Stages and Pathways of Drug Involvement

Examining the Gateway Hypothesis

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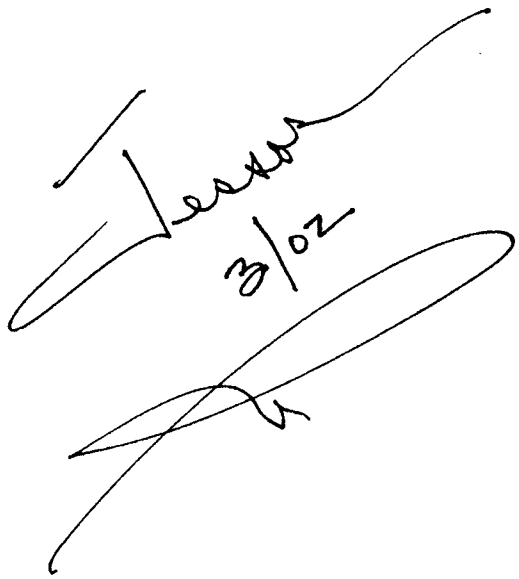

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The Gateway Hypothesis Revisited

Denise B. Kandel and Richard Jessor

The chapters in this volume present what is currently known about the Gateway Hypothesis. Their authors have explored the hypothesis from various perspectives ranging from developmental social psychology to prevention and intervention science, and from animal models and neurobiology to analytical methodology. The presentations have elaborated the nature and extent of the empirical support for the Gateway Hypothesis at this point in time. They have also revealed the complexities embedded in the formulation of the Gateway Hypothesis. In this brief Afterword, we revisit the hypothesis in light of what our colleagues have reported, and we discuss several conclusions that derive from their presentations.

Parsing the Gateway Hypothesis

Advancing understanding of the Gateway Hypothesis requires that it be parsed into three interrelated, component propositions. All three have emerged from the research and the reviews reported in the preceding chapters.

The first proposition embedded in the Gateway Hypothesis is that among adolescents *there is a developmental sequence of involvement with different classes or categories of drugs*, such that initiation into drug use begins with the drugs earlier in the sequence, namely, the legal drugs, alcohol and tobacco. Involvement with illicit drugs occurs later in the developmental sequence; and marijuana is the bridge in the sequence between the licit and the other illicit drugs. Although there are further

refinements to be made about the sequence of drug progression (especially about differentiation within the licit and illicit categories of drugs), the central assertion of the sequence proposition is that drug progression among adolescents proceeds sequentially, or in ordered stages, from licit drugs through marijuana to other illicit drugs. The evidence for this proposition and this particular sequence is quite robust across the last two decades in American society. There is also supporting evidence from other Western nations.

The second proposition embedded in the Gateway Hypothesis is that *the use of a drug earlier in the sequence is associated with an increased risk or likelihood of use of a drug later in the sequence.* The evidence in support of the proposition about association is also strong and derives both from etiological and prevention/intervention studies, including those presented in this volume.

The third proposition encompassed by the Gateway Hypothesis is that *the use of a drug earlier in the sequence, such as alcohol or tobacco, causes the use of a drug later in the sequence, for instance, marijuana.* This is the Gateway Hypothesis proposition that is most widely invoked in public discourse and in policy debates. Even among interventionists, it is often part of the rationale for a focus on programs to prevent initiation of drugs early in the sequence. The research reported in this volume and that reviewed in the various chapters provide no support for the proposition about causality. There is no compelling evidence that the use of a drug earlier in the sequence, in and of itself, causes the use of a drug later in the sequence or, for that matter, that it causes the use of any other drug or, indeed, any other behavior. The difficulty of establishing causality is, of course, intrinsic to much of social science. Making a causal claim is difficult in the absence of carefully controlled experimental designs. Even more important, perhaps, is the difficulty of ruling out alternative inferences that can explain the association between the initiation of an earlier drug and the initiation of a later drug in the developmental sequence. At this time, the causal interpretation of the Gateway Hypothesis is still without scientific foundation.

In the discussion that follows, we examine each of the three propositions in light of what has been learned from the chapters in this volume. Our discussion is framed within a perspective about adolescent development as a whole, a perspective in which drug initiation and progression must be seen as only one aspect of the larger process of development and change in both person and social context that characterizes this period of the life course. A full understanding of drug use in the life of an adolescent can only be achieved by understanding as well the rest of the adolescent's life and the everyday social world in which the adolescent is situated.

The Sequence Proposition

As noted earlier, the evidence for a prototypical developmental sequence of drug use from alcohol or tobacco, to marijuana, to other illicit drugs is well established in the United States and in other selected countries. From the larger perspective on adolescent development, a developmental sequence within a domain of behavior is not unusual and is even to be expected. Sexual development, for example, has well established developmental staging from autoerotic initiation, to so-called "necking" to "petting above the waist," to "petting below the waist," to "going all the way." The same is true for delinquent behavior, which begins with minor transgressions and status offenses and progresses to misdemeanors and then to felonies. In these other domains, it is perhaps more easily seen that the developmental sequence is socially organized and involves processes of exploration and learning, and of social opportunity and access. The normative regulation of developmental sequences in these domains applies just as well to drug use development, as do the issues of differential social opportunity and differential access. Indeed, it is clear that the developmental sequence of drug involvement is socially and historically determined and that it may vary over time and in different groups. The first drug that an individual will use is likely to be that which is most readily available and which is also legally and normatively acceptable.

