Published in final edited form as:
Monogr Soc Res Child Dev. 2009 ; 74(3): . doi:10.1111/j.1540-5834.2009.00528.x.

A dynamic cascade model of the development of substance-use onset

I. INTRODUCTION

In spite of massive efforts, the American war on drugs has not yet been won (Caulkins, Reuter, Iguchi, & Chiesa, 2005; Miron, 2008). Federal, state, and local law enforcement agencies spend over US$44 billion per year in interdiction, prosecution, and incarceration. It is expected that arrests for drug law violations in 2009 will exceed the 1,841,182 arrests of 2007 (Uniform Crime Reports, 2009). Treatment expenditures are also massive. Substance use disorder treatment expenditures exceed US$18 billion per year (Substance Abuse and Mental Health Services Administration, 2005). In spite of these efforts, the rate of substance use disorders has not changed appreciably in the past decade. Data from 2004 to 2007 indicate that 9.7% of adults aged 18 and older in the United States need treatment for a substance use problem as defined by the Diagnostic and Statistical Manual of Mental Disorders (Substance Abuse and Mental Health Services Administration, July 2, 2009). The rate among young adults aged 18–25 is over double that rate, at 21.1% of the population (Substance Abuse and Mental Health Services Administration, June 25, 2009).

During adolescence, problem behaviors, especially alcohol, tobacco, and other drug (ATOD) use, coalesce (Johnston, O'Malley, Bachman, & Schulenberg, 2008) and become costly in treatment, lost wages, and crime (Miller, 2004). Underage drinking alone costs the United States US$53 billion annually (National Research Council, 2004). The U.S. Office of National Drug Control Policy (1999) indicates that substance-use-related economic costs total US$377 billion annually and are on the rise. Miller (2004) estimated the annual cost of multiproblem youth at over US$400 billion. Cohen (2005) estimated the lifetime cost per adolescent persistent drug abuser at US$970,000, and the social–psychological consequences are even more devastating (Kendall & Kessler, 2002; Kessler et al., 2001).

These enormous costs, and the failure of law enforcement and treatment programs, have led to modest prevention efforts. Cohen (2005) has cited simulation experiments that show that American taxpayers would endorse higher levels of funding for prevention if, and only if, programs were first proven effective. Unfortunately, prevention programs have not yet demonstrated large-scale success (Ennett et al., 2003). School-based prevention efforts have received most of their funding from the Safe and Drug-Free Schools and Communities Act (Modzeleski, 2006). When the predecessor to this legislation was first enacted in 1986, it provided US$490 million for the DARE program. The program reached its zenith in 1992, when it received $502 million and penetrated virtually every public school system in the country. Randomized-controlled trials later proved DARE to be ineffective (Ennett et al., 1994). Although other programs have since proven efficacious in small experiments, at-scale prevention efforts have not yet realized effective impact (Ennett et al., 2003).

Toward the goal of developing empirically based prevention programs, epidemiologic studies have proliferated. These studies have taken largely a risk-factor approach following from the pioneering methods of Rutter and Garmezy (1983), in which individual-difference variables in childhood are statistically linked to later substance use. Empirical research has identified several dozen factors in childhood that enhance risk for substance use during adolescence (reviewed by Dahl & Spear, 2004; Hawkins, Catalano, & Miller, 1992; National Research Council, 2004; Weinberg, Rahdert, Colliver, & Glantz, 1998; Zucker,

2006b), but a laundry list of risk factors has not yet led to efficacious prevention programs. Although numerous theories of substance-use onset that compile these factors have been offered (e.g., Brook, Brook, Gordon, Whiteman, & Cohen, 1990; Catalano, Kosterman, Hawkins, Newcomb, & Abbott, 1996; Simons, Conger, & Whitbeck, 1988), none has sufficiently explained the developmental–transactional relations among risk factors and the ecological transitions that a child goes through on a path toward substance use in order to guide strategic preventive intervention (see Petraitis, Flay, & Miller, 1995, for a review of theories). The goals of this monograph are to (1) articulate a developmental theory that integrates the extant literature; (2) subject the proposed model to rigorous empirical testing through prospective inquiry; and (3) provide implications of the findings for prevention practice and public policy.

CONTEMPORARY THEORIES OF ADOLESCENT DEVELOPMENT

Moving well beyond the cataloguing of risk factors and the contentious debate between nature and nurture, contemporary theories build on Sameroff and Chandler's (1975) seminal transactional model and updated treatise (Sameroff, 2009) to emphasize the dynamic relations between the individual and multiple social contexts across development from birth through adolescence. Lerner and Castellino (2002) have noted, "(T)he forefront of contemporary developmental theory and research is associated with theoretical ideas stressing that the systemic dynamics of individual-context relations provide the bases of behavior and developmental change" (p. 124).

Theorists of development have articulated concepts of reciprocal influences, mediational mechanisms, transactional exchanges, dynamical systems, and interaction effects that cumulate over time. Gottlieb (1997) introduced the term coaction to describe the coordinated exchanges between a child and the environment across development. Dahl and Spear (2004) have described how rapid brain development during adolescence leads the youth to be especially influenced by environmental stimuli that alter brain structure, which, in turn, leads the youth to turn toward specific appealing environments. Steinberg et al. (2006) have provided a map for how myriad genetic, biological, social, and ecological factors conspire to produce disorders in adolescence, including substance abuse. Collectively, these theories tell a story of development that begins with an infant born into a social ecology that both shapes and is shaped by the infant. Brain structures guide the child to gravitate toward compatible and reinforcing environments, but those environments act on the child as well to shape brain development, much like the wind shapes the growth of a tree limb.

The developmental transaction between the child and the environment in emergent behaviors is played out at multiple levels. Microexchanges between a child and parent that occur across minutes tell the story of the onset of aggressive coercive acts (Granic & Patterson, 2006). Daily exchanges between a rapidly growing infant and a physical environment tell the story of the onset of walking (Thelan, Ulrich, & Wolff, 1991), and ongoing social exchanges involving aggression and social rejection that are played out across years tell the story of social–cognitive development (Fontaine, Yang, Dodge, Bates, & Pettit, 2008).

EMPIRICAL TESTING OF THEORIES OF ADOLESCENT DEVIANCE

Although developmental theory has become rich, empirically testing the complex dynamic interplay postulated by Sameroff and Chandler (1975) in the domain of complex phenomena such as adolescent deviance has proven daunting. Most efforts have been restricted to transactions between the child and only one other social unit. For example, Stice and Barrera (1995) found support for a transactional relation between adolescent problem behaviors and parenting, such that lack of parental support and control predicted adolescent substance use,

which, in turn, predicted decreases in parental support and control. Dishion and Owen (2002) found support for a transactional model of adolescent substance use and peer processes, in which deviant friendships predicted substance use, which, in turn, predicted later gravitation toward a growing deviant peer culture.

The most well-articulated dynamics systems model of deviant behavioral development has been offered by Granic and Patterson (2006), who described coercive exchanges between a child and a parent that accelerate and mutate into highly aggressive outcomes. Their description is compelling but is restricted to microlevel exchanges of aggression and does not offer a long-term or cross-context model of development from childhood into adolescence. Boker and Graham (1998) used dynamical systems theory to find support for an uncoupled linear oscillator model of frequency of cigarette and alcohol use across time in adolescents. Although they found the theory compelling, they acknowledged that future studies would need to integrate individual difference and environmental factors into a fuller developmental understanding. Gottfredson, Kearley, and Bushway (2008) have effectively integrated transactional models with dynamical systems theory to show that drug use, drug treatment, and crime influence each other across an 11-month period.

Charting transactional relations in microexchanges is important, but theories of development often posit transactional relations at a broader relationship level across longer periods of time. Very few studies have integrated dynamic relations across multiple social systems over long periods of time, and yet this integration is necessary for the design of preventive interventions. One challenging problem in such analysis is that the form of behavior evidenced in reciprocal relations changes across the child's development. For example, the child and parent may influence each other steadily across development with the child's "problem" behavior influencing the parent's "problem" behavior and vice versa, but the parent's problem behavior changes from noncontingent harsh disciplinary practices in early childhood to poor monitoring and supervisory practices in early adolescence. After reviewing studies of the transactional model, Sameroff and Mackenzie (2003) concluded that "(p)roblems remain in the need to theoretically specify structural models and to combine analyses of transactions in the parent–child relationship with transactions in the broader social contexts" (p. 613).

A multisystem transactional model of the development of adolescent aggression toward female partners has been postulated by Capaldi, Dishion, Stoolmiller, and Yoerger (2001). Following from Patterson (1986), they proposed that early coercive parent–child interactions influence a child's aggressive behaviors and the selection of the child's peer group. The peer group then takes on an increasing socializing role during adolescence, locking the youth into a trajectory that started with earlier family interactions. In their empirical analyses, parental coercion predicted boys' aggressive behavior, gravitation toward peer groups that engaged in hostile talk about women, and physical aggression toward a female romantic partner, in an escalating transaction.

