Chapter 3

The Theory of Triadic Influence: Preliminary Evidence Related to Alcohol and Tobacco Use

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There has been relatively little research on how biological influences contribute to tobacco and alcohol use. Our recent reviews of the theories of tobacco use (Flay and Petraitis 1994) and illicit drug use (Petraitis et al. 1995) concluded that biological influences are scarcely mentioned in the theories that dominate the field (e.g., the problem behavior theory). This is unfortunate because no understanding of tobacco and alcohol use is complete unless biological influences are addressed, and the mechanisms by which biological factors contribute to the use of tobacco and alcohol are carefully described.

In this chapter we want to show how the biological views presented in other chapters in this monograph represent one piece of a larger puzzle that includes both biological and nonbiological influences. The nonbiological influences include (a) the influence of modeling, when parents show children how much pleasure they derive from alcohol and tobacco; (b) developmental influences, in which lifelong patterns of alcohol and tobacco use might begin in an adolescent’s otherwise healthy desire for independence from parents; and (c) cultural influences, like advertising, in which tobacco and alcohol use
become linked as rewarding activities that people do to relax, to be sociable, and to treat themselves. We also want to focus on factors that cause adolescents to start smoking and start using alcohol. Toward these ends, we will describe (1) a theoretical framework that captures most (if not all) influences on experimental tobacco use (ETU) and experimental alcohol use (EAU) among adolescents; (2) some evidence for the causal processes that are articulated in our theoretical model, including research evidence on the onset of use of alcohol, tobacco, and other drugs (ATOD); and (3) implications of our model for understanding the causes of ETU and EAU, and for preventing ETU and EAU.

A THEORETICAL FRAMEWORK OF INFLUENCES ON ETU AND EAU

The dominant theories of ETA and ETU among adolescents include a long and diverse list of causal influences, ranging from factors that are intrinsically tied to ATOD use (e.g., beliefs about the consequences of smoking) to factors that on the surface have little to do with these problem behaviors (e.g., parenting styles and school characteristics) (Flay and Petraitis 1994; Petraitis et al. 1995). The diversity of theories and causal influences is not surprising given that ATOD use, like most behaviors, has a complex etiology. In fact, numerous scholars (e.g., Magnusson 1981;

**Figure 1.** Three ultimate causes of behavior: reciprocal determinism.
Bandura 1986; Sadava 1987; Frank-enhaeuser 1991; Jessor et al. 1991; DeKay and Buss 1992) have argued persuasively that a thorough understanding of any behavior must look at “the big picture.” That picture must be based on a comprehensive and integrative analysis of (a) the broad social environment or cultural milieu surrounding the behavior, (b) the more immediate social situation or context in which the behavior occurs, (c) the characteristics or dispositions of the person performing the behavior, (d) the behavior itself and closely related behaviors, and (e) the interaction among all of these.

Figure 1, in a very general sense, represents “the big picture.” It reminds researchers and theorists that ETU and EAU have roots in broad environmental or cultural factors (e.g., media depictions of smoking and alcohol use), situational factors (e.g., poor relationships with parents), and personal factors (e.g., individual sensitivities to nicotine) and that all of these factors affect each other and are also influenced by the behavior itself (i.e., reciprocal determinism). It also reminds us that any one factor is just one small part of a larger picture and is only likely to explain small portions of variance in either ETU or EAU.

The model depicted in figure 1 is not a testable theory; it is far too general and lacks important information about variables and relationships among variables. It is, however, the foundation of a theory that we call the theory of triadic influence (TTI).

According to TTI, the general cultural environment in which adolescents mature, the more immediate social situation in which adolescents find themselves from day to day, and intrapersonal differences among adolescents are the starting points for three “streams” of influence-streams that flow through different mediating variables and flow to different terminating variables.