A more penetrating consideration of the drug progression sequence raises a number of important issues. First, although well established, the sequence cannot be considered invariant. There are variations in the extent to which the basic pattern is observed in different groups and at different historical periods. Racial minorities are somewhat less likely to follow the orderly progression than Whites; younger cohorts, born after 1965, are less likely to exhibit that pattern than older cohorts born earlier; a higher proportion of heavy users have started their drug career with an illicit drug compared with individuals in the general population; and there is evidence of subgroups of individuals who initiate their drug use with drugs later in the sequence, or who skip stages, or who initiate drugs from different stages in the sequence, for example, alcohol and marijuana, simultaneously. What this means is that sequential

progression is not inherent in the nature of the drugs themselves but emerges from the social organization of their availability and the social and personal definitions of their use.

Second, initiation of a drug early in the sequence does not entail the beginning of an inexorable progression through the remainder of the sequence. Only a very small minority of youth progress to late-stage illicit drug use. It is only for this small segment of drug users that the sequence of drug progression as a whole is relevant.

Third, the sequence is based entirely on the use of different drugs or classes of drugs rather than on differential use of a given drug. Yet, there is progression within the use of a drug, from initiation and exploration to more regular use to daily use to heavy use to dependence. How this within drug progression maps onto the between drug progression has not been well explored.

Fourth, the sequence of drug progression reflects a developmental order based on drug use behavior alone, as if drug use were independent of the adolescent's involvement in other, non–drug use behavior occurring during the very same period of the life course. It is more useful to see the drug progression sequence as having been extirpated from a much richer and more complex developmental sequence that includes other key behaviors and experiences, such as sexual initiation, dropping out of school, starting a part-time job, joining a gang, experiencing psychiatric problems, or getting involved in church activities. Establishing the location of non–drug use experiences within the drug progression sequence would not only provide a deeper understanding of the sequence itself, but would embed drug progression within the course of adolescent development as a whole.

Finally, there is the issue of whether additional drugs need to be considered in the drug progression sequence. The potential importance of caffeine as an earlier licit drug that might well precede alcohol and tobacco in any sequence of drug progression has been raised. Similarly, prescribed psychotropic drugs may represent the last stage in the progression. Studies of the drug progression sequence will need to give more attention to these possibilities.

In sum, although the sequence is well established, its meaning and significance for understanding drug use careers and adolescent development as a whole, are not yet obvious. The issues we have touched on constitute part of an agenda for future research to achieve a deeper understanding of the larger significance of the sequence of drug progression.

The Association Proposition

The association proposition assumes that the use of an earlier stage drug is correlated with an increment in the risk of using a drug next in the sequence. For example, those who have used alcohol or tobacco are shown to be at increased risk of using marijuana compared with those who have never used alcohol or tobacco. Said otherwise, it is less likely that an adolescent will use a drug later in the sequence if he or she has not already used a drug earlier in the sequence.

The research reported in the chapters in this volume, both the studies concerned with etiology and those concerned with prevention, have provided strong evidence in support of the association proposition. The likelihood of initiating a drug later in the sequence is enhanced among those who have initiated use of an earlier drug, and prevention of the initiation of a drug earlier in the sequence has been shown to be associated with a reduced likelihood of initiation of a drug later in the sequence.

What emerges most compellingly about the association proposition from the preceding chapters is that increased risk of transition through the drug sequence is associated far more strongly with *intensity* of use than it is with use per se. This is a critical contribution to a deeper understanding of the Gateway Hypothesis. The key antecedent to risk of progression to a later drug is heavy or frequent involvement with a drug earlier in the sequence. This conclusion has implications for both etiological and prevention research. For the former, it calls for greater understanding of variation in the risk and protective factors that generate a pervasive commitment to or enduring reliance on a drug so that its use is central to daily life and warrants characterization as abuse or dependence; for prevention it raises at least an additional agenda, the prevention of abuse or heavy involvement rather than of use alone.

The Causal Proposition

The association between the use of an earlier drug and the increased risk of use of one later in the sequence is often interpreted as demonstrating causal influence. Such an interpretation is untenable because association does not establish causation. The latter requires that all reasonable alternative inferences be rejected and that there be an understanding of the processes underlying association. The most obvious alternative inference is that those factors that influenced the use of

the initial drug are also responsible for the use of the subsequent drug. There is a set of etiological "third variables" that underlie and explain the relationship between the two observed variables, the use of the initial drug and the increased risk of use of a subsequent drug. It is this important distinction that led us to parse the proposition of association from that of causation.

