.



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Validity of the Gateway Hypothesis is based on two criteria: sequencing of initiation of use between drug classes, and association in the use of drugs, such that use of a drug lower in the sequence increases the risk of using drugs higher in the sequence. Ultimately, association implies causation if all possibilities for spurious associations have been eliminated. Given the difficulties of establishing true causality in the social sciences, the term *association* rather than *causation* is emphasized in most of the chapters. Because of the theoretical and policy implications of the Gateway Hypothesis for understanding adolescent development and the formulation of prevention and intervention programs, a critical examination of the hypothesis is warranted. That is the purpose of the volume.

Although the Gateway Hypothesis had its origins in the mid-1970s, the concept of progression in drug use has a long history. It was first promulgated in the 1930s as the Stepping-Stone Theory (Etz, Robertson, Glanz, & Colliver, 1999; Goode, 1974). However, there is a crucial difference between the two concepts. The Stepping-Stone Theory considered the progression in drug involvement to be inexorable, with the use of marijuana invariably leading to heroin addiction. This position derived from an erroneous interpretation of data from clinical samples of treated heroin addicts. In these samples, most clients reported that the first drug they used prior to heroin was marijuana. From these selected samples, the investigators concluded that all those who used marijuana would eventually also use heroin. The subsequent prospective naturalistic studies of general population samples documented that in fact this was not the case. The Gateway Hypothesis, which emerged from this modern epidemiological tradition, emphasizes that although the use of certain drugs precedes the use of other classes, progression is not inevitable. Rather, the phases in drug behavior are facilitative. Entry into a particular stage is a common and perhaps even a necessary step but is not a sufficient prerequisite for entry into the next higher stage. Many youths stop at a particular stage without progressing further. In 1998, over 90% of young adults 20 to 30 years old in the United States had followed the assumed sequence. Of those who had used alcohol or tobacco, 49% proceeded to use one or more illicit drugs. Although steps in regression have been little studied, one early study reported that the same steps appeared in regression as in progression (Kandel & Faust, 1975).

Whereas much work has been done to identify the stages themselves, much less work has been carried out to identify the risk and protective factors underlying progression from one drug to another or the factors



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responsible for progression to heavier use of the same drug. A full accounting of developmental processes regarding drug use would also need to consider the place of drug use among a wider range of other problem behaviors in adolescence, including violence, delinquency, early sexual experimentation, premarital pregnancy, depression, suicide, dropping out of school, eating disorders, and even poor driving (see Dryfoos, 1990). These developmental progressions are not dealt with in this volume. The primary focus here is on progression through the use of different classes of drugs.

Numerous cross-sectional and longitudinal investigations have documented regular sequences of progression from legal to illegal drugs among adolescents and young adults of both sexes, irrespective of the age of first initiation into drugs, among different ethnic groups, in different countries, and at different historical periods spanning a 20-year interval. (For a review, see Kandel & Yamaguchi, 1999.) The sequence has been observed in countries other than the United States: in France, Israel, Australia, Japan, Spain, and Scotland (Adler & Kandel, 1981; Blaze-Temple & Lo, 1992; Oh, Yamazaki, & Kawata, 1998; Adrados, 1995; Morrison & Plant, 1991).

One group of cross-sectional studies does not take temporal order into account. An order has been inferred in the absence of information about timing of the onset of drug use. Evidence includes cross-sectional analyses based on Guttman scaling, a method that identifies unidimensional cumulative features of attributes (Adler & Kandel, 1981; Blaze-Temple & Lo, 1992; Brook, Hamburg, Balka, & Wynn, 1992; Brook, Whiteman, & Gordon, 1983; Donovan & Jessor, 1983; Kandel, 1975; Merrill, Kleber, Schwartz, Liv, & Lewis, 1999; Mills & Noyes, 1984; Single, Kandel, & Faust, 1974; Sorenson & Brownfield, 1989; Welte & Barnes, 1985; Yu & Williford, 1992). Other evidence derives from structural equation models based on cross-sectional data (Free, 1993; Hays et al., 1986; 1987; Huba & Bentler, 1982; Huba, Wingard, & Bentler, 1981; Marcos & Bahr, 1995; Martin, 1982; Potvin & Lee, 1980; Windle, Barnes, & Welte, 1989); latent class models (Sorenson & Brownfield, 1989); and log linear models (Miller, 1994). Historically, Guttman scaling has been the most frequently used method. Guttman scaling based on cross-sectional patterns of use reflects a hierarchical and unidimensional order of use, which, in the absence of age of onset or longitudinal data, does not necessarily reflect an order of initiation.