Sameroff and Mackenzie (2003) have suggested that "(t)here is good reason to believe that the onset of substance use and abuse may follow a transactional socialization process similar to the one outlined by Patterson and others for aggression and conduct problems" (p. 623). They cited the need to add even more complexity by accounting for the role of the social ecology in moderating the relations between the child and others. Sameroff and Mackenzie (2003) further noted that part of the problem with empirical studies of complex transactional models is that statistical methods have been applied primarily to, at most, two relationships and three time points (e.g., parenting predicts child behavior, which mediates future parenting). Without explicit empirical testing, Wills and Yaeger (2003) concluded that "(t)he evidence favors a transactional model in which family factors have largely mediated effects

on adolescent substance use through relations to adolescents' self-control, life events, and peer affiliations" (p. 222).

Recent conceptualizations of development of substance use behavior have capitalized on both transactional and dynamic systems theories. Masten, Faden, Zucker, and Spear (2008) called for models of underage drinking that incorporate principles of developmental psychopathology, especially person–environment interactions and transactions, multilevel analysis, and person as agent. They noted, "Underage drinking is a complex issue, deeply embedded in the developmental, multilevel, dynamic processes operating over time within and between individuals and their contexts" (p. S248). The metaphor of *cascades*, as in tumbling water that increases in speed and force as it is altered by, and alters, rocks in its path, captures some of the dynamic and transactional qualities of development that these theorists have proposed. Masten et al. (2005) were among the first to use the term "developmental cascade" to describe the relation between academic achievement and behavior problems in children across a 20-year period of development.

The current monograph represents one of the first attempts to broaden empirical tests of a transactional model to multiyear time points and relationships. We propose a dynamic cascade model, in which early ecological and child factors set in motion a chain of events that unfold, grow, and magnify over time into serious problem behavior in adolescence, in much the way that a stream meandering down a meadow joins other streams and gathers momentum and grows into a rushing rapid that cascades over rocks, carrying the rocks with it, albeit with various deflections from the rocks along the way. The rocks become part of the growing force that, by the end of the journey, leads to an outcome that in retrospect seems inevitable. In this effort, we attempt to provide an empirical, social–developmental realization of Waddington's (1962) germinal notion of an epigenetic landscape.

Our model involves transactional relations among the child, parents, and peers across development. Specifically, the model posits that early ecological and child risk factors make it difficult for a parent to parent effectively. Dysfunctional parenting, in turn, influences the young child to behave incompetently and disruptively upon school entry. This behavior pattern has an adverse effect on peers, who reject the child and increase conflict with him or her. These conflicts cause stress for the parent, which paradoxically leads the parent to withdraw from supervision, monitoring, and communication with the early-adolescent child just at a time when the child needs these parenting behaviors the most. In turn, the parent's withdrawal affords the youth the opportunity to associate in unfettered ways with deviant peers, which potentiates the onset of illicit substance use. We propose to test each of the hypotheses stipulated in this model with a novel application of partial least-squares (PLS) modeling. The result of this specific hypothesizing and empirical testing is a model that reduces a welter of correlations into a coherent dynamic developmental model.

DEVELOPMENTAL PATTERNS IN ADOLESCENT SUBSTANCE USE

By age 18, 72% of American adolescents report having tried alcohol, 55% report having been drunk, and 49% report using an illicit substance such as marijuana or inhalants (Johnston, O'Malley, Bachman, & Schulenberg, 2008). Plots of hazard rates by age-of-onset of these substances indicate that very few children initiate use before age 8. Risk of onset is particularly steep for each year between ages 10 and 18 and then declines sharply thereafter (Johnston et al., 2008; Kandel & Logan, 1984; Kandel & Yamaguchi, 1985).

These trajectories vary for different substances, but most substance users have begun use before the end of adolescence. Smoking almost always begins in adolescence: 89% of adult daily smokers began using cigarettes by or at age 18. In fact, 71% of adult smokers say that their smoking had become daily by adolescence (U.S. Department of Health and Human

Services, 1994). Risk for alcohol consumption increases sharply in adolescence (Chassin, Flora, & King, 2004), precedes risk for marijuana use by several years (Kosterman et al., 2000), and declines more sharply after age 18 (Kandel & Yamaguchi, 1985). Cloninger (1987) described two types of alcoholism disorders: Type 1, called late-onset, begins in the mid-20s (although use begins in adolescence), and Type 2, called early-onset, begins in adolescence. Personality variables distinguish between these types (Cloninger, Sigvardsson, & Bohman, 1988). Donovan's (2007) review of the various surveys identified a group of 3–8% of youth who initiate alcohol consumption on at least a weekly basis at a very early age (before age 12). He highlighted this group for further inquiry and possible preventive intervention.

In some empirical studies, measures of consumption of alcohol, tobacco, and other drugs have been combined into a single construct called ATOD (e.g., Needle, Su, & Lavee, 1989; Newcomb & Bentler, 1988; Wills, Sandy, Yaeger, & Shinar, 2001) or weighted-persistent substance use (Loeber, Stouthamer-Loeber, & White, 1999), and have even been combined with a broader array of behaviors to form a "problem behavior syndrome" (e.g., Jessor & Jessor, 1977). In contrast, the different prevalence rates, life-course patterns, and legal consequences of each behavior suggest that developmental analyses might keep them distinct (e.g., Dishion, Capaldi, & Yoerger, 1999; Jackson, Hendrickson, Dickinson, & Levine, 1997; Kosterman et al., 2000; Masse & Tremblay, 1997). We assert that the extent to which a substance-use construct aggregates scores across tobacco, alcohol, and illicit substances, as well as age of use, will diminish the likelihood of discovering important developmental–ecological factors in substance-use onset. Although similar logic might suggest that distinctions should be made within the group of illicit substances, the high degree of overlap in use and the relatively low base rates of single-type users suggest merit in studying the group of any illicit substance users. The current study focuses exclusively on any illicit substance use (i.e., marijuana, cocaine, heroin, inhalants, and other illegal drugs) annually from age 12 to 18, allowing us to distinguish early versus later onset during adolescence.

EARLY VERSUS LATER ONSET OF ADOLESCENT SUBSTANCE USE

Several perspectives suggest the importance of distinguishing early from later onset of substance use, including different prevalence rates, different long-term outcomes, and possibly different etiologies. The nearly ubiquitous nature of late-teenage drinking implies that merely experimenting with this behavior in a senior high school cultural context of social drinking is not a strong predictor of adult problem outcomes, even though almost all adult alcoholics begin drinking before adulthood. As Clark and Winters (2002) concluded, "(E)xperimentation with alcohol, tobacco, and other drugs is part of the normal developmental trajectory for adolescents" (p. 1214). However, early initiation of drinking or illicit substance use (during elementary or middle school) may be especially diagnostic of later problem outcomes. The U.S. nationwide Monitoring the Future Study indicates that, by the eighth grade, 39% of youth report drinking alcohol and 19% report using marijuana (Johnston, O'Malley, Bachman, & Schulenberg, 2008). In French-speaking Montreal, Canada, by age 15 years, 48% of boys report being drunk in the past year and 31% report using some other drug (Masse & Tremblay, 1997). Donovan's (2007) review identified a group of 3–8% of youth who initiate alcohol use even earlier, before sixth grade. These lifetime prevalence rates indicate that onset of substance use grows across adolescence, with groups of very-early users, early users, and normative users.

In a different but related domain, consensus understanding of antisocial behavioral development distinguishes early-starting conduct problems from adolescence-initiated delinquency (Dodge, Coie, & Lynam, 2006; Dodge & Pettit, 2003; Moffitt, 1993; Patterson, Capaldi, & Bank, 1991). The former is hypothesized to be more serious, longer lasting, and

impervious to treatment, whereas Moffitt (1993) has described the latter as socially normative.

The normativeness of high school drinking and substance use suggests that a similar distinction between early-onset substance use (which is initiated in a social context in which such behavior is deviant) and adolescence-onset substance use might be important. Although a sharp distinction by age of onset has not yet been formally applied to illicit substance use, empirical evidence indicates that the earlier one initiates substance use the graver the consequences. Early age of alcohol initiation is strongly linked to later alcohol misuse (Hawkins et al., 1997), progression to other drugs (Kandel, Yamaguchi, & Chen, 1992), lifetime alcoholism (Yu & Williford, 1992), and other problem behaviors (Gruber, DiClemente, Anderson, & Lodico, 1996; Robins & Przybeck, 1985). Likewise, early initiation of illicit substance use is a predictor of later use of other substances (Ellickson, Hays, & Bell, 1992), substance abuse (Kandel & Davies, 1992), and a variety of problem outcomes, including educational underachievement and unemployment (New-comb & Bentler, 1988), antisocial behavior (van Kammen & Loeber, 1994), and general maladaptation in adulthood (Kandel, Davies, Karus, & Yamaguchi, 1986). Although later-onset illicit substance use is relatively normative, it is still costly and may also have origins in experience factors that could be avoided. Whether the experience factors differ for early versus later onset is not yet known. The current study included an age-of-onset parameter to test whether predictors of onset vary with age of onset.