**The Three Streams: Definitions and Terminating Variables**

Figure 2 begins to reveal these three streams in more detail. According to TTI, the first stream represents characteristics of the broad cultural environment and general social values that contribute to or terminate at adolescents’ personal attitudes concerning ETU and EAU. Such cultural/attitudinal influences include local crime and employment rates, media depictions and government policies concerning tobacco and alcohol use, lack of commitment to conventional values, and social alienation. The second stream represents characteristics of adolescents’ more immediate social situations and more intimate social support systems that contribute to or terminate at the social pressure adolescents feel to experiment with tobacco or alcohol. Such interpersonal or social/normative influences include parenting styles, the strength of bonds between parents and adolescents, the strength of bonds between peers and adolescents, and tobacco and alcohol use by parents and peers.
The third stream represents characteristics of individual adolescents' biological makeup and basic personality that undermine their ability to resist pressures to smoke cigarettes and drink alcohol. Such *intrapersonal influences* include (among others) biological sensitivities to nicotine and alcohol, fundamental and stable personality characteristics (e.g., neuroticism, extraversion, openness, agreeableness, intellect), more transient affective states (e.g., depressed affect, low self-esteem), social skills, and, ultimately, resistance or refusal skills.

**TIERS OF INFLUENCE**

So far, our description of TTI has focused explicitly on its three streams of influence. Figure 3, however, reveals that there is more to TTI than just streams of influence; there are also several tiers or levels of influence. The lowest three tiers of figure 3 represent the most *proximal* level of influence. Proximal influences are fairly narrowly defined (e.g., intentions to smoke a cigarette within the next 30 days), are inherently tied to ETU and EAU (e.g., smoking-related attitudes), are probably the most immediate* causes of ETU and EAU, and are the strongest predictors of ETU (Flay and Petratis 1994) and EAU (Petratis et al. 1994a).

The third and fourth tiers (from the bottom) of figure 3 are factors from more intermediate or *distal lev-
els of influence. Distal influences are factors that probably contribute indirectly to ETU and EAU by contributing directly to tobacco- and alcohol-specific attitudes, normative beliefs, and self-efficacy.

Finally, the highest tier of figure 3 represents the ultimate influences. Unlike proximal and distal influences, ultimate influences (a) are beyond the easy control of adolescents, (b) are not inherently tied to alcohol or tobacco use, (c) are broader in scope and not as narrowly defined, and (d) are more deeply rooted in an adolescent’s environment, personality, or biological makeup. As such, ultimate influences are likely to affect ETU and EAU in a variety of ways.

**EMPIRICAL EVIDENCE FOR TTI**

In the previous section we argued that TTI’s three streams of influence have different tiers or levels of influence. According to TTI, however, each stream also has a unique set of mediating variables and causal pathways through which causal influences primarily flow. Furthermore, causal processes are not thought to exist exclusively within one stream or the other. Rather, factors in one stream may cross streams and influence factors in another stream. In this section, we briefly describe these pathways and some of their empirical support. We provide a detailed review
of studies of causal process in another paper (Flay et al. unpublished manuscript 1995).

**PATHS AND MEDIATING VARIABLES IN THE CULTURAL/ ATTITUDDINAL STREAM**

According to TTI, the cultural/attitudinal stream begins in the general cultural environment in which adolescents mature, the information they derive from their culture about tobacco and alcohol, and the general social values they adopt from their culture. In turn, these factors combine to affect (a) adolescents’ subjective expectations about the personal consequences of ETU or EAU, (b) their personal evaluations concerning the goodness-badness of those consequences, and (c) their overall attitudes toward their own ETU or EAU. This causal process is represented by path 1 in figure 3 and is supported by numerous empirical studies. For instance, after comparing the causal processes that lead to alcohol use in the United States and Greece, Marcos and Johnson (1988) concluded that teen alcohol use in the United States, unlike that in Greece, is a sign of social alienation and deviant attitudes. Grube and Wallack (1994) found that cultural images of beer in the United States (as provided by television advertisements) might affect attitudes toward drinking by leading many children to link “drinking with positively valued activities and consequences such as romance, sociability, and relaxation” (pp. 257-258).

TTI then argues that tobacco- or alcohol-related expectations, evaluations, and attitudes play a major role in shaping adolescents’ decisions or intentions to use (or avoid) tobacco and alcohol in the future (see path 2 of figure 3). In line with this, several studies have demonstrated that attitudes influence drug use through the mediating effects of intentions (e.g., Bentler and Speckart 1979; Schlegel et al. 1992; Flay et al. 1994; Flay et al. unpublished manuscript 1994; Petraitis et al. 1994).