A second group of studies documents an order by taking timing into account. These include studies based on age of initiation of the use of



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various classes of drugs (Fleming, Leventhal, Glynn, & Ershler, 1989; Golub & Johnson, 1994; Hamburg, Kraemer, & Jahnke, 1975; Johnson, 1973; O'Donnell & Clayton, 1982; Voss & Clayton, 1987; Welte & Barnes, 1985); cross-tabulations of use patterns at different point in time (Elliott, Huizinga, & Menard, 1989); modified log linear Guttman scale analysis (Ellickson, Hays, & Bell, 1992; Gould, Berberian, Kasl, Thompson, & Kleber, 1977); path or structural equation models of panel data (e.g., Kaplan, Martin, & Robbins, 1984; Osgood, Johnston, O'Malley, & Bachman, 1988); latent transition analysis (Collins, Graham, Long, & Hansen, 1994; Collins & Wugalter, 1992; Graham, Collins, Wugalter, Chung, & Hansen, 1991); event history and parametric event sequence analysis (Yamaguchi & Kandel, 1984a; 1996); and extensions of log linear models (Kandel, Yamaguchi, & Chen, 1992). Some investigators have combined an initial Guttman scale analysis of crosssectional data with an examination of patterns of subsequent transitions (Andrews, Hops, Ary, Lichtenstein, & Tildesley, 1991; Fleming, Leventhal, Glynn, & Ershler, 1989; Kandel & Faust, 1975).

Although illicit drugs other than marijuana are usually aggregated in a single group, several investigators have examined the order among specific illicit drugs other than marijuana (Ellickson, Hays, & Bell, 1992; Newcomb & Bentler, 1986; Mills & Noves, 1984; Single, Kandel, & Faust, 1974; Sorenson & Brownfield, 1989; Welte & Barnes, 1985). Distinctions are usually made among pills (e.g., barbiturates, stimulants), cocaine, hallucinogens, and heroin. The greater the number of drugs to be ordered, the poorer the fit. There is much similarity across studies in the order: marijuana, pills, cocaine, heroin. The position of hallucinogens is the most unstable; it preceded cocaine in the New York state sample studied in 1971 (Single, Kandel, & Faust, 1974) but followed cocaine in samples of Maryland high school students surveyed in 1978-1981 (Mills & Noyes, 1984) or White males surveyed in Seattle in 1980 (Mills & Noyes, 1984). Sorenson and Brownfield (1989), who conducted systematic analyses based on Guttman scaling and latent structure analyses, found that the most unscalable scale types resulted from an assumed order between cocaine and hallucinogens. Heroin was not included among the drugs scaled. Havs et al. (1987) and Windle, Barnes, and Welte (1989) differentiated illicit drugs other than marijuana into enhancers and dampeners but could not successfully identify a sequential order for these two classes.

Sequences of involvement can also be distinguished within specific classes of drugs, involving shifts from initiation, to experimentation,



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casual use, regular use, abuse, and dependence (Clayton, 1992; Glantz & Pickens, 1992; Werch & Anzalone, 1995). With the exception of early discussions of heroin (Robins, 1979), most of this work has focused on cigarette smoking (e.g., Chassin, Presson, & Sherman, 1984; Flay, 1993; Leventhal & Cleary, 1980; Werch & Anzalone, 1995) and alcohol (Tarter & Vanyukov, 1994; Zucker, Fitzgerald, & Moses, 1995). Stages of involvement within a particular drug class may provide important specifications of progressions across classes of drugs. For example, Donovan and Jessor (1983) identified problem drinking as intervening between the use of marijuana and the use of other illicit drugs. Similarly, Collins and her colleagues (Collins, 1991; Collins et al., 1994; Graham et al., 1991) emphasized the role of drunkenness as a stage between the use of alcohol and tobacco and advanced drug use. In general, increasing involvement within a particular drug class precedes progression to the next stage (Ellickson, Hays, & Bell, 1992; Kandel & Faust, 1975; Yamaguchi & Kandel, 1984b).