UNDERSTANDING EARLY-ONSET SUBSTANCE USE

Whereas later adolescence-onset substance use has received relatively little attention, explaining early-onset substance use and identifying unique predictors of early- versus late-onset substance use are matters of great controversy. Cloninger (1986, 1987) has argued that early-onset alcohol use is due to an inherited personality pattern that consists of high novelty seeking, low harm avoidance, and low reward dependence (defined as responsiveness to social rewards and not as impulsive reward sensitivity). These characteristics reflect actions of neurally mediated behavioral activation, inhibition, and maintenance systems, respectively.

Evidence consistent with this theory is plentiful. In a sample of 431 Swedish males, these three personality dimensions were significantly related to early-onset alcoholism (Cloninger, Sigvardsson, & Bohman, 1988). Wills, Vaccaro, and McNamara (1994) found that these characteristics predicted early-onset cigarette smoking, alcohol use, and marijuana use. Pomerleau, Pomerleau, Flessland, and Basson (1992) found that novelty seeking and harm avoidance, but not reward dependence, were correlated with cigarette smoking in adulthood. Masse and Tremblay (1997) found that novelty seeking and harm avoidance, but not reward dependence, measured by teacher ratings at age 6 predicted self-reports of early-onset alcohol and marijuana use between ages 10 and 15 years. The theoretical thrust of these findings is that core personality characteristics are responsible for early-onset substance use. Implied is the assertion that environmental events exert little impact on substance-use development.

In contrast, Dishion, Capaldi, and Yoerger (1999) have offered a more ecological perspective. They suggest that features of the home, school, and neighborhood settings (such as stigmatization, victimization, behavioral norms, and economic resources) provide a context that leads to early behaviors (such as antisocial behavior, negative affect, and problematic temperament) that might appear as “inherent” child characteristics. The same settings were hypothesized to foster the development of substance use. Furthermore, Dishion et al. (1999) hypothesized and found that family management practices of harsh discipline and poor monitoring and peer experiences of social rejection and association with deviant

friends have a direct impact on the development of early-onset marijuana use and partially mediate the effect of early context factors on marijuana use. Unfortunately, their measures of child characteristics were confounded in time with their measures of parenting, such that strong associations between a child's antisocial behavior at age 9 and early-onset substance use between ages 10 and 15 could not be interpreted definitively. They concluded that "both genetic and environmental theorists might endorse these findings as supportive" (p. 199). Furthermore, they could not distinguish (either in time or statistically) the separate impacts of family versus peer experiences on substance-use development. They ultimately aggregated all of these factors into a parsimonious but theoretically unsatisfying single construct that they called "childhood risk structure" that accounted for 34% of the variance in substance use. The current study offers time-specific measurement of key constructs in an ecological model in order to test the unique role of each factor.

REVIEW OF RISK FACTORS FOR YOUTH SUBSTANCE USE

The following brief review of the literature of the various types of risk factors provides a context for our proposed comprehensive model of the development of early-onset substance use. Emphasis is given to prospective studies and replicated findings. The review will appear more like a laundry list of risk factors than an integrated developmental story, and so the section following the review will weave the risk factors together into a developmental model.

Child Factors—Heritability has been posited as a driving force in problematic and early-onset substance use. The evidence is consistently supportive for genetic effects on alcoholism in males but not in females. Twin studies (Hrubec & Omenn, 1981) reveal higher concordance among male monozygotic twins than dyzogotic twins, and adoption studies (Cadoret, Cain, & Grove, 1980) indicate rates of alcoholism up to 27% for adopted sons of alcoholics compared with only 6% for adopted males without a biologic alcoholic parent. However, studies that include females have found no such effects (Murray & Stabenau, 1982), and studies evaluating genetic transmission of early-onset illicit substance use have yet to reveal consistent patterns (Hawkins, Catalano, & Miller, 1992).

Dick et al. (2006) have identified specific genes (e.g., GABRA2) that place adolescents at risk for substance-use problems, but they noted that genetic risk is confounded with risk for other conduct problems and probably represents a general genetic factor of deviance proneness. More likely than a direct genetic effect on illicit substance use is a genetic effect on cognitive and physiological factors that affect the development of a variety of deviant behaviors including substance use. A heterogeneous set of genes may be related to a heterogeneous set of cognitive biases toward immediate reward and sensation seeking as well as molecular markers of tolerance or susceptibility to addiction (Dick et al., 2006; Institute of Medicine, 1994; Nestler & Landsman, 2001; Tarter et al., 1999). Thus, a genetically informed developmental model of substance use might include measures of early conduct problems as precursors of later substance use and must wrestle with a possible common genetic cause.

But a common genetic cause does not rule out a role for environmental influences in determining which form of deviance occurs and in accounting for the link between early conduct problems and later substance use. Furthermore, more empirically striking than genetic main effects are gene–environment interaction effects (Dick et al., 2009) that indicate that genetic expression occurs only in the context of specific environments. The genetic findings mirror behavioral discoveries in adolescent developmental psychology: Life events shape the form of deviance that is expressed. This conceptualization suggests that heritable risk requires specific life experiences to potentiate and mediate the risk.

The complexity and ambiguity of mechanisms of heritable risk are highlighted in robust empirical findings that living with a parent who abuses alcohol or illicit substances increases risk of early-onset substance use (Merikangas et al., 1998; Weinberg & Glantz, 1999). Parental alcoholism (Cloninger, Bohman, Sigvardsson, & von Knorring, 1985; Goodwin, 1985) and substance use (Brook et al., 1990; Hops et al., 1990; Johnson, Schoutz, & Locke, 1984) substantially increase a child's likelihood of early-onset alcohol use (Chassin, Curran, Hussong, & Colder, 1996) and illicit substance use (Costello, Erkanli, Federman, & Angold, 1999). Dishion, Capaldi, and Yoerger (1999) followed 206 boys in the Oregon Youth Study and found that parental alcohol use and marijuana use (but not over-the-counter drug use) when the boys were in fourth grade significantly predicted boys' alcohol and marijuana use by age 15. Similarly, Kaplow et al. (2002) followed 387 kindergarten boys and girls from four geographic sites and found that parental substance use predicted children's substance use by age 12. Although parental substance use indexes an empirically important risk factor, the causal mechanism of this effect is unclear and could variously reflect genetic influences, a family context of psychopathology, or parental modeling of deviant behavior.

Whether markers of genetic risk or not, constitutionally endowed temperament and early behavior-problems constructs have been posited as child risk factors for alcohol and substance use (Tarter & Vanyukov, 1994). After reviewing the evidence from extant longitudinal studies of behavioral development, Zucker (2006a) noted, "This work, coming from six long term prospective studies carried out over the past quarter century, provides a remarkable convergence with the genetic literature in demonstrating that externalizing symptomatology appearing in early childhood is predictive of SUD (substance use disorder) outcomes some 15–20 years after the first appearance of the drug-nonspecific behavioral risk" (p. 616).

Two temperament factors derive from Gray's (1987) theory of neural control. A strong behavioral activation system is reflected in exhilaration that is activated by novel stimuli (high novelty seeking). The behavioral inhibition system adaptively heightens responsiveness to aversive stimuli, and a weak system will fail to facilitate the inhibitive behaviors that avoid harm (low harm avoidance). Zuckerman (1987) has found that novelty seeking (which he calls sensation seeking) is linked biochemically to low platelet monoamine oxidase activity, which is correlated with early-onset alcoholism (Tabakoff & Hoffmn, 1988). Cloninger et al. (1988), Pomerleau et al. (1992), and Wills et al. (1994) all found associations between both of these factors and substance use. Most impressively, Masse and Tremblay (1997) reported that these two factors assessed at age 6 predicted onset of alcohol and illicit substance use between 10 and 15 years of age.

Other related temperament factors, including high activity level, negative withdrawal responses to new stimuli, arrhythmicity, rigidity, and distractibility, have been found to be significantly correlated with adolescent substance use (Weinberg & Glantz, 1999; Wills, DuHammel, & Vaccaro, 1995; Windle, 1991). Using the Revised Dimensions of Temperament Survey (Windle & Lerner, 1986) and the Emotionality, Activity, and Sociability Inventory (Buss & Plomin, 1984), Wills et al. (2001) found that a temperament composite of high activity level and negative emotionality correlated significantly with a combined measure of self-reported ATOD use among sixth, seventh, and eighth graders surveyed in school. Because the substance use measure combined types of substances, it is not clear whether the temperament composite related significantly to illicit substance use. Likewise, prospective follow-up of 5-year-old children into young adulthood revealed that early difficult temperament, characterized by slow adaptability to change, negative mood, and withdrawal responses to new stimuli, predicted adolescent ATOD use (Lerner & Vicary, 1984).

Dishion et al. (1999) found that mothers' nine-item ratings of a child's "early difficulties" in the first 5 years of life (e.g., sleep problems, physical development problems) predicted later alcohol and marijuana use. In a major review, Zucker (2006b) concluded that a child factor of externalizing symptoms displayed early in life places a child at risk for later substance use problems, but this factor interacts and transacts with other factors across development to determine whether the form of externalizing disorder involves substance abuse or other behaviors.