Several issues regarding the causal proposition have been clarified by the chapters in the volume. Consideration of causal influence needs to be concerned with intensity of use rather than use per se, because intensity appears to be more strongly associated than use with transition in the drug sequence. Moreover, any effort to establish causality will have to resolve the fundamental issue that the antecedent risk and protective factors that influenced earlier drug use, especially intense drug use, may also be the factors that influenced transition or progression to a later stage drug. Analyses that control for relevant antecedent influences in longitudinal designs are well represented in the chapters in this volume. Such efforts advance understanding by articulating the network of factors relevant to variation in adolescent drug use or intensity of use. They also have shown that the association between a lower-stage and a higher-stage drug can be reduced substantially by instituting such controls. The reduction in the magnitude of the association reflects the explanatory role played by the antecedent or control variables, and it reveals the success of the authors in achieving a substantial explanatory account of drug use and drug progression.

When an association remains after controlling a large variety of relevant variables, that residual association has sometimes been imputed to drug use itself, or to intensity of use, as representing its direct causal influence. However, such claims need to be tempered by the limitations of the existing evidence. First, all the relevant variables cannot be controlled in any single study. Even the most rigorous investigation can gain control over only a subset of the influences at work in an adolescent behavior as complex as drug use. Second, most investigators have given greater attention to assessing the adolescent than to assessing the larger social ecological factors and the immediate context of drug use behavior. The social environment is often summarized by a handful of sociodemographic attributes that are relatively remote from behavior, or by perceived models and pressure for drug use. Much less attention has been paid to assessment of the models themselves, to social norms, to cultural and subcultural values, to the social organization of access and availability of supplies, and to ethnographic assessments of the social meaning of

adolescent drug use behavior in the various contexts of daily life. This is a key lacuna of uncontrolled variation in contemporary research on adolescent drug use.

Third, the domain of relevant variables under consideration should be broadened to include biological and genetic factors, and may benefit from the use of genetically informative samples, such as twins or adoptees.

Fourth, the research on delineating the factors that can account for variation in drug use has focused almost exclusively on establishing risk factors and, until very recently, has tended to ignore protective factors, factors that promote positive, prosocial behavior and that buffer the impact of exposure to risk. Clearly, variation in involvement with drugs, whether use or heavy use, is a joint outcome of the pattern of risk and protection that characterizes any sample of youth.

Fifth, it will be important to identify factors that may be differentially relevant at different stages of drug involvement, and for different subgroups of young people.

Finally, a more systematic approach to controlling the factors relevant to variation in drug involvement is to rely on theory to specify those variables. Greater reliance on theory would increase the likelihood of exhausting the multiple sources of variance involved.

Thus, it remains possible that uncontrolled sources of influence underlie whatever residual association obtains between earlier- and later-stage drug use. To reject that possibility, research that is comprehensive across all domains of influence from genetics to culture would be required. This is the reason why causal claims in the Gateway Hypothesis – the causal proposition – are still beyond reach.

Conclusion

The chapters in this volume demonstrate the understanding that contemporary behavioral science has achieved about how adolescents become involved in drug use and progress to the use of different drugs or drug classes. A major objective of the volume, to expand the range of approaches to the Gateway Hypothesis and to examine their convergence, has been achieved. The more common etiological studies were supplemented by prevention/intervention studies, by state-of-the-art analytic methodology, and by the contributions of animal models and neurobiology. These different approaches were all brought to bear on a single issue of broad societal concern – the Gateway Hypothesis. In this regard the enterprise is unique and its accomplishments are provocative.

Clearly, much remains to be done. Our parsing of the Gateway Hypothesis into propositions about sequence, association, and causality has suggested a research agenda that derives from each of those topics. Perhaps the overriding challenge for the future is to achieve a better understanding of heavy involvement with drugs and of the role of heavy involvement and of other factors in the transitions along the sequence of drug development. This challenge can be met by engaging new domains of knowledge and by more systematically articulating the domains we are already familiar with. With respect to new domains of knowledge, the work on animal models is promising. The pursuit of such models can capture some of the critical processes involved in drug use behavior and progression, such as the effects of prior exposure to drugs and the impact of stress. However, the limitations of these models for representing the personal meanings and the social aspects of drug use behavior will test the ingenuity of animal modelers in their future research. The work in neurobiology and the new developments in behavioral genetics represent other areas of increasing relevance to understanding processes underlying drug intensification and drug progression.

With regard to the more familiar domains of inquiry, there is a need for more penetrating assessments of the contexts of daily adolescent life, that is, for better contextual models. Greater attention also needs to be paid to protective factors in the adolescent, in the social context, and in the culture. Such efforts will expand our understanding of the factors relevant to variation in drug initiation as well as drug intensification and drug progression. The more exhaustive the explanatory account, the less likely there will be residual association to permit inferences about cause to be attached to drug use per se, or even to intensity of drug use.

We conclude that interpretations of the Gateway Hypothesis should be restricted to the propositions about sequencing and association. The causation proposition is without evidential support at this time. A research agenda that can help clarify where in the overall nomological network the causal vector should be located is what is clearly called for if we are to advance the science of drug initiation and drug progression. Implementing that agenda is the task before all of us.