# Issues Underlying the Gateway Hypothesis

There has been a resurgence of interest in the Gateway Hypothesis as a framework for understanding adolescent drug involvement. This interest has been fueled, in part, by the national concern with adolescent smoking, by the role of tobacco in the progression into drug use, by the increase in the use of marijuana by adolescents during the last decade, and by recent reports pointing to a potentially common biological basis underlying addiction to different substances. However, the Gateway Hypothesis is not universally accepted. Showing that there is a sequence of initiation is not the same as showing that there is a causal link in the use of different drugs. Furthermore, as noted earlier, many users of a particular drug do not progress to the use of other drugs.

To evaluate properly the Gateway Hypothesis, several important issues need to be resolved. These include not only conceptual issues but also issues of substance, methods, underlying mechanisms, and policy.

The notion of a Gateway drug itself is vague. What makes a drug a Gateway drug? Is it any lower-ranked drug whose use precedes the use of a higher-ranked substance? As noted earlier, the most common hierarchy assigns alcohol and cigarettes to the initial stage, followed by marijuana, and by other illicit drugs; an order among illicit drugs other than marijuana has been difficult to establish. Even this general ordering has been challenged. Furthermore, the basis underlying any obtained ordering remains to be elucidated. Is the ordering the result of cultural



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definitions for normative behavior, psychological traits related to deviancy, differential availability, or the commonality of biological processes underlying addictive behaviors? Should one not refer to "Gateway use" of a drug rather than to a "Gateway drug"?

A fundamental issue pertains to the nature of the sequences themselves. What specific developmental stages and pathways of drug involvement can be identified currently among young people in the United States? Does the basic sequence from legal to illegal drugs characterize the general population and subgroups within it? Are there regular patterns of regression as well as progression? How do these pathways vary for different subgroups in the population, especially those distinguished by age, gender, or ethnicity, and for general population versus selected samples of heavy drug users? Have there been historical changes in the patterns? Are there identifiable pathways across drug use and other adolescent behaviors?

Another crucial issue pertains to the causal role of the use of one drug on initiation of another. The existence of sequential stages of progression does not necessarily imply causal linkages among different drugs. The observed sequences could simply reflect the association of each class of drugs with different ages of onset or individual attributes rather than the specific effects of the use of one class of drug on the use of another.

The evidence that addresses these questions derives mainly from naturalistic observational studies of a population. Two additional sources of data have been little utilized: programs implemented among youth to prevent or reduce drug use and animal experiments in which the drug seeking and behaviors of animals primed with specific drug classes can be observed. The implementation of drug prevention programs provides an imperfect substitution for an unrealizable social experiment in which adolescents would be randomly assigned to initiate the use of different drugs. Much can be learned, however, from prevention programs that have attempted to delay onset of the use of specific drug classes and to reduce levels of use among those who start. To test the Gateway Hypothesis, such programs would ideally focus on single drug classes to monitor more precisely the effect of the program on progression to higher-stage drugs. As we will see, in reality, very few such programs have been implemented. Most target several drug classes simultaneously. Animal models provide a test of progression in which the drug seeking of animals can be observed in relation to well defined prior experiences with specific drugs and independently of any social, legal, or moral definitions regarding the use of various substances.



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Another set of issues pertains to the explanation of progression among classes of drugs and a specification of the risk and protective factors that predict transitions through the pathways. What are the biological, psychological, interpersonal, community, and cultural factors that promote or constrain progression? How do these factors vary by users' characteristics and by stage of drug use? Can one develop animal models to examine the nature of sequences and the predictors of progression, including factors that promote and those that inhibit progression? What explanatory insights can be provided by the new understanding of the biology of addiction?

There are methodological issues. How good is the statistical evidence for the existence of pathways and predictors of progression or regression? The identification of pathways and predictors of progression depends on the adequacy of the statistical evidence. The methodological approaches best suited to identify potential pathways, their risk and protective factors, the advantages and disadvantages of alternate approaches need to be evaluated. True sequential patterns need to be distinguished from chance occurrences. Association and causation need to be documented in addition to sequencing. Indeed, timing and sequence are not sufficient criteria for establishing developmental linkages. It is necessary to assess the extent to which the use of a drug at a particular stage actually determines initiation of the use of a drug at a higher stage.