In spite of the strong gender and race associations with externalizing behaviors (Dodge, Coie, & Lynam, 2006), and the strong associations between externalizing problems and substance use, substance use (particularly late-onset use) does not show similar associations with ethnicity and gender. Unlike studies of conduct problems (which find that African Americans are at greater risk than European Americans; Dodge et al., 2006), surveys indicate slightly lower rates of substance use among African American adolescents than European American adolescents (Costa, Jessor, & Turbin, 1999; Johnston, O'Malley, & Bachman, 1995; Maddahian, New-comb, & Bentler, 1988), or no differences (Chilcoat & Anthony, 1996; Wills et al., 2001). In contrast, Kosterman et al. (2000) found higher rates among African American youth than European American youth. Kaplow et al. (2002) found that African American children are at higher risk than European Americans for very early-onset (sixth grade) alcohol and substance use. It may be that minority ethnicity (and its environmental disadvantages) is correlated with early-onset substance use but not more normative use in adolescence.

Whether predictors of substance use vary across ethnic groups is a matter of debate, with some studies indicating that peer factors play a relatively stronger role in cigarette smoking for European Americans (Landrine et al., 1994), whereas family factors play a stronger role in illicit substance use for African Americans (Krohn & Thornberry, 1993). Bray, Adams, Getz, and McQueen (2003) found no ethnic group differences in the role of peers' behavior in adolescents' alcohol use, and Gottfredson and Koper (1996) found very few differences in risk factors for substance use among 981 African American and White 6th–10th graders.

Males appear to be at greater risk than females for early-onset alcohol and illicit substance use (Costello et al., 1999; Kaplow et al., 2002; Liu & Kaplan, 1996; Thomas, 1996) and for serious substance-use disorder that is comorbid with other psychiatric disorders (Kandel et al., 1997; Lewinsohn, Rohde, & Seeley, 1995). However, the differences tend to be so small that Armstrong and Costello (2002) concluded that "the similarities between the sexes have been more remarkable than the differences" (p. 1234). Recent data show that girls' use is almost equal to that of boys, particularly at younger ages (National Center on Addiction and Substance Abuse, 2003). Data from the National Household Survey on Drug Abuse show an increase in alcohol initiation among early adolescent girls (Substance Abuse and Mental Health Services Administration, 1997). In addition, while the age of first usage is getting younger for both boys and girls, it is dropping at a faster rate for girls. Three decades ago, initiation of alcohol use in the group of young teen girls ages 10–14 was only 7% but has grown in the last decade to 30.9%. This increase for young girls' initiation rates is compared with a relative increase from 20.2% to 35.4% for boys.

It was concluded that measures of gender, temperament, and early behavior problems are essential child risk factors for empirical analysis in the current study, but measures of later behavior problems probably confound genes and life experiences so much as to be less useful.

Early Family Social–Ecological Factors—Coexistent with child risk factors for early-onset illicit substance use is social–ecological factors within the family context during the

child's early life. Although there is outdated evidence that high parental education and upper income levels are associated with slightly greater marijuana use among high school seniors (Bachman, Lloyd, & O'Malley, 1981; Zucker & Harford, 1983), extreme economic poverty is also a risk factor for alcohol and illicit drug use (Robins & Ratcliff, 1979). More recently, Costa, Jessor, and Turbin (1999) and Dishion, Capaldi, and Yoerger (1999) found a negative association between family socioeconomic status and problem drinking in adolescence. Kaplow et al. (2002) found that children from the lowest socioeconomic-status group were at higher risk for very early-onset (before age 13) alcohol or substance use than other children.

In addition to family socioeconomic disadvantage, other early family contexts that have been demonstrated to enhance risk for early-onset substance use include being reared in a family missing a biological parent (Costa, Jessor, & Turbin, 1999), parental disorganization and emotional instability (Block, Block, & Keyes, 1988; Brook et al., 1990), and parental stress as indexed by child care problems, family medical conditions, unemployment, and the ratio of children to adults in the household (Dishion et al., 1999).

Early Parenting and Caregiving Factors—As found by Dishion et al. (1999), the development of early-onset substance use is more directly predicted by family interactions that a child experiences during his or her early years than the context into which that child is born. The ecological context might lead to family interactions that account for the impact of the context on development. The most-studied early parenting behavior is discipline style. Dishion et al. (1999) observed parent–child interaction at home at age 9 and indexed a poor-discipline factor that included nattering (ineffectual and annoying talking), abusive parenting (verbal attacks, physical strikes, and threats), and erratic discipline practices. This poor-discipline construct predicted boys' alcohol and illicit substance use by age 15.

At the extreme of harsh discipline is physical abuse. Child maltreatment, which encompasses physical abuse, neglect, and sexual abuse, has been found to pose risk for substance use, especially substance-use disorder (Kilpatrick et al., 2000; Widom, Ireland, & Glynn, 1995). Early sexual abuse enhances risk for substance-use problems in girls (Kendler et al., 2000) and boys (Clark, Lesnick, & Hegedus, 1997). Physical abuse in the absence of sexual abuse also poses enhanced risk for substance-use problems (Kaplan et al., 1998), although not as strongly. Distinct from a harsh discipline style are nonviolent discipline practices that involve verbal reasoning and discussion. Kaplow et al. (2002) found that reasoning and discussion styles of discipline protect children from early-onset substance use, and Kosterman et al. (2000) found that a proactive family management style protected children from subsequent marijuana use.

Yet another relevant early parent-interaction factor is warmth and involvement between parent and child. Kandel and Andrews (1987) and Penning and Barnes (1982) found that lack of maternal involvement with a child increases risk for substance use. Shedler and Block's (1990) direct observations of mothers' cold nonresponsiveness and lack of encouragement of their child at age 5 predicted frequent marijuana use in adolescence. Kaplow et al. (2002) found that parents' lack of involvement in their kindergarten child's education at school also predicted later substance use. Brook et al. (1990) reported a causal pathway in which early strong parent–child attachment led to the child's internalization of mainstream norms and values, which, in turn, led the child to associate with nondeviant peers and to nonuse of drugs.

Other early parenting behaviors that have been associated with the child's onset of illicit substance use include parental inconsistent permissiveness (Baumrind, 1983), mothers' unclear rules for child behavior (Brook et al., 1990), and lack of family rules about daily

chores, homework, and so on (Costa, Jessor, & Turbin, 1999). Parental failure to discourage deviant behavior early in life (the inverse is sometimes labeled as parental approval for drug use, although few parents directly encourage substance use before age 15) has been associated with adolescent substance use in numerous studies (Barnes & Welte, 1986; Brook, Gordon, Whiteman, & Cohen, 1986; Hansen et al., 1987) that span multiple ethnic groups (Jessor, Donovan, & Windmer, 1980). Less direct early parenting behaviors that enhance risk for a child's early-onset substance use include their modeling of deviant behavior (see the findings regarding parental substance use noted above), including their modeling of marital discord (Simcha-Fagan, Gersten, & Langner, 1986) and their numerous marital transitions since the child's birth (Dishion et al., 1999).

Early Child Behavior Factors—The child's early behavior-problem levels most likely both reflect the genetically based child-factor contribution to later problems and grow from parenting practices (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000). Of all early child behaviors that have been examined in this context, aggression toward peers has been most consistently predictive of later substance use. Kellam, Ensminger, and Simon (1980) found that aggressive behavior in the first-grade classroom predicted later drug use, whereas shyness did not (unless coupled with aggressive behavior). Kaplow et al. (2002) reported a similar relation for first-grade aggressive behavior as indexed by parents' daily reports, and Dishion et al. (1999) found a similar relation for fourth-grade antisocial behavior. Supportive findings have been reported by Boyle et al. (1992), Lewis, Robins, and Rice (1985), and Reinherz et al. (2000), among others.

McMahon et al. (2000) examined the role of psychopathology assessed in kindergarten and 1st grade in predicting initiation of tobacco use in Grades 4–7. Discrete-time survival analyses indicated that children exhibiting psychopathology of one or more types (e.g., conduct disorder, attention-deficit hyperactivity disorder [ADHD]) show a two- to threefold increase in risk of onset of tobacco use by Grade 7. Both community studies (Armstrong & Costello, 2002) and clinical studies (Clark, Parker, & Lynch, 1999; Disney, Elkins, McGue, & Iacono, 1999) show that early disruptive behavior disorders temporally precede eventual early-onset substance use. In fact, of all child psychiatric disorders that have been linked to adolescent substance use, conduct disorder stands out as the most consistent and strongest marker of risk (Glantz & Leshner, 2000), so much so that Glantz (2002) has called for randomized trials of interventions to reduce conduct disorder as a test of substance-abuse prevention.