**PATHS AND MEDIATING VARIABLES IN THE SOCIAL/ NORMATIVE STREAM**

Social/normative influences have their roots in the characteristics of adolescents’ parents, peers, and immediate surroundings, especially the tobacco- and alcohol-related attitudes and behaviors of family members and those peers to whom adolescents most closely bond. As depicted by path 3, TTI asserts that these characteristics affect (a) adolescents’ subjective perceptions about the normativeness of ETU and EAU, (b) with whom adolescents are most motivated to comply (e.g., conventional parents or deviant peers), and (c) the social pressures adolescents feel to experiment with either tobacco or alcohol. Several studies support this assertion. For instance, we found that parental and peer smoking indirectly affected adolescents’ smoking initiation and escalation by first affecting adolescents’ outcome expectancies regarding smoking, perceived approval of smok-
ing, and refusal skills self-efficacy (Flay et al. 1994).

Perceived norms, motivation to comply, and social normative beliefs are important because they are among the few factors that directly affect adolescents' decisions or intentions to use (or avoid) ETU or EAU in the future (see path 4 on figure 3). Several studies have demonstrated that attitudes influence drug use through the mediating effects of intentions (Ellickson and Hays 1991, 1992; Flay et al. 1994; Flay et al. unpublished manuscript 1994).

PATHS AND MEDIATING VARIABLES IN THE INTRAPERSONAL STREAM

Whereas the first two streams began with characteristics of the general culture in which adolescents mature and characteristics of their more immediate social situations, the intrapersonal stream begins with fundamental and stable characteristics of the individual adolescents (e.g., their personalities and biological makeups), their general levels of competence (e.g., social and academic skills), and their sense of self (e.g., the strength of their self-concepts). As depicted by path 5 on figure 3, these influences are thought to affect (a) adolescents' skills at dealing with situations where they are offered cigarettes or alcohol, (b) their determination to either use or avoid cigarettes and alcohol, and (c) their tobacco- and alcohol-related self-efficacy. In support of this causal process, Dielman and colleagues (1989) found that adolescents' locus of control influenced alcohol use indirectly by first affecting adolescents' self-esteem, social competence, and social skills.

The second link in the causal process of intrapersonal influences runs to tobacco- or alcohol-related decisions or intentions from refusal skills, self-determination, and self-efficacy (see path 6 on figure 3). Supporting this link, Ellickson and Hays (1991) found that resistance self-efficacy regarding use of alcohol, cigarettes, and marijuana affected adolescents' use of these drugs both directly and indirectly through intentions.

INTERSTREAM PATHS: INTERACTIONS AND MODERATION

So far, this section has focused on within-stream, or intrastream, pathways. Paths 1-6 probably explain most of the variance in ETU and EAU. However, we do not believe that all causal processes lie neatly within one stream or the other. Rather, factors in one stream might exert some influence on factors in other streams. Moreover, such interstream influences are sometimes the effects of variables on a higher level within one stream being mediated by variables at a lower level in another stream. These interstream influences are depicted by paths a-f in figure 3.

Interstream influences may also take the form of statistical interactions such that the effect of a variable in one stream moderates the influence of a variable in another stream. For instance, intrapersonal influences...
(e.g., personality characteristics and biological makeups) might make some adolescents more susceptible to some social influences (e.g., peer pressures to smoke). In line with this, Bauman and colleagues (1992) showed that high testosterone levels (an intrapersonal characteristic of adolescents) exaggerate the effects of peer or parental smoking (which are social influences in TTI). The stress-coping model (see chapter 6 in this monograph; see also Wills and Shiffman 1985) is a special case of this type of interaction. This model postulates that experiences of stress (from higher in the intrapersonal stream) may be moderated by expectancies, reasons for use, or coping functions (from lower in the cultural/attitudinal stream).