There are also important policy issues. One set of issues pertains to the implications of findings from research on the Gateway Hypothesis for the development and implementation of prevention and treatment programs. The Gateway Hypothesis provides a useful organizing framework around which to develop specific theories of initiation, progression, and regression in drug use and specific intervention strategies to deal with the various stages of participation in drug behaviors. The hypothesis provides for an optimal specification of the periods in the life span when intervention efforts should be initiated, the types of drugs to be targeted, and the individuals who should be the target of the intervention. Ultimately it will help identify the characteristics of population groups at a particular stage who are most at risk for progression to the next stage or stages. By isolating populations at risk for progression from one drug stage to the next, it becomes possible to identify more accurately the factors that impact on those transitions.

Another set of policy issues pertains to the research that needs to be carried out to advance knowledge in the field. The work reported in this volume makes it clear that a transdisciplinary approach that cuts across



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the traditional disciplines of sociology, psychology, epidemiology, and biology will be essential for further advances in the field. The study of drug abuse in general and of the Gateway Hypothesis in particular provides unique opportunities for bridging meaningfully the social and psychological influences on drug seeking behavior with the molecular underpinnings of the reward system of the brain.

# Organization of the Volume

The volume is organized into six parts. Following an overview in Part I, Part II presents empirical evidence regarding developmental pathways of drug involvement in different groups in the United States and selected risk and protective factors underlying the observed progressions. Several studies are based on prospective longitudinal follow-ups (Labouvie & White, Chapter 2; Hawkins, Hill, Guo, & Battin-Pearson, Chapter 3); others are based on retrospective reports of ages of onset into various substances (Kandel & Yamaguchi, Chapter 4; Golub & Johnson, Chapter 5). The prospective studies are based on adolescents; the retrospective studies are based on adults. Golub and Johnson's study is unusual because it deals with a group of hard core users and attempts to identify a comparison group of minority subjects drawn from national surveys.

Part III documents the impact of drug prevention programs on youths' subsequent drug progressions. These programs include a school-based prevention intervention targeting tobacco, alcohol, and marijuana in urban schools, based on the Life Skills program, now widely used throughout the United States (Botvin, Griffin, & Scheier, Chapter 6); an ambitious community wide effort that engages the efforts of relevant community groups and institutions, including parents, schools, mass media, local groups, and health policymakers (Pentz & Li, Chapter 7); and a school-based intervention specifically targeted to smoking in eight western communities in the United States (Biglan & Smolkowski, Chapter 8). This is the first time that drug prevention programs have examined the impact they have had on drug progression and the relevance of the intervention for evaluating the Gateway Hypothesis.

Part IV presents descriptions and applications of three major statistical approaches to the study of developmental pathways, including log linear methods (Yamaguchi & Kandel, Chapter 9); a modified version of structural equation modeling (Bentler, Newcomb, & Zimmerman, Chapter 10); and latent transition analysis (Collins, Chapter 11). These



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chapters discuss specific methodological approaches and present important substantive findings. In the last chapter in this section, Yamaguchi (Chapter 12) provides an integrated discussion of the advantages and disadvantages provided by each approach; he identifies aspects of the Gateway Hypothesis that each method can best illumi-

Part V focuses on biological approaches. Two of the chapters describe work based on animal experiments and discuss animal models of relevance to the Gateway Hypothesis (Grunberg & Faraday, Chapter 13; Schenk, Chapter 14). Koob (Chapter 15) summarizes what is presently known about the molecular biology of drug behavior and addiction.

nate and aspects that each is less suited for.

In Part VI, in a concluding chapter (Chapter 16), Kandel and Jessor integrate the major findings presented in the volume, emphasize what is known and what remains to be learned, and suggest further lines of inquiry.

A unique contribution of this volume is the juxtaposition of epidemiological, intervention, animal, and neurobiological studies. This juxtaposition represents a new stage in the evolution of drug research, an evolution in which epidemiology and biology inform one another in the understanding of drug abuse.

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