ADHD (Mannuza, Klein, Bessler, Malloy, & LaPadula, 1998) has also been linked to later substance-use problems, but this relation has been attributed to its comorbidity with conduct disorder. Farmer, Compton, Burns, and Robertson (2002) concluded that "ADHD may indirectly increase risk of substance use disorders by increasing risk for antisocial disorders" (p. 1267). Likewise, early medication treatment for ADHD has been correlated with early-onset substance use (Kaplow et al., 2002), but medication may be a risk factor simply because it marks the presence of ADHD.

Internalizing behaviors have also been correlated with substance-use problems in adolescence (Kandel et al., 1999) and may immediately precede substance use in the short term (Deykin, Buka, & Zeena, 1992), but little evidence exists that internalizing problems early in childhood mark risk for substance use. In fact, early anxiety and other internalizing symptoms in the absence of disruptive behavior may actually protect a child from later alcohol use (Kaplow, Curran, Angold, & Costello, 2001), substance use (Kaplow et al., 2002), and tobacco use (Costello et al., 1999), perhaps because internalizing behaviors may prevent a child from interaction with a peer group that exposes the child to substances and substance-use culture.

Early Peer Relations Factors—As important in substance-use development as the child's behavior toward others is the reaction of the peer group to that behavior. Kaplow et al. (2002) found that social rejection by the first-grade peer group, indexed by the social preference score (number of liking nominations minus number of disliking nominations), predicted very early-onset illicit substance use. Likewise, Dishion, Capaldi, Spracklen, and Li (1995) used the same score collected in fourth grade and found that it predicted tobacco, alcohol, and marijuana use by age 15.

Transactional theory (Sameroff, 2009) suggests that features of the child's behavior toward peers during this period may lead to the peer group's reactions and may also be a response to peer group rejection, but in either case both the child's behavior and the peers' reactions have been found to be important markers of later substance use. Whether the peer group's reactions increment the prediction of later substance use beyond the child's behavior has not been tested sufficiently. Greene et al. (1997, 1999) found that early social impairment predicted later substance-use disorder even after controlling for conduct disorder, other psychiatric disorders, and social class.

Early social competence, indexed in various ways, has been a consistent protective factor in substance-use development, even after controlling for conduct problem behavior. Jackson et al. (1997) found that third- and fifth-grade children with low teacher-rated social competence (separate ratings of social skills, self-confidence, and academic abilities) were at least twice as likely to report early use of alcohol as children with high competence. Other measures of social competence that have uniquely predicted later substance use include social problem-solving deficits and hostile attributional biases measured by responses to hypothetical vignettes (Kaplow et al., 2002), poor behavioral self-control skills (Griffin, Botvin, Epstein, Doyle, & Diaz, 2000), and expectations and aspirations for success in life (Costa, Jessor, & Turbin, 1999; Newcomb & Felix-Ortiz, 1992).

The particular importance of social competence is highlighted by non-robust effects of two related constructs in predicting substance use: self-esteem and intelligence. Measures of self-esteem have yielded contradictory findings. Although Costa et al. (1999) found that low self-esteem marked risk for alcohol use, Dishion et al. (1999) found no such relation. Likewise, measures of intelligence are not consistently predictive of substance use. *High* scores on intelligence tests predicted earlier and more frequent use of alcohol in an inner city sample (Fleming, Kellam, & Brown, 1982); in contrast, *low* scores on the Wechsler Intelligence Scale for Children — Revised in first grade predicted earlier substance use in Kaplow et al.'s (2002) four-site sample. In their review, Hawkins, Catalano, and Miller (1992) concluded that "(t)he available evidence suggests that social adjustment is more important than academic performance in the early elementary grades in predicting later drug abuse" (p. 84).

Although intelligence is probably not predictive of substance use, social–behavioral factors that are related to schooling have been identified as risk factors for substance use, including a low degree of commitment to school (Johnston, O'Malley, & Bachman, 1985), disliking of school (Kelly & Balch, 1971), academic underachievement (Dishion et al., 1999), and truancy (Gottfredson, 1988).

Parenting and Sociocultural Factors in Early Adolescence—As a child moves into early adolescence, parenting factors continue to mark risk for substance-use development, but the relevant parenting factors shift away from harsh discipline to overall supervision, monitoring, and control of the youth's sociocultural environment. Chilcoat and Anthony (1996) followed 926 8–10-year-old urban-dwelling children into adolescence and found that a 10-item child-report measure of parental supervision and monitoring predicted

later marijuana, cocaine, and inhalant use. Furthermore, decreases in parental monitoring across time signaled a subsequent increase in risk of initiating illicit substance use. Parental knowledge of a child's whereabouts, activities, and friends has been found to predict ATOD initiation (Barnes, Reifman, Farrell, & Dintcheff, 2000; Dishion et al., 1995; Flannery, Vaszonyi, Torquati, & Fridrich, 1994; Fletcher, Darling, & Steinberg, 1995).

Numerous studies support the importance of parental monitoring in protecting early adolescents from moving toward substance use (Baumrind, 1985; Dielman, Butchart, Shope, & Miller, 1991; McCarthy & Anglin, 1990), although Dishion et al. (1999) were surprised that their measure of monitoring, which predicted tobacco use, did not reach significance for marijuana use.

Stattin and Kerr (2000; Kerr & Stattin, 2000) have called into question the importance of parental monitoring behavior by pointing out that monitoring knowledge is less an outcome of parents' monitoring behaviors than it is of unsolicited child disclosure. Eaton, Krueger, Johnson, McGue, and Iacono (2009) took this point a step further by finding that child personality characteristics account for the effect of monitoring knowledge on child outcomes. The distinction between monitoring as knowledge (of whereabouts, companions, etc.) and monitoring as process (through what means do parents acquire this information) is now widely accepted. Furthermore, the distinction between monitoring and control/supervision is crucial. Fletcher, Steinberg, and Williams-Wheeler (2004) distinguished among parental knowledge, monitoring, and control behaviors in a longitudinal study of 2,568 adolescents. Their measure of control/supervision consisted of six youth-reported items: (1) How late at night I can stay out; (2) Which friends I spend time with; (3) How I spend my money; (4) Whether or not I can drink alcohol; (5) How much time I spend with friends; and (6) When I can start dating. They found that even controlling for knowledge and child disclosure measures, parental control/supervision behaviors significantly incremented the prediction of future substance use.

It is possible that parental supervision is especially important in particular ecological settings, such as neighborhoods that provide ready access to drugs, or for particular children who are prone to deviance. Pettit, Bates, Dodge, and Meece (1999) found that parental supervision predicted adolescent aggressive behavior, but only in high-risk neighborhoods and only for high-risk children. Similarly, Beyers, Bates, Pettit, and Dodge (2003) found that the protective effect of parental monitoring was more striking for those living in neighborhoods that census data indicated are high in residential instability and, thus, likely to have fewer informal social controls on children.

Finally, quality of the parent–child relationship in early adolescence has been found to mark risk (Hundleby & Mercer, 1987). Hawkins et al. (1992) refer to "low bonding to family" as the critical construct in characterizing early adolescents who are at risk for becoming involved with illicit substances.

Adolescent Peer Relations Factors—In early adolescence and throughout puberty, rapid brain development renders youths especially vulnerable to the attraction of immediate rewards and sensational stimuli (Dahl & Spear, 2004). During this era of life, peers provide pressure to seek immediate gratification and offer exposure to sensational stimuli, including drugs. In addition, peers grow in influence as parents' influence wanes; thus, adolescent peer relations factors are strong predictors of substance-use initiation (Bogenschneider, Wu, Raffaelli, & Tsay, 1998). Like parenting, though, the important aspect of peer relations changes. The predictive factor shifts away from overall acceptance by the mainstream peer group to association with deviant peers (Dishion & Owen, 2002). Early-adolescent involvement with peers who display deviant behavior, especially substance use, is perhaps

the strongest predictor of subsequent initiation of substance use (Hawkins et al., 1992), presumably through processes of peer norms, modeling, and pressure.

Of course, deviant behavior by an adolescent might lead that youth to gravitate toward deviant peers, which would indicate a selection factor rather than causal impact of association with deviant peers. However, Gatti, Tremblay, Vitaro, and McDuff (2005) found that deviant influences in gang membership during midadolescence (ages 14–16) facilitated substance use, even controlling for selection factors into gang membership.

Transactional effects and reciprocal influences are plausible. Bray, Adams, Getz, and McQueen (2003) found that tenuous efforts to individuate from parental influence may even accentuate peer influences on alcohol use in adolescence. Furthermore, they found that the tendency to affiliate with deviant peers and one's own alcohol use reciprocally influenced each other over time, suggesting a spiraling path toward deviance across Grades 7–9.

Allison et al. (1999) found that middle and high school peer-group norms regarding the frequency and acceptability of alcohol and drug use were significant predictors of individual alcohol and drug use. Both actual peer-group drug use and the individual's perception of peers' drug use were unique predictors, suggesting that both peer behaviors and the youth's beliefs about peers contribute to drug use. A related mechanism through which association with deviant peers may increase substance use may be social in nature. Dishion et al. (1999) suggest that "(s)moking may serve as a mechanism by which boys with troubled peer relations have commerce in a peer group … One may hypothesize that early onset smoking is a peer adaptation and has functional use in the life of the at-risk youth" (p. 199). Supportive findings come from studies across a wide range of ethnic groups and geographic contexts (Brook et al., 1990; Elliott, Huizinga, & Ageton, 1985; Griffin et al., 2000; Jessor et al., 1980; Kandel & Andrews, 1987; Kosterman et al., 2000).