Interstream influences of ethnicity and gender are of particular interest. Although we assume that TTI describes the causal process of ETU and EAU for all ethnic groups, we also assume that the specific weights applied to particular paths probably vary by ethnicity. For example, we found that the effects of friends’ smoking on adolescents’ initiation of smoking were both direct and indirect—the latter mediated through refusal skills for whites and Hispanics, whereas the effects of friends’ smoking for blacks were only direct (Flay et al. 1994). The TTI describes the causal process of ETU and EAU for both males and females, even though the specific weights applied to paths probably differ and males and females probably react differently to some known predictors of ATOD use. As with ethnicity, we suspect that some of these differences are due to biological differences whereas others are due to cultural differences in how males and females are raised.

Several studies suggest that factors in one stream do, in fact, moderate factors in the other two streams. Risk factors from two (sub)streams may interact such that one increases the effects of the other. For example, high-risk-taking adolescents may be less susceptible to family influences than are low-risk-takers because the former are more likely to seek independence. Similarly, adolescents with low refusal skills are more prone to peer pressure than those with adequate refusal skills. We tested these hypotheses in the context of smoking onset from grade 7 to grade 8. Data for the analyses were collected as part of the Television, School and Family Project (TVSFP) smoking prevention program in Southern California (Flay et al. 1988, 1995). Baseline data were collected in 1986 from 6,695 seventh grade students from 47 schools in Los Angeles County and San Diego County.

Figure 4 shows the interactive effects of two distal risk factors: family conflict (a 3-item scale, Cronbach’s alpha = 0.74, median split into low and high family conflicts) and risk-taking (a 3-item scale, Cronbach’s alpha = 0.77, median split into low- and high-risk-takers). Smoking onset rates were 25.9 percent for low-risk-takers with low family conflict and 41.8 percent for
low-risk-takers with high family conflicts, whereas the onset rates for high-risk-takers were 34.6 percent and 38.4 percent for low and high family conflict, respectively. The relative risk of smoking onset imposed by family conflict is higher for low-risk-takers than for high-risk-takers (odds ratios 2.06 versus 1.18). These findings suggest that low-risk-taking adolescents are more reactive to family environment than high-risk-taking adolescents.

Figure 4 also shows the interactive effects between two proximal risk factors: friends' smoking and refusal skills self-efficacy. The relative risk of smoking onset imposed by friends' smoking shows a decreasing trend with increasing refusal skills (odds ratios are 2.97, 2.73, and 1.75, respectively, for low-, medium-, and high-refusal-skill adolescents). These results indicate that adolescents with inadequate refusal skills self-efficacy are more susceptible to peer pressure.

**Feedback and Longitudinal Effects**

Feedback or reciprocal effects postulated by TTI (Flay and Petraitis 1994) reflect the broad idea of reciprocal determinism (cf. Bandura
Feedback influences are concerned with the role of prior behavior. Though it has been well documented that prior drug use is the best predictor of later drug use, there is little research on how experiences derived from prior drug use influence later drug use. TTI proposes a feedback mechanism by which this might happen. Specifically, 'feedback loops connecting prior and later behaviors are formed in each stream of influence through a dynamic interplay between causal factors and behaviors (figure 5). Initially, drug use is affected by variables such as social bonding and attitudes. Once adolescents start to use drugs, the causal ordering reverses and these variables are subsequently shaped by the drug use behavior. These modified factors, in turn, affect later drug use.

Several studies have provided support for the idea of a feedback mechanism. For example, Krohn and colleagues (1985) found that prior smoking affected smoking maintenance both directly and indirectly through positive and negative consequences of smoking. Similarly, Kaplan and colleagues (1988) found that earlier drug use influenced later drug use both directly and through its impact on association with drug-using peers and perceived negative social sanctions. In a well-

![Figure 5. Longitudinal and developmental effects according to the theory of triadic influence. ATT = attitude; BEH = behavior; ENV = cultural environment; PER = biology/personality; SE = self-efficacy; SNB = social normative beliefs; SOC = social situation; t1-t5 = times 1-5.](image-url)
known study, Bentler and Speckart (1979) tested the effects of prior behavior within the framework of Fishbein and Ajzen’s (1975) theory of reasoned action and found that some of the effects of prior drug use were mediated by intention and some of the effects were direct. In a replication and extension of this study, our group (Petraitis et al. 1994b) found that prior smoking had several indirect (mediated) effects: (1) it promoted more positive attitudes toward smoking, (2) it altered adolescents’ beliefs in the normative nature of smoking, and (3) it undermined adolescents’ resolve to resist smoking; it also had direct effects on intentions to smoke in the future and on future smoking.

Unfortunately, these studies only focused on two-wave data and ignored the dynamic nature of the effects. In figure 6, we model both feedback and longitudinal effects using three-wave data from the TVSFP. In this model, drug use is a latent variable with smoking and alcohol use as indicators. Risk-taking and friends’ smoking are latent predictor constructs measured over time. Note how drug use at one wave “feeds back” to cause changes in the risk predictor variables. Note also that the size of these effects may change over time.

Figure 6. Three-wave latent variable model of two risk factors and drug use. \( \chi^2 = 122.09 \) (df = 33). Adjusted goodness-of-fit index = 0.970. All coefficients are standardized and are significant at \( p < 0.05 \). Data are from the Television, School and Family Project smoking prevention program in Southern California.
Role of Related Behaviors

Influence of Related Behaviors

Related behaviors (e.g., tobacco, alcohol, and marijuana use) may influence each other. It has been frequently found that adolescents who use one drug are more likely to use another one, leading to the widely accepted conclusion that the use of one drug serves as a gateway for use of other drugs. This unidimensional conceptualization of drug use behaviors, however, may be too simplistic without considering other risk factors.

TI postulates that the influence of related behaviors might be modified by psychosocial factors. Figure 7 shows the interactions of two variables, one cultural and one interpersonal, with prior alcohol use on the onset of cigarette smoking. Among those who did not regularly attend religious worship, prior alcohol users were more likely to initiate cigarette smoking than those who had not used alcohol (odds ratio = 3.5). For those who regularly attended religious worship, the risk for the transition from alcohol use to cigarette smoking was much less (odds ratio = 1.9).

Figure 7 also shows that the risk for the transition from alcohol use to cigarette smoking was much higher for those who had low refusal skills self-efficacy than for those who had medium and high refusal skills self-efficacy (odds ratios were 3.76, 2.00, and 1.90, respectively). Alcohol use puts students at risk for future smoking, as the gateway hypothesis predicts, but much more so for those who do not attend church, and more so for those with low refusal skills self-efficacy.

The preceding example (figure 7) describes how other risk factors affect the influence of alcohol use on the use of cigarettes. This moderating process, however, does not stop there. The line in figure 8 shows the well-known phenomenon of prior alcohol and tobacco use increasing the risk for marijuana use, illustrating the gateway drug hypothesis.

The gateway metaphor, however, may be too simplistic when we consider the effects of risk factors in the process of drug escalation, because related behaviors may also interact with risk factors to influence subsequent behavior. Figure 8 shows that the basic gateway drug hypothesis is true only for students who are otherwise also at high risk (who score high on risk-taking and whose friends also smoke cigarettes); it is not true for those not otherwise at risk (who score low on risk-taking and whose friends do not smoke). Specifically, for those students who were low-risk-takers and had no smoking peers, marijuana use was independent of prior use of alcohol and cigarettes. For those who were high-risk-takers but had no smoking peers and for those who were low-risk-takers but had smoking peers, marijuana use was much higher for prior cigarette smokers compared with those who used alcohol. This kind of “stepping-stone” phenomenon of adolescent drug involvement was most pronounced for the
Theory of Triadic Influence

A. A cultural background variable: Religious Worship or not

B. An intrapersonal variable: Refusal Skills Self-Efficacy

Figure 7. Risk of smoking by grade 8: Two interactions with prior (grade 7) alcohol use. (A) Interaction of religious worship with prior alcohol use. (B) Interaction of refusal skills self-efficacy (RS SE) with prior alcohol use. Data are from the Television, School and Family Project smoking prevention program in Southern California.

students at highest risk (they were both high-risk-takers and had smoking peers). These findings suggest that relying on a single risk factor orientation runs counter to the known heterogeneity of behavior. We need to consider multiple risk factors and their interactions to find out who is at risk and under what conditions.