In spite of these strong correlations, the causal status of deviant-peer influence remains under debate. As noted, the relation between youths' own deviance and peers' deviance has been found to be reciprocal. Changes in peers' use of substances increase a child's risk for substance-use initiation, but a child's initiation of deviant behavior also influences the peer group (Curran, Stice, & Chassin, 1997). Second, gravitation toward deviant peers is predictable from earlier peer rejection and externalizing problems (Laird, Jordan, Dodge, Pettit, & Bates, 2001) and negative affectivity (Shoal & Giancola, 2003), suggesting that association with deviant peers might be epiphenomenal to substance-use development unless these factors are controlled. Third, strong relationships with one's parents have been found to buffer a child from the effects of associating with peers who are deviant (Brook et al., 1990). Finally, not all early adolescents are susceptible to peer influence, even in the context of association with deviant peers. Goodnight et al. (2006) found that adolescents who are low in characteristics of impulsivity/reward sensitivity are relatively less susceptible to peer influences on delinquent behavior. The concept of "resistance efficacy" has been introduced to understand moderation effects of deviant peer associations and has been targeted by prevention programs as a way to enhance resistance to substance use (Botvin, 1986).

Nonetheless, association with deviant peers who provide exposure to drugs, use drugs, and act as models for how to use drugs represents the most proximal pathway to onset of substance use in the proposed cascade model.

THEORETICAL AND EMPIRICAL INTEGRATION

Both the diversity and redundancy in a list of risk factors call for empirical and theoretical integration. Rutter and Garmezy (1983) proposed a risk-factor counting approach, which has been used successfully to optimize the number of predictor variables in empirical models.

For example, Kaplow et al. (2002) used an array of variables to find that, although any single risk factor increased risk of adolescent substance use from <10% (for zero risk factors) to 30%, a child with two risk factors had over 50% risk and a child with three risk factors had over 60% risk.

A counting of risk factors to predict later substance use is still devoid of developmental understanding. Theoretical integration is needed. Most multicomponent developmental theories have been articulated in temporally sequential fashion. Simons et al. (1988) have suggested a multistage social learning model that posits initial risk from parental modeling of substance abusing behaviors, through the child's experimentation, followed by peer-group reward for using substances, and further increases in substance use. Dishion, Capaldi, and Yoerger (1999) also suggest a multicomponent model that includes risk factors of ecological context, family management, and peer environment. Although these models are integrative, they do not account for the full diversity of risk factors reviewed here, they do not account for the different ways that parents and peers influence youth at different points in development, and they do not suggest reciprocal relations in an ongoing transaction between the youth and the social world. Furthermore, they have not been fully tested in longitudinal inquiry.

The approach taken by theorists in developmental psychopathology captures most accurately the series of ongoing transactional effects that describe how child risk factors influence the environment but are also shaped by the environment across time (Dahl & Spear, 2004; Steinberg et al., 2006; Masten et al., 2005). Following this tradition, we propose a dynamic, cascading, multistage, incremental, transactional social learning model that is depicted in Figure 1. This model integrates diverse risk factors in a sequential fashion that posits the manner in which risk factors build upon each other to lead to early-onset substance-use initiation in adolescence.

Other cascading models of social development have been posited recently, in outcome domains of internalizing symptoms (Obradovic & Masten, 2007), externalizing symptoms (Dodge, Greenberg, Malone, & the Conduct Problems Prevention Research Group, 2008), psychiatric disorders (Koot & Timmermans, 2007), and academic competence (Moilanen & Shaw, 2007). These models have some features in common with the current model, but none of the prior models spans as many socializing domains as the current formulation, nor do they utilize the data-analytic approaches that are used here. The current model is described in preliminary form by Dodge et al. (2006).

The model begins with child factors and family socioecological factors in very early life, including temperament as a child factor and demographics and socioeconomic status as ecological factors. The model posits that a child with a difficult temperament and a family history of substance use who is born into a family of poverty and stress, headed by a single, socially isolated, teenage, alcohol-using mother who gives birth following an unplanned pregnancy with medical complications, is at heightened risk for substance use 15 years later. Not all of these factors are necessary, of course. It is posited that these factors place a child empirically at risk for later use of substances. These factors tell us little about the mechanisms through which that development occurs, which is left to subsequent stages.

The next step of the model involves early parenting and caregiving. Following from McLoyd (1990), it is hypothesized that early child and ecological risk factors make it difficult for parents to manage their child effectively. Furthermore, it is hypothesized that inadequate parenting during these early years predicts later deviant behavior, including substance use. Specifically, the model posits that (1) negative parenting experiences in the first 5 years of life are predictable from temperament risk factors and adverse ecological

contexts; (2) negative parenting experiences in the first 5 years of life increase a child's risk for adolescent substance use, above and beyond the risk imposed by previous child and family factors; and (3) these parenting factors partially mediate the effect of previous factors on substance-use development.

Thus, like other models of youth problem behaviors (e.g., Dodge, Pettit, & Bates, 1994a; Sampson & Laub, 1994), it is posited that risks induced by ecological–structural and child factors operate partially through their adverse effect on early caregiving. Children who are born into poverty and adversity are at risk to receive care that is characterized by harsh discipline (even physical abuse), a lack of positive parenting, interparental conflict, exposure to violence, and values that support deviant behavior, with a high rate of nonmaternal (but not paternal) child care. In turn, these caregiving experiences increase a child's risk for using drugs in adolescence and describe the processes through which early poverty and adversity cascade into later substance use.

The proposed model is a cascade, in that problems in early parenting, which partially emerge from previous child and context challenges, propel subsequent processes that account for the predictive effects of early factors. At the next step of the proposed model, children who have received deficient early parenting are likely to enter elementary school displaying aggressive conduct problems. The literature strongly supports the impact of early dysfunctional parenting on growth in aggressive behavior. Furthermore, early conduct problems are among the most robust predictors of adolescent substance use, especially early-onset use. It is hypothesized that the child's early conduct problems predict later substance use, are predicted from early parenting, and mediate the impact of early parenting on later use. The growing model thus moves from child and ecological factors at birth to early dysfunctional parenting to aggressive conduct problems at school entry and ultimately to adolescent substance use. But how do relatively conduct problems in first grade lead to substance use in adolescence?

Caprara, Dodge, Pastorelli, and Zelli (2007) have proposed a theory of how initial marginal deviations in a child can grow into larger problems and disorder over time, through processes of social feedback, self-cognition, and dynamic transactions with the peer and adult environment. A child's aggressive conduct problems often incite negative reactions in peers and teachers, specifically, social rejection by the classroom peer group, which reciprocally exacerbates the child's conduct problems (Dodge et al., 2003). Instead of reacting in a way that would bring the child's behavior back into the mainstream, peers paradoxically push a marginally deviant child toward greater deviance (Caprara, Dodge, Pastorelli, Zelli, & the Conduct Problems Prevention Research Group, 2006). One of the characteristics of dynamic cascade models is that each step of development offers an opportunity for the child or the environment to disrupt the cascade (as in a dam across a river) or to exacerbate the process (as in a waterfall).

It is hypothesized that the experience of peer rejection exacerbates the child's risk for later substance use, beyond the effect of behaviors that led to social rejection. The mechanisms of this impact probably include increases in negative affectivity, as well as reduced opportunity for positive peer influence, although these mechanisms are not empirically tested here. Nonetheless, it is hypothesized that peer social rejection partially accounts for the effect of conduct problems on later substance-use outcomes.

Beyond exacerbating conduct problems, how does peer conflict lead to substance use? Like other transactional models, this model posits that a child's peer relations, which have been influenced by the child's previous behavior and parenting patterns, will, in turn, predict later parenting patterns. Thus, the model posits reciprocal, transactional exchanges between

parenting and peer relations. As the child moves into early adolescence, however, the key tasks of parenting change, and so the relevant features of parenting differ from the relevant features of parenting earlier in childhood. In early childhood when the parent's direct socializing influence is paramount, the key tasks of parenting are behavior management and teaching self-regulatory skills; in early adolescence, when the youth is surrounded by a myriad of other social influences, the key task becomes management of the youth's activities and exposure to other socializing forces.

The proposed model posits that social rejection in the conduct-problem child is likely to exacerbate conflict with his or her parents during the early adolescent years because of the trouble that peer conflict causes at school and in the neighborhood. Repeated peer fights and trouble, leading to parents' unwanted trips to the school, school suspensions, and perhaps disruptions at extracurricular activities and neighborhood centers, wear on parents over time. As a result, the parents become likely to give up attempts at socializing their child and to withdraw from monitoring and supervising their young teen, as Patterson, Reid, and Dishion (1992) have demonstrated empirically. Thus, it is hypothesized that previous child peer relations problems will predict a parental pattern of low monitoring and poor supervision.