**Prediction of Closely Related Behaviors**

Researchers are often asked whether or not the various drug use behaviors are part of the same latent construct. These behaviors have been conceptually characterized to form a unidimensional structure, that is, problem behaviors (Jessor and Jessor 1977). TTI suggests that closely related behaviors may have the same, or very similar causes, and less related behaviors less so (Flay and Petraitis 1994). The more dissimilar the behavior, the less the overlap of the causal structure. The overlap varies most at the most proximal levels and least at the most distal levels (see figure 7 in Flay and Petraitis 1994). The ultimate or root causes of related behaviors are generally the same.

Related behaviors obviously do correlate with each other (that is, after all, part of what we mean by...
Figure 8. Risk of marijuana use by grade 8: Effects of prior behavior (grade 7), peer behavior, and risk-taking. Data are from the Television, School and Family Project smoking prevention program in Southern California.

“related”). They probably also have most of the same causes. We may ask whether these causes interrelate with each other and influence related behaviors in the same ways. If alcohol use and smoking had a similar causal structure and influenced related behaviors in the same ways, the model shown in figure 9 would describe the relations among their causes. In this model, smoking and alcohol behaviors are characterized as one latent phenomenon, which is influenced by risk-taking preferences and the number of smoking friends. Demographic variables like gender and race mainly affect the adolescents’ risk-taking (personality) and the exposure to smoking friends. This model does, indeed, provide a good statistical fit to our data.

Despite the good fit of the “one-factor” model, we can fit an alternative “two-factor” model. In this model, smoking and alcohol use are treated as separate constructs, but they are assumed to have the same causes. However, the model allows for the causes to behave in different ways; that is, it allows for different causal processes. Even the same causes may act in slightly different ways with respect to different behaviors. The model shown in figure 10 (which does fit better than the model of figure 9 at a statistically significant level), suggests that gender, ethnicity, risk-taking and friends’ smoking all act differently for
alcohol and tobacco use. In particular, gender and race still have differential direct effects on smoking and alcohol use, even after accounting for their indirect effects through the other risk factors. In broad terms, white adolescents drink more (and earlier) than adolescents of other races, and girls drink less than boys.

This finding suggests that we revisit our earlier model of the longitudinal effects of drug use, this time separating alcohol and tobacco use. In figure 11, we see that the risk variables not only predict the two behaviors at different levels, but that experience with the behavior also changes these relationships over time.

IMPLICATIONS AND CONCLUSIONS

The relations between alcohol use and cigarette smoking are complex. The traditional gateway or unidimensional explanations for experimentation may be too simplistic. So far, we have demonstrated four main points. First, alcohol use and cigarette smoking do share several causes and pathways, which are described by the generic theoretical framework provided by TTI. Second, the relation between alcohol use and cigarette smoking can be modified by other risk factors. For example, the transition from alcohol use to cigarette
smoking is more likely to occur among adolescents who have other risk factors, such as low refusal skills and exposure to smoking peers. Third, alcohol use and cigarette smoking can be conceptualized either as one latent phenomenon or as different behaviors. Generally speaking, the two behaviors (perhaps along with other deviant behaviors) have more similarities than differences in terms of causes. However, differences in causal processes may have important implications for understanding and prevention. Fourth, the relation between alcohol use and cigarette smoking is dynamic, as are the relations between other risk factors and drug use. In order to understand the developmental process of drug use, we need to take several effects into consideration, including growth effects (longitudinal or time effects), reciprocal effects, mediating effects, and moderating effects. These effects are all incorporated into TTI.