It is further hypothesized that poor parental supervision will increase the child's risk for initiating substance use, beyond the risk from peer relations problems, and it is hypothesized that parental monitoring will partially mediate the effect of early peer experiences on subsequent substance use. But what is the interpersonal process through which this effect occurs?

Ironically, just at the developmental era when the high-risk child needs increased monitoring and supervision by parents, the high-risk youth is often left to roam the neighborhood during after-school and weekend hours with no one charting her or his whereabouts. So, the final step of the proposed model involves another set of reciprocal relations. It is hypothesized that adolescents whose parents do not supervise their behavior adequately are likely to gravitate toward deviant peer groups. Thus, adolescent parenting patterns, which had developed partially from earlier peer relations patterns, are hypothesized to influence another aspect of peer relations, association with deviant peer groups. The deviant peer group, in turn, exposes the youth to new deviant activities and culture, including illicit substances. Whether the motive is sensation seeking, self-medication, or group acceptance, the youth's ready access to drugs through the deviant peer group affords the opportunity that makes using drugs a high probability. Thus, it is hypothesized that low parental supervision will predict association with deviant peers. It is also hypothesized that deviant peer associations, in turn, will predict the onset of substance use, even after controlling for parental supervision. Finally, it is hypothesized that deviant peer associations will partially mediate the impact of adolescent parenting problems on later substance use.

The model incorporates the major risk factors at various developmental eras and ties them together in a theoretically sensible, although perhaps not obvious, manner. Each risk factor builds on previous risk factors by both mediating the impact of previous risk factors on later development and incrementing that risk through a new interpersonal process. The major players of parent, youth, and peers are always involved but in evolving ways that reflect the youth's stage of development. The combination of all risk factors provides a powerful empirical prediction of adolescent substance use and a plausible account of how these factors operate on the youth.

GENDER

Numerous studies have consistently found that males are at greater risk than females for both early-onset substance use (Bray, Adams, Getz, & McQueen, 2003; Kaplow et al., 2001)

and later-onset substance use (Siebenbruner, Englund, Egeland, & Hudson, 2006). However, the literature is much less clear about gender-specific predictors of substance use. Gottfredson and Koper (1996) found little evidence to support gender specificity in a longitudinal study of 981 youth using measures of parental supervision, peer influence, and self-efficacy as predictors. All of these factors predicted frequency of drug use, and the strength of the relations did not vary across gender. We did not propose the developmental cascade model in a gender-specific manner. Nonetheless, in the current study we tested the hypothesis that models of prediction of substance-use trajectories would differ across gender groups.

EMPIRICAL HYPOTHESES

The model in Figure 1 suggests seven sets of hypotheses, each pertaining to one of the seven predictor domains for adolescent substance use outcomes. When possible, within each set, five noncompeting, complementary hypotheses were tested that followed similar logic:

Ha1: *Domain*$_i$ will predict adolescent substance use.

Ha2: *Domain*$_{i-1}$ will predict adolescent substance use.

Ha3: *Domain*$_{i-1}$ will predict *domain*$_i$.

Ha4: *Domain*$_i$ will mediate partially the impact of *domain*$_{i-1}$ on adolescent substance use.

Ha5: *Domain*$_i$ will increment the prediction of adolescent substance use beyond *domain*$_{i-1}$.

First, variables in a domain (*domain*$_i$) were hypothesized to predict adolescent substance use onset. Second, variables in the immediately antecedent domain, *domain*$_{i-1}$, were also hypothesized to predict adolescent substance use. Third, variables in *domain*$_{i-1}$ were hypothesized to predict variables in *domain*$_i$. Fourth, variables in *domain*$_i$ were hypothesized to mediate partially the impact of variables in *domain*$_{i-1}$ on adolescent substance use. Fifth, variables in *domain*$_i$ were hypothesized to increment the prediction of adolescent substance use beyond variables in *domain*$_{i-1}$.

Another five hypotheses within each set tested similar hypotheses but with *domain*$_{i+1}$, instead of adolescent substance use, as the outcome, as follows:

Hb1: *Domain*$_i$ will predict *domain*$_{i+1}$.

Hb2: *Domain*$_{i-1}$ will predict *domain*$_{i+1}$.

Hb3: *Domain*$_{i-1}$ will predict *domain*$_i$.

Hb4: *Domain*$_i$ will mediate partially the impact of *domain*$_{i-1}$ on *domain*$_{i+1}$.

Hb5: *Domain*$_i$ will increment the prediction of *domain*$_{i+1}$ beyond *domain*$_{i-1}$.

As with Hypothesis a1, Hypothesis b1 states that variables in a domain (*domain*$_i$) would predict variables in a later domain, this time *domain*$_{i+1}$. Second, Hypothesis b2 states that variables in the immediately antecedent domain, *domain*$_{i-1}$, would also predict variables in *domain*$_{i+1}$. Hypothesis b3 is that variables in *domain*$_{i-1}$ will predict variables in *domain*$_i$. This hypothesis is actually the same as Hypothesis a3 but is repeated for ease of following the process. Next, Hypothesis b4 states that variables in *domain*$_i$ would mediate partially the impact of variables in *domain*$_{i-1}$ on variables in *domain*$_{i+1}$. Hypothesis b5 states that variables in *domain*$_i$ would provide an increment in the prediction of variables in *domain*$_{i+1}$ beyond variables in *domain*$_{i-1}$.

A final set of hypotheses addressed the full model simultaneously.

DATA ANALYTIC CHALLENGES

Historically, at least nine data analytic challenges have impeded testing the hypotheses listed above. These problems have been addressed in the current study through the application of contemporary methods and adherence to several decision rules.

The first challenge is that the "factors" (i.e., domains) in the proposed model are not hypothesized to be scales with highly correlated markers of a unitary underlying factor. With scales, it is assumed the measured indicators are caused by a single latent construct, so that the indicators are perfectly correlated with each other except for measurement error. Examples are scales of neuroticism and verbal intelligence. In contrast, an index is an aggregated sum of theoretically similar variables that are not presumed to be caused by a single latent construct or to be highly correlated with each other. Nonetheless, these variables are summed into an index because they are hypothesized to exert similar impact on other outcomes in a cumulative way. It is very plausible that variables might not be caused by the same construct but could still have similar impact on some outcome. One example is a stressful life events index. Although events such as a parent's death, loss of a job, and divorce are not assumed to be caused by the same source, or even significantly associated with each other, they have been found to exert similar adverse impact on one's health. Thus, it is justified to aggregate them into a single index.

Consider the current study's early parenting domain, which includes the variables nonmaternal child care, harsh discipline, positive parenting, and father involvement, among others. A major theoretical contribution to the field by Parke, Burks, Carson, Neville, and Boyum (1994) has been the discovery that various parenting variables are *not* indices of a single underlying construct. Instead, they are theoretically distinct and (relatively) empirically independent variables. Empirical studies validate this hypothesis. Pettit, Bates, and Dodge (1997) found that measures of positive parenting are empirically independent of measures of harsh discipline, and Bates et al. (1994) found that parents' child care decisions are only modestly associated with their disciplinary strategies. In the domain of later parenting, Pettit, Keiley, Laird, Bates, and Dodge (2007) found that parental monitoring is independent of discipline patterns. Laird, Pettit, Dodge, and Bates (2003) found that changes in parents' monitoring knowledge are only modestly related to parent–child relationship quality and discipline practice. Thus, variables in the parenting domain fit the model of an index better than that of a scale.

Kraemer (2008) has identified numerous problems with conducting mediational analyses in which constituent variables are treated as a scale when they violate the assumption of unity of the latent construct. Thus, it is neither theoretically nor statistically plausible to fit a single latent factor to this heterogeneous array. On the other hand, keeping every variable separate in data analysis would lead to an inefficient omnibus regression with 35 predictor variables, so the challenge is how to aggregate variables in a coherent way that retains their statistical and theoretical independence.

We identified PLS analysis (Chin, 1998) as a data-analytic approach, which is theoretically consistent with our hypothesized model. PLS is a variation of principal components analysis that results in a composite that is maximally related to some criterion. This composite is a weighted sum of observed scores and not a latent variable as in a factor analysis. This is a key distinction because PLS modeling allows theoretically related variables to be collated without the assumption that they are all indicators of a common source of variance, which is assumed in structural equation modeling (SEM). One of the implications is that correlations among the component variables need not be as high in order for the model to converge as

would be necessary for a successful confirmatory factor model. Thus, standards for empirical correlations among variables within a domain can be appropriately relaxed.

Consider the domain of sociocultural risk, which in the current study includes family socioeconomic status, teenage pregnancy, and family stress (among others). SEM requires the assumption that one underlying construct *causes* all of these indicators, whereas PLS allows the constituent variables to be independently caused with relatively low intercorrelations. The assumptions of PLS more accurately represent the domains under inquiry in the present study. Although PLS has been used in the past to relate constructs to each other (e.g., Chin, 1998), the current study is innovative in using PLS in mediational analyses involving three constructs (i.e., a predictor, an outcome, and a mediator).

The second challenge is how to model both the occurrence and timing of onset (early or late) of the dichotomous outcome variable of substance use as a function of multiple predictor variables. Discrete-time survival analysis (Willett & Singer, 1993) is a flexible tool for modeling the timing of events in a panel design. The models can be manipulated to test specific hypotheses, such as changes in the hazards of onset over time.

The third challenge concerns the typically high stability of individual differences in behavioral variables, making detection and prediction of change difficult. Cole (2006) argued that the study of developmental transitions improves the testing of change (and processes in change) because the transition itself signifies relative instability. In the current study, we addressed this challenge by focusing measurement on two important developmental transitions, to school and to adolescence.

Four more challenges relate to the testing of mediation. Ideally, a test of mediation includes three time points with all three variables (predictor, mediator, and outcome) measured at all three points, in order to distinguish developmental influences from mere stability in behavior (Cole, 2006; Cole & Maxwell, 2003). However, some developmental phenomena involve emergent behaviors that are not measurable at all time points. Substance-use onset is such an emergent behavior. On the other hand, it may be that stability in the *pattern* or construct of behavior, rather than the form, characterizes development. An apparent emergent behavior such as substance use might simply represent continuity in a pattern of deviance whose form changes with environmental and developmental context. Little, Weaver, King, Liu, and Chassin (2008) have identified a robust association between a construct of “deviance proneness” and marijuana use. Because this correlation may vary with secular trends, gender, and other moderators, marijuana use is not merely an indicator of the construct but is consistently correlated with it. Thus, the fourth challenge is to control for variables that might indicate an underlying pattern of deviant behavior.

In the current study, the best indicators of a pattern of early deviant behavior were mother-rated infant temperament scores and early behavior-problem levels. Consideration was also given to controlling for recent behavior in addition to early behavior, in order to generate the tightest test possible of the impact of a predictor variable on an outcome. However, the hypotheses of the cascade model suggest that the pattern of behavior itself may be influenced by developmental transactions with the environment, and so a test of the impact of a predictor on an outcome must be careful not to include as a covariate an endogenous outcome variable that is itself actually influenced by the predictor variable.

Consider that an outcome O at T4 is being predicted from a predictor P at T2 and a mediator M at T3. At what time point should the “prior” level of O be measured? O at T1 represents the underlying construct of the outcome. O at T2 represents the underlying construct of O at T1 plus any environmentally induced change in O. Thus,

$$O_{T2} = O_{T_1} + (O_{T2} - O_{T1}).$$

In this equation, the term ($O_{T2} - O_{T1}$) is entirely determined by environmental or intrapersonal variables at T2 and is by definition endogenous to the developmental process. If the test includes as a covariate the outcome variable at T2 or T3, then a true relation between the predictor and the outcome may be masked. In the current study, the initial level of an underlying construct of behavior problems, indexed by temperament and the T1 behavior problems score, was used as a covariate.

The fifth challenge is that conventional tests of mediation tend to be underpowered to detect indirect effects (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002a). Furthermore, they assume a normal distribution with symmetric confidence intervals, an assumption that rarely holds in mediation tests (Bollen & Stine, 1992). The current study uses indirect effects that were estimated as the product of the two component path co-efficients with an asymmetric empirical confidence interval that was derived by bootstrapping. This is the test that MacKinnon and colleagues recommend as apt to yield accurate significance tests.

The sixth challenge is that developmental transactional models such as the one posited here have historically been articulated at a more elaborate level than the level at which they have been tested. Models that have been described as multistep, sequential paths have been tested simply through multiple regression analyses with β coefficients, or, at best, as three-variable structural equation models. The tests used here map directly onto the level at which the full model has been articulated.

The seventh challenge concerns testing of moderation through interaction effects within mediation models. Multiple-group analyses have been developed to examine gender specificity in predictive models, and they were used here.

The eighth challenge is a common one in prospective inquiry over a long period of time; sporadic missing data for individual variables often cumulate to compromise the data set under list-wise deletion. The solution is that missing data can be multiply imputed (Schafer, 1999), and this method was applied presently.

The final challenge is another common one in large long-term prospective studies, although it is rarely discussed. Because of the time and expense of collecting data, many variables are collected. Thus, a data set such as this one has many more variables than can possibly be utilized in any single report. Selection of variables for inclusion in constructs, and constructs for inclusion in model testing, necessarily has some arbitrariness that may seem flawed in retrospect. Sometimes investigators will arrive upon an optimal set of variables after multiple failed tests and will report the final set without reporting the previous failed tests. We followed a process of reviewing the literature, generating hypotheses, and then selecting variables and constructs based on these hypotheses, with the constraint that all variables must be measured in temporal sequence. That is, because the hypotheses are temporally based, all variables that are hypothesized to predict a later variable must be measured before the later variable. There is one exception, which is that measurement of mother's lifetime alcohol use occurred after several variables that it is hypothesized to predict. Because we had failed to measure this variable at Time 1 and yet it is so crucial to most theories, we elected to retain it. Most importantly, though, we tested hypotheses with the a priori selected variable set even though we might have been able to increase internal consistency of constructs and the strength of cross-construct correlations by post hoc addition and deletion

of items. We do not doubt that stronger correlations could be found within this data set, but we believe that our process provides a fair test of the hypothesized model.

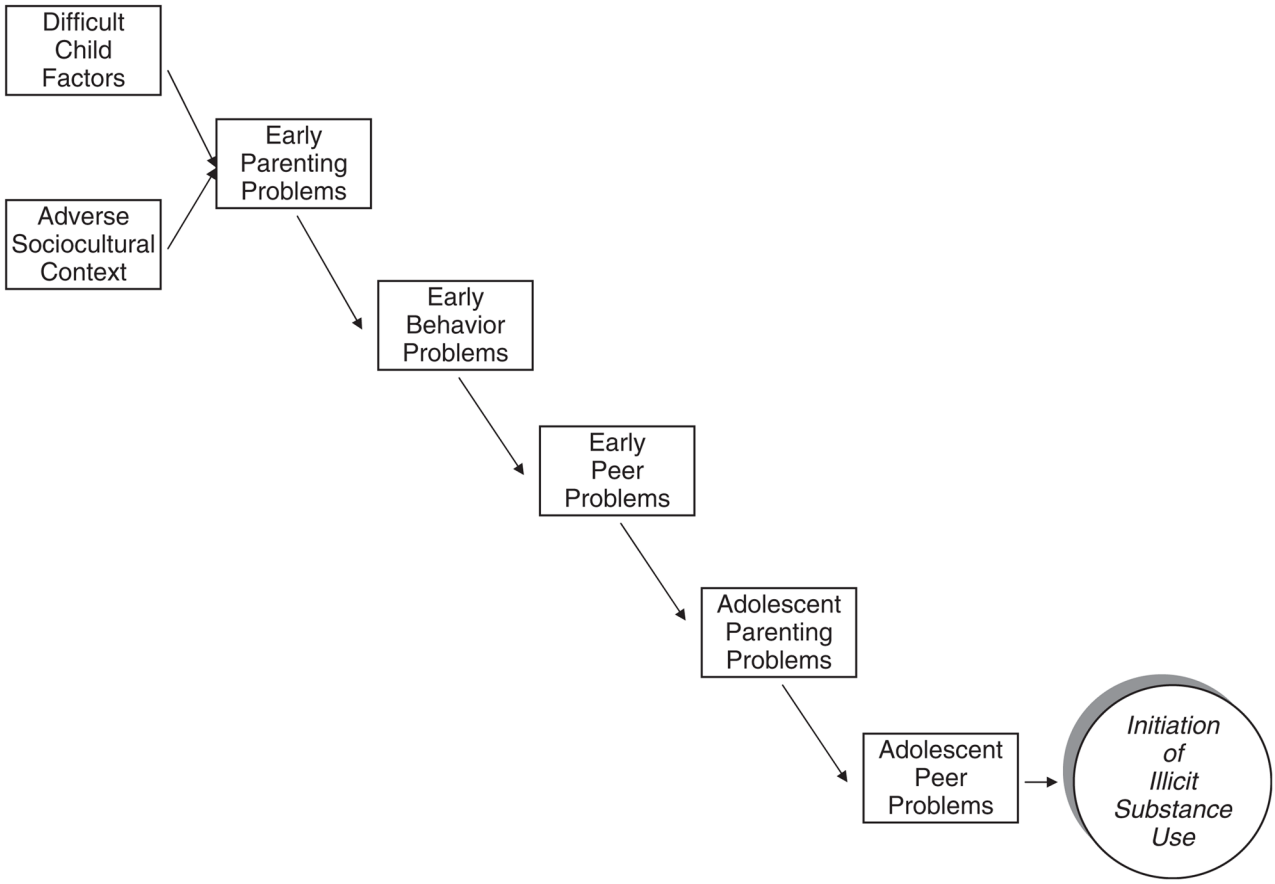

Figure 1.
A multistage transactional social learning model of substance-use development.