All of the points made above have implications not only for understanding why adolescents start to experiment with alcohol or tobacco, but also for behavior change in general and prevention in particular. The mul-

Figure 10. Two-factor model of smoking and alcohol onset. $\chi^2 = 2.38$ (df = 5). Adjusted goodness-of-fit index = 0.997. All coefficients are standardized. Data are from the T&vision, School and Family Project smoking prevention program in Southern California.
tilevel and multidimensional nature of TTI suggests that intervention and prevention programs for adolescent alcohol and tobacco use should also be multilevel and multidimensional (figure 12). At more distal levels, TTI suggests that legislative, media, and community interventions are needed to change the informational, cultural/values, normative, and social support aspects of the broader cultural or social environment. At more proximal levels, adolescents (and parents) need to be taught appropriate attitudes (and supportive knowledge and values), normative beliefs (with appropriate models and social approval/disapproval), and social skills (with appropriate opportunities for practice and reinforcement).

RECOMMENDATIONS FOR FUTURE RESEARCH

Research is needed to establish more clearly the links and causal relationships between intrapersonal, interpersonal, and environmental risks and protective factors, and the relationships of all of these with behavior. We make the following recommendations for future research:

- Studies of causal process. In general, we encourage more studies of the
causal processes by which risk factors contribute to adolescents’ experimental use of tobacco and alcohol. As TTI postulates, the etiology of ETU and EAU use is complex and involves both direct and indirect paths, moderating effects, and feedback or reciprocal effects. Unfortunately, past research has too rarely reflected this complexity. Although there are signs of change among more recent studies, too often past studies have stopped at describing univariate or multivariate predictors of alcohol and tobacco use and have not described the complex causal process by which variables contribute to drinking and smoking. Therefore, we encourage researchers to move from finding predictors to describing causal processes.

**Studies of causal processes involving sociocultural environmental factors.** Although future studies should focus on causal processes in general, there is a particular need for studies that focus on the causal processes by which sociocultural factors contribute to tobacco and alcohol use. More research is needed to examine the influences of macrolevel sociocultural factors, such as public policies, neighborhood, poverty, and media. Grube and Wallack (1994) provided one of the few examples of such a study by examining how exposure to al-

![Diagram of intervention points](attachment:image.png)
coho1 alcohol advertisements influences adolescents’ drinking intentions.

- **Studies of causal processes involving intrapersonal characteristics.** We also recommend that more studies focus on the causal pathways by which ETU and EAU are rooted in intrapersonal characteristics, especially biological factors and fundamental personality traits. In fact, our recent review of theories of experimental drug use (Petraitis et al. 1995) suggested no clear causal pathways that link biological or personality variables to drug use. More research is also needed on the interactive effects of biological and psychosocial factors on tobacco and alcohol use.

- **Studies of causal processes Leading to dependence and addiction.** Although there are numerous studies on the etiology of ETU (Flay and Petraitis 1994) and experimental drug use (Petraitis et al. 1994a; Flay et al. unpublished manuscript 1995), few studies describe the predictors of tobacco or alcohol dependence and addiction, and even fewer studies describe the causal processes of becoming dependent or addicted. Therefore, more research is needed to examine the natural history of tobacco and alcohol use and the potentially different etiologic factors and pathways associated with experimental use and abuse.

- **Studies of developmental changes.** In addition, we recommend more studies of developmental changes in the influence on tobacco and alcohol use. Influences on tobacco and alcohol use may vary with different stages of adolescence. A time-varying design (e.g., multiple waves of observations over critical periods of adolescence) and a time-varying statistical analysis (e.g., random effects or multilevel models) are crucial in investigating the developmental paths leading to tobacco and alcohol use.

- **Multiwave and intervention studies of tobacco and alcohol use.** The preceding recommendations are based on our belief that too few studies have attempted to untangle the complex etiology that leads to the use of tobacco, alcohol, or both. Our final recommendation is that researchers design more studies that can untangle the causal factors, moderating effects, feedback effects, and other direct and indirect effects and can test complex causal processes. In particular, we recommend that researchers design future studies of etiology with enough waves of data (probably six or more) to allow them (a) to carry out sensitive tests of complex and interwoven causal processes, (b) to test feedback mechanisms and reciprocal effects, and (c) to test for developmental changes over time. In addition, the suggested causal relationships should be subjected to the ultimate test-by designing interventions to change the presumed causes and then determining if such change leads to changes in behavior (less or no alcohol or tobacco use). It is only with such
linked tests of theory and preventive interventions that a true science of prevention can develop fully (Kellam 1994).

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